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OF MEDICINE

New York State Journal of Medicine

MEDICAL SOCIETY OF THE STATE OF NEW YORK
ORGANIZED 1807

VOL 92
NO 1
JANUARY
1992

OBSERVATIONS ON THE NATIONAL PRACTITIONER DATA BANK

DEER, TICKS, AND LYME DISEASE

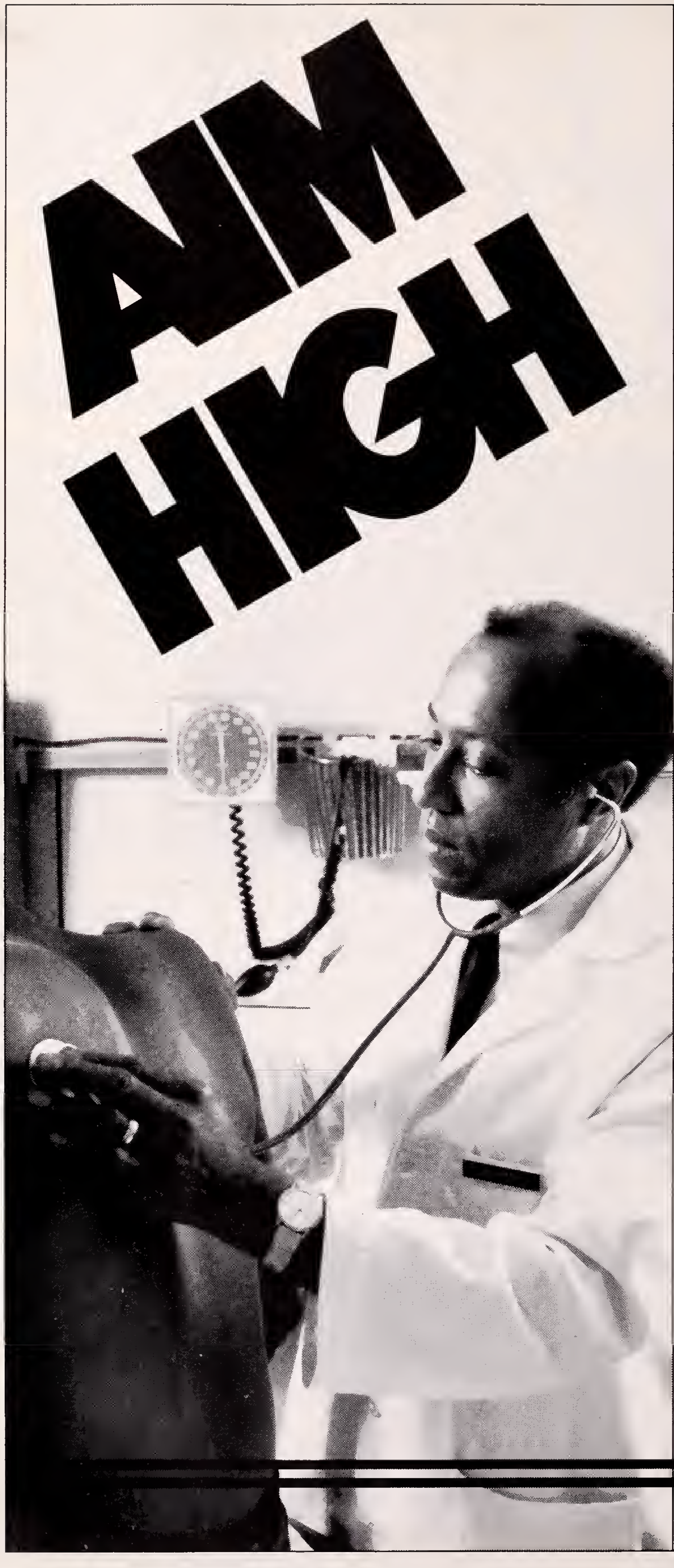
INCIDENCE AND PREVALENCE OF LYME DISEASE IN A SUBURBAN WESTCHESTER COUNTY COMMUNITY

MARITAL STATUS AND STAGE AT DIAGNOSIS IN CANCER

NATIONAL PRACTITIONER DATA BANK

UPDATE IN EPILEPSY

EOSINOPHILIC GLOBULES RESEMBLING MALLORY BODIES IN A RENAL CELL CARCINOMA



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FEBRUARY 1992

AROUND THE STATE

MANHATTAN

Feb 28–Mar 1. **NYU/Bellevue Hospital Center Psychiatry Board Review.** 24 ½ Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295.

MANHASSET

Feb 28. **Psychosocial Advances in the Treatment of Cancer Patients.** North Shore University Hospital. Contact: Irene Leff, MA, Department of Health Education/Community Affairs, North Shore University Hospital, Manhasset, NY 11030. Tel: (516) 562-0100.

AROUND THE NATION

ARIZONA

Feb 24–27. **Practical Radiology.** Loews Ventana Canyon Resort, Tucson. *Feb 27–29.* **Geriatric Medicine Update and Board Certification Review Course.** Doubletree Hotel, Tucson. Contact: Continuing Medical Education, Arizona Health Sciences Center, Tucson, Arizona 85724. Tel: (602) 626-7832.

CALIFORNIA

Feb 4–7. **Advanced Seminars in Diagnostic Imaging.** 20 Cat I Credits. Le Meridien Hotel, Coronado. *Feb 10–13.* **Mammographically Detected Early Breast Cancer.** 18 Cat I Credits. Ritz-Carlton, Rancho Mirage. *Feb 27–Mar 1.* **Advanced Seminars in Diagnostic Imaging.** 20 Cat I Credits. Ritz-Carlton Laguna Niguel. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773

(*Continued on p 11A*)

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Description: Yohimbine is a 3a-15a-20B-17a-hydroxy Yohimbine-16a-carboxylic acid methyl ester. The alkaloid is found in Rubaceae and related trees. Also in Rauwolfia Serpentina (L) Benth. Yohimbine is an indolalkylamine alkaloid with chemical similarity to reserpine. It is a crystalline powder, odorless. Each compressed tablet contains (1/12 gr.) 5.4 mg of Yohimbine Hydrochloride.

Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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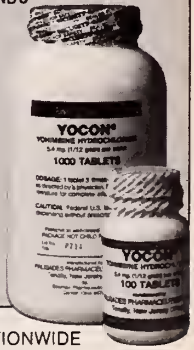

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JANUARY 1992 / Volume 92, Number 1

Contents

COMMENTARIES

1 **Observations on the National Practitioner Data Bank**
HENRY FERNANDEZ, JD, FRANK MUNOZ, JD

2 **Deer, ticks, and Lyme disease**
CATHERINE C. LASTAVICA, MD, MPH

RESEARCH PAPER

5 **Incidence and prevalence of Lyme disease in a suburban Westchester County community**
BARBARA ALPERT, MD, PHD; JOHN ESIN; STEVEN L. SIVAK, MD; GARY P. WORMSER, MD

8 **Marital status and stage at diagnosis in cancer**
KAMRAN NAYERI, MA; GREGORY PITARO, MD; JOSEPH G. FELDMAN, DRPH

SPECIAL ARTICLE

12 **National Practitioner Data Bank**
JAMES ROBB

REVIEW ARTICLES

14 **Update in epilepsy—Part I: Diagnosis and treatment of epilepsy**
CHAICHON LOCHARERNKUL, MD; DAVID PRIMROSE, MD; WEBSTER H. PILCHER, MD, PHD; LINDA M. OJEMANN, MD; GEORGE A. OJEMANN, MD

(continued on p 6A)

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New York State
Journal of Medicine

CASE REPORTS

18 **Eosinophilic globules resembling Mallory bodies in a renal cell carcinoma**
PAMELA UNGER, MD; GIORGIO PERINO, MD; HOWARD SCHIFF, MD; SWAN N. THUNG, MD; RONALD E. GORDON, PhD; MAMORU KANEKO, MD

20 **AIDS-related *Pneumocystis* pneumonia complicated by diffuse pneumatocele formation**
CHRISTINA N. JACOB, MD; SHERIF HENEIN, MD; A. ROSS HILL, MD

22 **Neonatal gastrointestinal complications of maternal cocaine abuse**
REGINA SPINAZZOLA, MD; KENNETH KENIGSBERG, MD; SHAISTA S. USMANI, MD; RITA G. HARPER, MD

LETTERS TO THE EDITOR

24 **Headaches and children: A reply to "An urban primary school health program"**
ALEXANDER MAUSKOP, MD

24 **Rhabdomyolysis associated with pneumococcal pneumonia and sepsis in an abstinent alcohol abuser**
ONYEKACHI IFUDU, MD; MARIANA MARKEL, MD; ROBERT BARTH, MD

25 **Enteral nutrition supplied by jejunostomy can result in jejunal infarction**
JOHN W. ODOM, MD; JANIS A. PASTENA, MD; PERRY RITOTA, MD; BARTHOLOMEW J. TORTELLA, MTS, MD; JAMES BLACKWOOD

26 OBITUARY LISTINGS

HOUSE OF DELEGATES

27 Sessions; Officers, Councilors, and Trustees; Members; Nominating and Reference Committees; Report of the House Committee on Bylaws

3A MEDICAL MEETINGS AND LECTURES

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Blank space indicates that no such activity has been reported. Table adapted from Facts and Comparisons 1991 and Catalano RB. The medical approach to management of pain caused by cancer. *Semin. Oncol.* 1975; 2; 379-92 and Reuler JB, et. al. The chronic pain syndrome: misconceptions and management. *Ann. Intern. Med.* 1980 588-96.

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Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression.

Head Injury and Increased Intracranial Pressure: The respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a preexisting increase in intracranial pressure. Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.

PRECAUTIONS:

Special Risk Patients: VICODIN/VICODIN ES Tablets should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.

Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN/VICODIN ES Tablets are used postoperatively and in patients with pulmonary disease.

Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN/VICODIN ES Tablets may exhibit an additive CNS depression. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.

Usage in Pregnancy:

Teratogenic Effects: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN/ VICODIN ES Tablets should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever.

Labor and Delivery: Administration of VICODIN/VICODIN ES Tablets to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN/VICODIN ES Tablets, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS:

The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include:

Central Nervous System: Drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, psychic dependence and mood changes.

Gastrointestinal System: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation.

Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.

Respiratory Depression: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing. If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated.

DRUG ABUSE AND DEPENDENCE:

VICODIN/VICODIN ES Tablets are subject to the Federal Controlled Substance Act (Schedule III). Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN/ VICODIN ES Tablets should be prescribed and administered with caution.

OVERDOSAGE:

Acetaminophen Signs and Symptoms: In acute acetaminophen overdosage, dose-dependent, potentially fatal hepatic necrosis is the most serious adverse effect. Renal tubular necrosis, hypoglycemic coma, and thrombocytopenia may also occur. Early symptoms following a potentially hepatotoxic overdose may include: nausea, vomiting, diaphoresis and general malaise. Clinical and laboratory evidence of hepatic toxicity may not be apparent until 48 to 72 hours post-ingestion.

Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

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COMMENTARIES

Observations on the National Practitioner Data Bank

The National Practitioner Data Bank (NPDB) was established by Congress as part of Title IV of Public Law 99-660, the Health Care Quality Improvement Act of 1986, as amended. Regulations governing the Data Bank are codified in 45 CFR Part 60. The NPDB is designed to address several Congressional findings related to the delivery of health care services. The Congressional findings, listed 42 USC 1101, are as follows:

- The increasing occurrence of medical malpractice and the need to improve the quality of health care services are nationwide problems, requiring solutions beyond the capability of the individual states.
- There is a need to restrict the ability of incompetent practitioners to move from state-to-state without disclosure of previous damaging or incompetent performance.
- This nationwide problem can be remedied through effective peer review.
- The threat of private money damage liabilities under federal laws, including treble damage liability under Federal antitrust law, unreasonably discourages physicians from participating in effective professional peer review.
- There is an overriding national need to provide incentive and protection for physicians engaging in effective professional peer review.

The NPDB is operated by the UNISYS Corporation under contract to the Division of Quality Assurance and Liability Management of the US Department of Health and Human Services (DHHS). Final regulations were published in the Federal Register in October 1989 and were followed in early 1990 by a series of educational conferences conducted by UNISYS and DHHS representatives for state licensing authorities, insurance companies, and hospital associations. Subsequent meetings with representatives of state licensing authorities and various professional associations tackled problems identified at the conferences. Finally, the Bank became operational in September, 1990.

The NPDB's purpose is to collect and disseminate information relating to professional performance. Four types of information must be reported to the Bank:

- Payments made in settlement of malpractice claims.
- Adverse actions affecting membership in a professional society.
- Adverse actions of more than 30-days duration taken by hospitals and other health care entities, such as health maintenance organizations and group medical practices.
- Disciplinary actions taken by state licensing authorities which revoke, suspend, censure or otherwise restrict the license to practice, or which place a licensee on probation.
- Surrender of a license while disciplinary proceedings are pending must also be reported. However, if the surrender of a license during treatment for substance abuse is made outside of the regular discipline process and in accordance with applicable law, it is not reportable. This applies, for example, to physicians enrolled in the impaired physicians' program.
- A corrective report to the Data Bank is made only if a court actually changes or reverses a disciplinary action, or if a court orders new proceedings which result in a change or dismissal.

The regulations require hospitals and other health care entities to submit Adverse Action reports to the Data Bank through a designated Medical or Dental Board within 15 days of the effective date of the action. In New York, Adverse Action reports are sent to the Office of Professional Discipline of the New York State Education Department, which in turn is responsible for forwarding the reports to the Data Bank within 15 days. Adverse Action reports of malpractice settlements involving physicians are sent directly by the insurer to the Data Bank. The insurer sends a copy of the report to the Office of Professional Discipline.

The Office of Professional Discipline sends a copy of all Adverse Action reports on physicians to the Department of Health.

Reports received by the Bank are verified with the sender, and the subject is informed by the Bank of the contents of the report. If the subject disputes the accuracy of a report, and the dispute cannot be resolved directly between the subject and the sender, an appeal may be made to the Secretary of DHHS. If the Secretary finds that the report is in error, it will be corrected or deleted, and all parties who have received the incorrect information in response to an inquiry will be notified of the correction. The reports constitute the Bank's record on individual licensees. Information from these reports is sent to parties querying the Bank.

The federal regulation imposes a duty on hospitals to query the Data Bank before hiring or granting privileges to a health care practitioner. In addition, hospitals must query the Bank every two years on health care practitioners who are on staff or have privileges. Failure to query the Bank results in a legal presumption in a malpractice action that the hospital actually had the information reported with respect to the physician or practitioner.

The Data Bank may also be queried by state licensing boards, health care authorities other than hospitals and professional societies. Attorneys filing malpractice actions or claims against a hospital, or a plaintiff representing himself or herself (*pro se*) in such action, may query the Bank as to a practitioner also named in the lawsuit, upon a showing that the hospital failed to query the NPDB as to that practitioner. Lastly, a practitioner may request information concerning himself or herself.

The Data Bank regulations state that reports are confidential and persons who receive such information may use it "solely with respect to the purpose for which it was provided." Penalties for violations range up to $10,000.

The legislation establishing the Data Bank does not provide guidance or specific standards for evaluating and using the information received in response to a query. The weight given by a hospital to a history of malpractice settlements, for example, will vary depending on a number of factors, such as the number of settlements compared to the experience of other practitioners in the specific practice and geographic area, the size of the settlements, the explanations offered by the applicant, and the needs of the facility.

The requirement for mandatory querying by hospitals is intended to ensure that decisions on hiring and granting clinical privileges are based on an applicant's complete professional history. Because a hospital failing to query the Bank is legally presumed to have known the contents of the Bank's report, prudence suggests that a hospital be aware of the disciplinary and malpractice history of every staff member and every practitioner with clinical privileges, and that it be able to articulate a rationale for hiring or granting privileges to someone with a significant adverse history. The threat of legal liability for negligent credentialing decisions is a powerful incentive for hospitals to comply with their querying requirements.

New York is typical of other licensing jurisdictions in relying on the self-reporting of an adverse professional history by applicants for licensure. In New York, membership in several national disciplinary clearinghouses supplements the information volunteered by applicants. As deposits are made over time, routine querying of the Bank as part of the licensing process will provide a complete profile of applicants and ensure that licensing decisions by the states are fully informed.

Many provisions of the Data Bank have their parallel, in some form, in already existing New York law.

- Section 2803(e) of the Public Health Law requires hospitals to report a wide range of actions against physicians to the Department of Health.
- Section 315 of the Insurance Law requires payments made in settlement of malpractice actions based on fraud, incompetence, or negligence against physicians to be reported to the Department of Health.
- Section 2805(k) of the Public Health Law requires hospitals to investigate the disciplinary and malpractice history of physicians before hiring or granting privileges.

New York law is more inclusive than Data Bank regulations regarding the type of actions against physicians which must be reported. Many actions which must be reported to the Department of Health may never be reported to the Data Bank because they are of less than 30-days duration or because they were taken without following the specific procedures contained in Data Bank regulations. On the other hand, Data Bank regulations mandate reporting a wider range of malpractice payments than is required under New York law.

The Data Bank should be a valuable resource for state licensing and disciplinary authorities. Its most significant affect on the quality of medical care, however, will likely come from the querying procedure hospitals are required to follow. The pressure for improvement is exerted through hospital credentialing committees with access via the Bank to complete information on which to base staffing decisions. Physicians will become aware that a significant adverse history may pose barriers to continued successful practice, and the consumer of medical services will be able to hold both the health care institution and the practitioner ultimately accountable.

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Deer, ticks, and Lyme disease

Another step in the march of Lyme disease across our landscape is described in the study of Alpert et al which appears in this issue of the *Journal* and documents the rising risk of acquiring infection in a residential suburban community.[1] Chappaqua, Westchester County, New York, is a site where reforestation has been accompanied by a burgeoning population of deer, *Odocoileus virginianus,* the definitive hosts for the northern deer tick, *Ixodes dammini,* the vector of the Lyme disease spirochete, *Borrelia burgdorferi,* in the northeast and upper midwest regions of the United States.[2,3] The spread of Lyme disease has been characterized by focal outbreaks,[4–7] increased impact of the disease in established sites,[8] and progression to the suburban doorstep.

In recent years, Lyme disease has emerged from the wildlife cycles in nature, in which the spirochete is maintained.[9] That the vector tick was present but went unnoticed, has been demonstrated by Persing et al, who used the polymerase chain reaction to detect spirochete-specific DNA sequences in museum specimens of *I. dammini,* and found that *B. burgdorferi* infected *I. dammini* on Long Island, New York, during the 1940s, a generation before its appearance in humans.[10] In nature, larval and nymphal *I. dammini* feed on the white-footed mouse, *Peromyscus leucopus,* which remains highly infectious for extended periods of time, thereby perpetuating the spirochete. An overpopulation of deer promotes an explosive increase in the vector tick, and transmission may be intense in newly established foci.[7] Under such conditions, aggressive *I. dammini* attach to a variety of incidental hosts including humans and domestic animals; dogs can hardly escape infection.[11] Thus, it is, as a zoonosis, that *B. burgdorferi* demands our attention.

The range of Lyme disease in North America has been greatly expanded. Transmission seems to occur wherever the *Ixodes* vector has become well established.[3] *B. burgdorferi* may be introduced into new locations by the rare occurrence of transovarial transmission and the transport of infected larval *I. dammini* on migrating birds.[12] Alternatively, certain species of these birds may also serve as reservoir hosts for the spirochete.[13,14] In the Saint Croix River Valley, an endemic region for Lyme disease in east central Minnesota and northwestern Wisconsin, fifteen migrant species were found to be parasitized by immature *I. dammini,* and nearly three-quarters of those were accounted for by ground-foragers such as veery, ovenbird, northern water thrush, common yellow throat, and swamp sparrow. Recaptured migrant birds with infected ticks indicate that they transmit *B. burgdorferi* to larval *I. dammini* suggesting that birds may disperse the zoonosis over long distances.[15]

The spread of Lyme disease is not limited to the western hemisphere. In Europe, the Old World deer tick, *I. ricinus*, is the vector for both European tick-borne encephalitis and Lyme disease, and mice of the genus *Apodemus* occupy an ecological niche similar to that of the white-footed mouse in the United States.[16] Lyme disease, first described in Sweden[17] and long appreciated in Europe,[18] has been reported from virtually every country of that continent;[19,20] the rising incidence of Lyme disease in Europe parallels that in the United States and coincides with a dramatic increase in the abundance of deer since the second World War.[16] In Scotland, fallow deer, known for their boldness in approaching humans, are increasing at scenic tourist attractions near large metropolitan areas,[21] and foci of infection have been emerging throughout the British Isles.[22] In Switzerland, asymptomatic seroconversions by *B. burgdorferi* appear to be common.[23] Lyme disease has occurred in diverse foci across the Soviet Union.[24] In Asia, where *I. persulcatus* is the vector, the zoonosis is becoming a public health problem in China.[25] Lyme disease has become the most prevalent arthropod-borne illness in the north temperate zone.

Sites like the one described in Chappaqua, New York, by Alpert et al in this issue of the *Journal*,[1] are not uncommon, and more may be expected as epidemics of Lyme disease emerge in suburban areas. Thousands of cases, including many persistent infections with late manifestations of disease, are reported annually in the United States mainly from the northeast, upper midwest, and northern California where *I. pacificus* is the vector. The number of reported cases of Lyme disease nearly doubled each year from 1982 through 1989,[26] and many go unreported. In newly established foci of the zoonosis, infections need to be documented, and neighbors, as well as family members, investigated. Arriving at an accurate understanding of the incidence of Lyme disease requires active surveillance.

As Lyme disease expands its range into suburban sites, the less prevalent companion infection, babesiosis, is emerging at the same time and increasing in some locations.[27] This malaria-like piroplasm, *Babesia microti*, is a cosmopolitan parasite of rodents and may be transmitted by transfusion,[28] as well as by *I. dammini*. The piroplasm generally produces subclinical infection, but severe illness may occur in elderly, splenectomized or otherwise immunocompromised patients. The incubation period of two to four weeks after the tick bite and the seasonal risk, which is greatest in May and June and corresponds to periods of increased transmission, are similar to those of Lyme disease. In the eastern part of North America both the agents of Lyme disease and babesiosis are perpetuated in white-footed mice. *B. microti* is less intensely transmitted and appears to infect a more limited range of hosts than does the spirochete. In contrast to the spirochete, the piroplasm appears not to be inherited by the vector tick, and birds appear not to serve as competent hosts. Thus, human babesiosis invades new regions much more slowly than does Lyme disease.[29]

The long-term strategy for reducing the impact of these zoonoses needs to include ecological approaches to reduce or interrupt transmission. The coincidence of an abundance of deer at every location where Lyme disease outbreaks have occurred in the northeast, the correlation between larval *I. dammini* attached to white-footed mice and the density of deer on thirteen Massachusetts islands,[30] and the reduced abundance of immature *I. dammini*, after the elimination of deer on Great Island, off West Yarmouth in Cape Cod, Massachusetts,[31] are convincing evidence that deer are responsible for recent outbreaks. Management of these animals is basic to environmental efforts seeking to reduce the risk of people acquiring Lyme disease and babesiosis.

In colonial times, the deer herd was affected by political and economic pressures. Before 1600, the Indians maintained a stable relationship with their fauna.[32] A park-like landscape, conducive to the development of deer was formed by deliberate burning, and the Indians concentrated their hunting during the fall and winter months. The early settlers in southern New England found deer to be quite plentiful. They used them for food year-round and their skins for both clothing and to meet the demands of an export market. The Indians, less able to compete with the newcomers and suffering from epidemics of infectious disease, military conflicts, and political turmoil, became more sedentary and began to exploit their available resources. Furs, hides, and meat were the primary commodities which the Indians sold to the settlers, resulting in exploitation of the remaining large animals. Deer were so reduced toward the end of the seventeenth century that Massachusetts enforced its first closed season on their hunting in 1694. In 1739, the closed season was extended form January 1 to August 1 in conjunction with a law passed the same year establishing uniform protection by providing for the appointment of two persons in each town, later called *deer reeves*, whose special duty it was to see that the closed-season law was enforced. The laws enacted for the protection of deer during the 1700s were no doubt feebly enforced. By 1800, deer were quite scarce, and by 1850, practically eliminated. Only in the woods and thickets of the Cape Cod region were they definitely known to have survived in small numbers.[33]

By 1870, the Cape Cod deer were apparently quite numerous again. Further deer-protecting laws were passed allowing deer kills at specified times or when crops were being damaged. Other laws indemnified farmers and fruit growers for crop damage by deer. Economic changes causing widespread reversion of farmlands to forests, lack of predators and public sentiment toward animal rights have allowed the deer population to continue to increase to this day.

The chronicle of Lyme disease has spanned more than a century,[18] and some of the chapters are still to be written. When an epidemic form of arthritis in Old Lyme, Connecticut, was reported in 1977, it was thought to represent a previously unrecognized clinical entity; the clustering of cases suggested an infectious etiology, and transmission by an arthropod vector was entertained.[4] Today the spectrum of clinical manifestations of Lyme disease continues to increase,[34] and the ecological relationships continue to unfold around the world.[3] In regions such as the northeastern part of the United States, residents of suburban communities need to consider carefully the sometimes controversial problem of deer and their relationship to human well-being.

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1. Alpert B, Esin J, Sivak SL, et al: Incidence and prevalence of Lyme disease in a suburban Westchester County community. *NY State J Med* 1991; 91: 5–8.
2. Spieldman A, Wilson ML, Levine JF, et al: Ecology of *Ixodes dammini*-borne human babesiosis and Lyme disease. *Ann Rev Entomol* 1985; 30:439–460.
3. Lane RS, Piesman J, Burgdorfer W: Lyme borreliosis: Relation of its causative agent to its vectors and hosts in North America and Euorpe. *Annu Rev Entomol* 1991; 36:587–609.
4. Steere AC, Malawista SE, Snydman DR, et al: Lyme arthritis: an epidemic of oligoarticular arthritis in children and adults in three Connecticut communities. *Arthritis Rheum* 1977; 20:7–11.
5. Hanrahan JP, Benach JL, Coleman JL, et al: Incidence and cumulative frequency of endemic Lyme disease in a community. *J Infect Dis* 1984; 150:489–496.
6. Steere AC, Taylor E, Wilson ML, et al: Longitudinal assessment of the clinical and epidemiological features of Lyme disease in a defined population. *J Infect Dis* 1986; 154:295–300.
7. Lastavica CC, Wilson ML, Berardi VP, et al: Rapid emergence of a focal epidemic of Lyme disease in coastal Massachusetts. *N Engl J Med* 1989; 320:133–137.
8. Lyme disease–Connecticut. *MMWR* 1988; 37:1–3.
9. Telford III SR, Pollack RJ, Spielman A: Emerging vector-borne infections. (Animal Associated Human Infections) *Inf Dis Clin North America* 1991; 5:7–17.
10. Persing DH, Telford III SR, Rys PN, et al: Detection of *Borrelia burgdorferi* DNA in museum specimens of *Ixodes dammini* ticks with the polymerase chain reaction. *Science* 1990; 249:1420–1423.
11. Eng TR, Wilson ML, Spielman A, et al: Greater risk of *Borrelia burgdorferi* infection in dogs than in people. *J Infect Dis* 1988; 158:1410–1411.
12. Piesman J, Donahue JG, Mather TN, et al: Transovarially acquired Lyme disease spirochetes (*Borrelia burgdorferi*) in field-collected larval *Ixodes dammini* (Acari: Ixodidae). *J Med Entomol* 1986; 23:219.

13. Anderson JF: Mammalian and avian reservoirs for *Borrelia burgdorferi*. *Ann NY Acad Sci* 1988; 539:180–191.

14. Anderson JF, Magnarelli LA, Stafford III KC: Bird-feeding ticks transstadially transmit *Borrelia burgdorferi* that infect Syrian hamsters. *J Wildl Dis* 1990; 26:1–10.

15. Weisbrod AR, Johnson RC: Lyme disease and migrating birds in the St. Croix River Valley. *Appl Environ Microbiol* 1989; 55:1921–1924.

16. Matuschka FR, Spielman A: The emergence of Lyme disease in a changing environment in North America and Central Europe. *Experimental & Applied Acarology.* 1986; 2:337–353. Elsevier Science Publishers B.V. Amsterdam. (Review)

17. Afzelius A: Erythema chronicum migrans. *Acta Derm Venereol* (Stockh) 1921; 2:120–125.

18. Dammin GJ: Erythema migrans: A chronicle. *Rev Inf Dis* 1989; 11:142–151.

19. Stanek G, Pletschette M, Flamm H, et al: European Lyme borreliosis. *Ann NY Acad Sci* 1988; 539:274–282.

20. Schmidt R, Kabatzki J, Hartung S, et al: Epidemiologic aspects of erythema chronicum migrans disease in the Federal Republic of Germany. *Zbl Bakt Hyg* 1986; A263:435–441.

21. Hamlet N, Nathwani D: Lyme disease (letter). *N Engl J Med* 1989; 320: 1626.

22. Muhlemann MF, Wright DJM: Emerging pattern of Lyme disease in the United Kingdom and the Irish Republic. *Lancet* 1987; 1:260–262.

23. Fahrer H, van der Linden SM, Sauvain M-J, et al: The prevalence and incidence of clinical and asymptomatic Lyme borreliosis in a population at risk. *J Infect Dis* 1991; 163:305–310.

24. Dekonenko EJ, Steere AC, Berardi VP, et al: Lyme borreliosis in the Soviet Union: A cooperative US-USSR report. *J Infect Dis* 1988; 158:748–753.

25. Ai CX, Wen XY, Zhang YG, et al: Clinical manifestations and epidemiological characteristics of Lyme disease in Hailin county, Heilongjiang Province, China. *Ann NY Acad Sci* 1988; 539:302–313.

26. Lyme disease surveillance—United States, 1989–1990. *MMWR* 1991; 40: 417–421.

27. Babesiosis—Connecticut. *MMWR* 1989; 38:649–650.

28. Mintz ED, Anderson JF, Cable RG, et al: Transfusion-transmitted babesiosis: A case report form a new endemic area. *Transfusion* 1991; 31:365–368.

29. Spielman A: Lyme disease and human babesiosis: Evidence incriminating vector and reservoir hosts. *The Biology of Parasitism.* 147–165, Alan R. Liss, Inc, 1988.

30. Wilson ML, Adler GH, Spielman A: Correlation between abundance of deer and that of the deer tick, *Ixodes dammini* (Acari: Ixodidae). *Ann Entomol Soc Am* 1985; 78:172–176.

31. Wilson ML, Telford SR III, Piesman J, et al: Reduced abundance of immature *Ixodes dammini* (Acari: Ixodidae) following elimination of deer. *J Med Entomol* 1988; 25:224–228.

32. Cronon W: *Changes in the Land: Indians, Colonists, and the Ecology of New England*. New York, Hill and Wang, 1983.

33. The management of white-tailed deer. Massachusets Bureau Wildlife Management 1951; *Res Bul* 13:3–6.

34. Steere AC: Medical Progress: Lyme disease. *N Engl J Med* 1989; 321:586–596. (Review)

THE GALVANO CAUTERY AS A FACTOR IN PERITONSILLAR ABSCESS

The etiology of peritonsillar abscess is in all probability very closely akin to the pathology of this painful condition. Heretofore the books have ascribed the incidence of abscess around the tonsil to the continuation of an attack of acute follicular tonsillitis, to exposure, to sexual excess, etc., but it is quite likely that these factors are of only secondary importance. Pus is formed in the loose connective tissue around the tonsil as a result of a bacterial invasion either through the crypts of the tonsil or between the tonsil and its anterior or posterior pillar. Curiously enough the tendency of this pus is to burrow upward toward the soft palate; hence in relieving the condition the incision is made into the anterior or posterior superior quadrants.

From a knowledge of the etiology and pathology of peritonsillar abscess it is, therefore, quite obvious that any process, either natural or artificial, which seals over the oral openings of the crypts, or which binds the pillars firmly to the tonsillar tissue, prevents drainage and produces in the tonsillar region all the conditions necessary to thorough incubation of retained bacteria. In three cases that have recently come to our notice this state of things was apparently brought about by the indiscriminate use of the galvano cautery in the hands of the general practitioner. It is noteworthy that in each case the tonsils were of the firm fibrous variety and were found on microscopical examination to contain a large amount of connective tissue. In one case cauterization had been carried out twice in a week for six weeks without reducing the size of the tonsil to any appreciable extent. Both the anterior and the posterior pillars were bound down by dense adhesions, so that in making the incision difficulty was experienced in deciding at what point to introduce the knife because the old landmarks were entirely obliterated. The pus in these cases was very foul and not at all unlike that found in ischio-rectal abscesses. There was also some discharge of cheesy material which had been unable to pass out through the proper channel, namely, crypts.

There is, therefore, every reason to believe from presumptive evidence, actual proof being quite difficult, that the use of cauterants and so-called "shrinking methods" in tonsillar disease is often unwarranted and undesirable. In many cases there is little relation between the size of a tonsil and the pathological evidence of disease in it. It is quite possible to have a tonsil which projects considerably beyond the pillars, but which, when excised and examined, does not show special predisposition to act as a nidus for disease germs or to undergo parenchymatous degeneration. On the other hand one may find associated with a small insignificant amount of amygdaloid tissue a large amount of degeneration and decomposition with perhaps many nodules indicating the presence of cervical adenitis. The frequency with which tonsillitis occurs upon the stumps left behind after a tonsillotomy is sufficient to make this fact patent.

It is probable that the physician would find it wise to limit galvano cauterization to the small ragged tonsil which lies fairly free in its bed and which is not adherent to the pillars. Large fibrous tonsils when imbedded are best removed by free dissection according to the method of Freer, or by the punch or snare. In any case one should be careful not to interfere with free drainage from the crypts.

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(*NY State J Med* 1909; 9:166)

Incidence and prevalence of Lyme disease in a suburban Westchester County community

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ABSTRACT. This study was designed to determine the incidence and prevalence of Lyme disease in a section of Chappaqua, NY, a residential community in which Lyme disease is epidemic, and to identify risk factors for this disease. On the basis of clinical history and baseline serologic testing, the overall prevalence of Lyme disease for 114 persons entering the study was 8.8%. The incidence during the 5-month study period of May through September 1989 was 2.6%; all three incident cases had erythema migrans (EM). Hours outdoors per week in play or exercise correlated with the occurrence of Lyme disease.

(NY State J Med 1992; 92:5–8)

Westchester County is a suburban community just north of New York City, where Lyme disease is epidemic. This study was designed to investigate the prevalence and summertime incidence of Lyme disease in a section of northern Westchester County.

Methods

Chappaqua is a village in the town of New Castle, New York. New Castle covers approximately 24 square miles, much of which is wooded or "re-forested," and inhabited by a large population of white-tailed deer, *Odocoileus virginianus.* Sixteen thousand people reside in the town. This site was chosen on the basis of convenient access for the investigators.

The study population was composed of all persons over one year of age who resided in any of the households situated in a discrete section of Chappaqua adjacent to the Saw Mill River Parkway. Questionnaires were mailed in April 1989 to 144 households listed by the town office in the assessment rolls. The questionnaire requested information on the number of residents in the house during the study period (May through September 1989), earlier history of Lyme disease, basis for earlier diagnosis of Lyme disease, history of tick bites, terrain of property, duration of residency, household pets, outdoor activity, presence of wildlife (including deer) in yards, preventive measures taken against tick bites, and the presence of signs and symptoms associated with Lyme disease.

Blood testing was offered to all residents on three occasions during the study period: early May, late July, and early October. Follow-up questionnaires were mailed both in late July and in early October. Those individuals who reported a history of Lyme disease or in whom symptoms developed during the study period were personally interviewed by one of us. An additional questionnaire was mailed to the non-participating households in December 1989. The households answering the December mailing did not undergo blood testing.

Lyme disease was defined on the basis of either EM, or a commonly accepted manifestation of Lyme disease, such as arthritis with objective joint swelling or facial nerve palsy, plus a positive serologic test for antibodies to *Borrelia burgdorferi*, in accordance with the surveillance definition of the New York State Department of Health.[1] To be considered as likely EM, for the purposes of the study, the rash had to have been red, round, greater than two inches across, expanding, and persisting for several days.

The incidence of Lyme disease was calculated on the basis of the number of persons clinically identified as having Lyme disease over the study period, plus the number of asymptomatic seroconverters. The prevalence of Lyme disease was calculated on the basis of the number of individuals with a clinical history of Lyme disease, plus those without such a history but who were seropositive at entry.

Coded, batched serum samples stored at −70°C were analyzed by a polyvalent enzyme-linked immunosorbent assay.[2,3] Positive samples were defined as those with a titer of 1:160 or higher.

For continuous unmatched variables, univariate analysis of differences of means was obtained by using a two-tailed t test.[4] For discrete variables, differences in proportions were compared using the χ^2 test. Differences were considered statistically significant when $p < 0.05$. Confidence intervals were calculated using the formulas for the binomial distribution. For the purposes of statistical analysis, all missing data, both clinical and serologic, were assumed to be negative for Lyme disease.

Results

Fifty eight of 144 households elected to participate. The total number of persons residing in the participating households during the 5-month study period was 173, or three per household on average. The participation rate among these households was 114 of 173 or 66%. Based on an assumption of three residents per household for non-participating households, the estimated population of the study site was 432. Thus, approximately 26% (114 of 432) of the possible study sample participated. The majority of these (86 of 114, 78%) both completed the questionnaires and submitted blood samples on all three test dates. Reasons for incomplete participation included moving from the area ($n = 9$), late entry into the study ($n = 6$), or voluntary withdrawal ($n = 13$).

Of 114, eight (7.0%) participants reported a history of Lyme disease: Seven had EM, and one had arthritis and a positive serologic test (Table I). Their ages ranged from 7 to 69 years (mean ±SD = 41.6 ±22.5 years); six (75%) were male. Three individuals reported multiple episodes of Lyme disease. These eight participants felt well at the time of entry into the study and did not significantly differ from other study participants in any parameter addressed by the questionnaire except for tick bite recollection. Recollection of a tick bite occurred more commonly (6 of 8, 75%) in this group than in the participants without an earlier history of Lyme disease (19 of 106, 17%, $p = 0.0009$).

Seven (6.5%) of the 108 blood samples obtained in May of 1989 were positive for antibodies to *B burgdorferi* (Table I). Six persons entered the study in July 1989, and all six were seronegative. Thus, the overall seroprevalence at initial test date was 6.1%. Positive titers remained positive on subsequent testing throughout the study period. The age range of seropositive persons was 7–69 years (mean ±SD = 35.9% ±25.5 years); five of the seven persons were male (71.4%).

Of the eight persons with a clinical history of Lyme disease, five

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(67.5%) were seropositive. Of the two (1.9%) seropositive individuals without clinical history of Lyme disease, one had been previously diagnosed as having juvenile rheumatoid arthritis because of an illness which may, in retrospect, have been Lyme disease.

The seropositive individuals differed significantly from those who were seronegative in only two respects: providing a history of Lyme disease or a tick bite. The presence of antibodies to *B burgdorferi* was significantly associated with a clinical history of Lyme disease, 5 of 7 versus 2 of 107 ($p < 0.0001$), even if seropositivity were excluded from the case definition. Five of the seven seropositive persons recalled earlier tick bites (71.4%), compared with 20 of 107 (18.7%) participants who had no serologic evidence of Lyme disease ($p = 0.0052$).

Three cases of Lyme disease occurred among the 114 (2.6%) participants during the study period (Table I). All three were males and had EM, as defined by a physician. One of three felt well, whereas the other two were highly symptomatic. One symptomatic individual described fatigue, fever, headache, myalgias, and arthralgias, and the other individual had a monoarticular arthritis. The latter individual was the only person to seroconvert. One individual specifically recalled a tick bite that preceded the rash. When hours per week of outdoor recreational activity were examined, there was a significant association with the development of Lyme disease, 16.67 hours (Lyme disease) versus 4.95 hours (no Lyme disease) ($p = 0.0015$). None of the other variables was associated with the occurrence of Lyme disease during the study period.

To address sampling bias, a questionnaire was mailed to non-participating households in December 1989. This mailing provided information on an additional 90 residents who resided in 30 households. The reported incidence and prevalence data for Lyme disease in this group were as high or higher than in the main study group. Specifically, 25 persons (28%) reported a history of a tick bite before 1989, while 9 (10%) reported an earlier history of EM. In addition, two individuals (2.2%) reported the occurrence of EM during the 5-month study period.

Discussion

Lyme disease is epidemic in Westchester County, with 740 confirmed cases in 1988 and 1,159 confirmed cases in 1989 (Fig 1). The majority of cases has occurred among residents in the northern portion of the county, in which the Chappaqua study site is situated. The results of this study may help explain in part why a large number of Lyme disease cases are being reported locally (Table I).

Our study findings are compared with those observed in three other epidemiologic investigations conducted in the United States (Table II). Two sites had a transient human population at island summer resorts: Fire Island, New York[5]; and Great Island, Massachusetts.[6] A third site was a coastal community (Ipswich, MA)[7] located adjacent to a nature preserve. These geographic locations are distinctly different from the Chappaqua site, which we believe to be representative of numerous residential, ½- to 1-acre zoned communities in northern Westchester County. Chappaqua is a suburban community, with a stable year-round population that is not engaged primarily in recreational activities, as would occur in a summer community, and is not located adjacent to a beach or a large nature preserve. The landscape of this region is distinct from coastal or island communities, with forests and lawns instead of beaches, dunes, scrub pines, and protected park land.

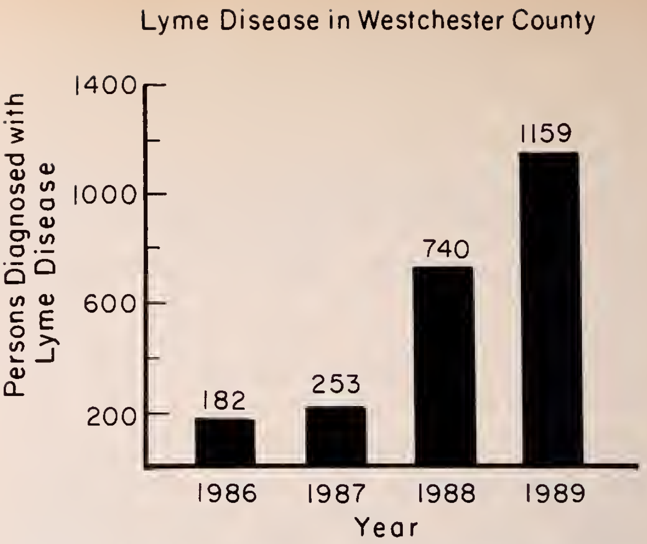

FIGURE 1. Persons with confirmed cases of Lyme disease in Westchester County from 1986 through 1989. Cases were reported and then confirmed by the Westchester County Department of Health after physician contact.

TABLE I. Characteristics of the Population Under Study

Study Parameter	*n*	Percentage	95% Confidence Interval
Historical parameters			
Prior tick bite	25	21.9	14.3–29.5
Prior clinical diagnosis of Lyme disease	8	7.0	2.3–11.7
Seropositivity at beginning of study	7	6.1	1.7–10.5
Prior clinical diagnosis plus seropositivity at beginning of study (overall prevalence)*	10	8.7	3.5–13.9
Study findings, May through September of 1989			
Tick bite	20	17.5	10.5–24.5
Clinical diagnosis	3	2.6	0–5.5
Seroconversion	1	0.8	0–2.5
Clinical diagnosis plus seroconversion (overall incidence)*	3	2.6	0–5.5

*Each individual counted only once.

Other methodologic differences among the various investigations include year and duration of study, case definition of Lyme disease, and type of serologic testing. Nevertheless, the overall results are quite similar. The incidence of new Lyme disease cases at the various sites and time periods ranged from 0.7% to 10%, with possibly a significant difference between the Chappaqua site and the others found only for the years 1984 and 1985 at the Ipswich site.

In contrast with the other reports, we found that the number of hours spent outdoors per week in recreational activity directly correlated with the development of Lyme disease. Greater variability in recreational practices of residents of a suburban residential community compared with that of a summer resort area may account for this discrepancy. Alternatively, differences in the endemicity of *B burgdorferi* infection in ticks and wildlife in different geographic settings may be the explanation.

The incidence and prevalence of Lyme disease in the present study must be interpreted cautiously for several reasons. Some asymptomatic seronegative participants may eventually develop manifestations of Lyme disease from an infection acquired during the 5-month study period. More importantly, only 26% of the estimated study population agreed to participate. The results of the additional questionnaire solicitation to non-participating households in December 1989, however, suggests that the findings based on the study cohort are likely to be representative of the entire population in the study site. Furthermore, we cannot exclude the possibility that the study participants with Lyme disease contracted it outside of the study site.

Further studies will be needed to determine if the findings of the study can be extrapolated to other areas of Westchester County with similar or different ecology or to other residential communities endemic for Lyme disease.

TABLE II. Comparison of Epidemiological Studies of Lyme Disease

						Prevalence			Incidence		
Study	Ref.	Location	Number of Participants	Year	Duration of Observation	Baseline Clinical Diagnosis	Baseline + Serology	Overall*	Clinical Diagnosis	Seroconversion During Study	Overall*
Hanrahan, et al	5	Fire Island, New York	200	1982	July–October	7.5% (4.1–10.9)[†]	8.5%[‡] (4.6–12.4)	13.0% (8.3–17.7)	1.0% (0–2.4)	1.5%[‡,§] (0–3.2)	1.5% (0–3.2)
Steere, et al	6	Great Island, Massachusetts	162	1980	12 mo	11.1% (6.3–15.9)	—	—	0.7% (0–2.1)[‖]	—	—
				1981	12 mo	—	—	—	2.2% (0–4.7)	—	—
				1982	12 mo	—	—	—	1.5% (0–3.6)	—	—
				1983	12 mo	—	—	—	1.6% (0–3.7)	—	—
Lastavica, et al	7	Ipswich, Massachusetts	190	1984	12 mo	—	—	—	—	—	10[‖,¶]
				1985	12 mo	—	—	—	—	—	10
				1986	12 mo	—	—	—	—	—	5
				1987	12 mo	—	—	—	—	—	4
Present study		Chappaqua, New York	114	1989	May–Sept.	7.0% (2.3–11.7)	6.1% (1.7–10.5)	8.7% (3.5–13.9)	2.6% (0–5.5)	0.8% (0–2.5)	2.6% (0–5.5)

*Each individual counted only once. See methods for definitions of terms.

[†] [95% confidence interval is given within parenthesis throughout table.]

[‡] These percentages are slightly different from those reported, because all participants not tested were assumed to be negative, rather than excluded, for a minimal estimate, to correspond with the present study.

[§] One out of the three individuals was an asymptomatic seroconverter.

[‖] These percentages are as reported in the original studies in which the denominator was reduced by the number of individuals previously infected.

[¶] Confidence intervals are not provided, because the original report did not provide absolute numbers for the population at risk.

Acknowledgments. The authors thank Susan Bittker, MS, Denise Cooper, BS, and Lee Kreilsheimer, BSMI, for technical assistance; William Kopp for providing supplies; Dr Durland Fish and Dr Russell Kamer for helpful discussion; Dr Ben Mojica at the Westchester County Department of Health for providing data for Figure 1; and Dr Louis A. Magnarelli for serologic testing and critical review. The authors also wish to thank Mrs Michelle Obenauer for help in preparing the manuscript.

REFERENCES

1. Lyme disease supplemental data from DOH-392 (6/90), New York State Department of Health, Bureau of Communicable Disease, Control Arthropod-Borne Disease Program, ESP, Corning Tower, Room 651, Albany, NY 12239-0627.
2. Magnarelli LA, Meegan JM, Anderson JF, et al: Comparison of an indirect fluorescent-antibody test with an enzyme-linked immunosorbent assay for serological studies of Lyme disease. *J Clin Micro* 1984; 20:181–184.
3. Magnarelli LA, Anderson JF: Enzyme-linked immunosorbent assays for the detection of class-specific immunoglobulins to *Borrelia burgdorferi*. *Am J Epidemiol* 1988; 127:818–825.
4. Colton T: *Statistics in Medicine,* 1st ed. Boston, Little, Brown and Company, 1974.
5. Hanrahan JP, Benach JL, Coleman JL, et al: Incidence and cumulative frequency of endemic Lyme disease in a community. *J Infect Dis* 1984; 150:489–496.
6. Steere AC, Taylor E, Wilson ML, et al: Longitudinal assessment of the clinical and epidemiological features of Lyme disease in a defined population. *J Infect Dis* 1986; 154:295–300.
7. Lastavica CC, Wilson ML, Berardi VP, et al: Rapid emergence of a focal epidemic of Lyme disease in coastal Massachusetts. *N Engl J Med* 1989; 320:133–137.

Marital status and stage at diagnosis in cancer

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ABSTRACT. The association of marital status and stage of diagnosis in cancer was examined for seven common disease sites. Data were drawn from the New York State Cancer Registry for Brooklyn residents diagnosed between 1978 and 1982. Married persons were found to have a better chance of early diagnosis only for cancers of the breast and prostate. When the analysis was stratified for race, this advantage was limited to whites. Married whites were 1.8 times more likely to be diagnosed with early prostatic cancer ($p < 0.01$) and 1.2 times more likely to be diagnosed with early breast cancer ($p < 0.05$). The earlier stage of diagnosis may be due to differences in a complex of social, economic, behavioral, and biological factors. These results suggest the need for additional educational and screening programs for the unmarried.

(NY State J Med 1992; 92:8–11)

This paper examines the relationship between marital status and stage of diagnosis of cancer. There are few such published studies, in spite of a relatively large literature on the relationship between marital status and mortality dating back to the middle of the 19th century, and extensive studies of the relationship between cancer mortality and incidence with marital status.[1,2] Generally, the literature indicates that the married fare better than the non-married. Married persons have been reported to be at a lower risk for cancer at many sites.[3] One recent large study has found that married persons were also more likely to be diagnosed at local stages for many sites and fare better in terms of treatment and survival.[4] Since early diagnosis has a significant impact on treatment and survival, it is important to verify whether in fact marital status is associated with stage of diagnosis and, if so, at what sites. This question is of particular importance for the practitioner, either in the individual health evaluation setting or in community intervention programs.

METHODS

This study used cancer incidence data for the population of Brooklyn, NY for the 4-year period 1978–1982. The data were collected by the New York State Cancer Registry, a population-based registry, and include information on marital status and stage of disease for many cancer sites at the time of diagnosis, as well as sex, race, and age.

The cancer sites chosen were female breast, cervix, ovary, corpus uteri, prostate, colon, and rectum. They were selected for analysis on the basis of the availability of data, frequency of occurrence, and feasibility of screening. Marital status was grouped into three categories: never married; married; and divorced, widowed, or separated (hereafter referred to as formerly married).

There has been some controversy regarding the best way to categorize marital status.[5] There are arguments against combining widowed with divorced and separated, on the basis of differences in life events. In the first instance, a person loses a partner, usually in middle age or old age, because of a particular external factor. In divorce or separation, however, it is not the intervention of an outside factor, but rather the inability to sustain marriage that causes the cessation of married life. It has been noted, however, that some widows will go on to remarry, thus diminishing the significance attached to separation of the two sub-categories.[6]

Stage of disease was grouped into localized, regional, and distant. Cases with in situ disease or unknown stage were excluded.

Cases unknown for sex, and all persons less than 20-years old, were excluded. Unless otherwise specified, we used four age groups: 20–39, 40–59, 60–74, 75 years and older. Comparisons by race were limited to blacks versus whites, with Hispanics included among the whites.

Associations were first examined in multiple contingency tables. Loglinear models were used to examine simultaneously the impact of marital status on stage, controlling for gender and age. A backward elimination approach was used.[7] Two steps in this analysis are compared and reported. First are the results for the best reduced model, which includes the interaction of marital status and stage. We call this model one. The second model is exactly like the first, except that it excludes the interaction between marital status and stage. We call this model two. The significance of the association between marital status and stage was based on the contribution of their interaction to the fit of the model and was measured by the difference between the two models.

Odds ratios (ORs) for marital status were also calculated for the best fitting loglinear model for each cancer site. In fitting the loglinear models, marital status and stage were grouped into three categories as described earlier. For the purpose of comparison with other studies,

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ORs were calculated using married versus never married and local stage versus non-local groupings. Finally, to examine the effect of race, the analysis was stratified for whites and blacks.

RESULTS

The distribution of marital status for various cancers is given in Table I. Persons with unknown marital status comprised anywhere from 1.7% to 7.4% of the cases, depending on the site, and were excluded from the analysis. The distribution of never married ranged from 9.7% (prostate cancer) to 17.3% (cervical cancer); the proportion of married varied from 35.7% (rectal cancer, females) to 70.7% (rectal cancer, males); the formerly married ranged from 15.4% (rectal cancer, males) to 49.9% (rectal cancer, females).

Figures 1 and 2 show the distribution of stage by marital status for various cancer sites. The proportion of patients diagnosed with localized disease (stage 1) varied from 27.1% (ovary) to 74.8% (corpus). The percentages of localized cases by marital status ranged from 30.7% (ovary) to 59.3% (cervix) for never married; 29.6% (ovary) to 77.5% (corpus) for married, and 22.5% (ovary) to 71.2% (ovary) for formerly married.

The loglinear analysis revealed that there were no significant associations between marital status and stage of diagnosis, except for cancers of the prostate and breast. ORs of diagnosis at later stages of disease did not differ from unity for cancers of colon, ovary, corpus, rectum, and cervix. The ORs for early diagnosis of cancer of the prostate (OR = 1.7) and cancer of the breast (OR = 1.2) indicated that married persons were more likely than the never married to be diagnosed with local disease (Table II).

When the analysis was stratified by race, the results indicated statistically significant differences between white married and white unmarried persons with prostate cancer (OR = 1.8) and breast cancer (OR = 1.2). For blacks, the ORs of local to advanced disease at each site did not differ by marital status (OR = 1.0 for married versus never married persons) (Table III).

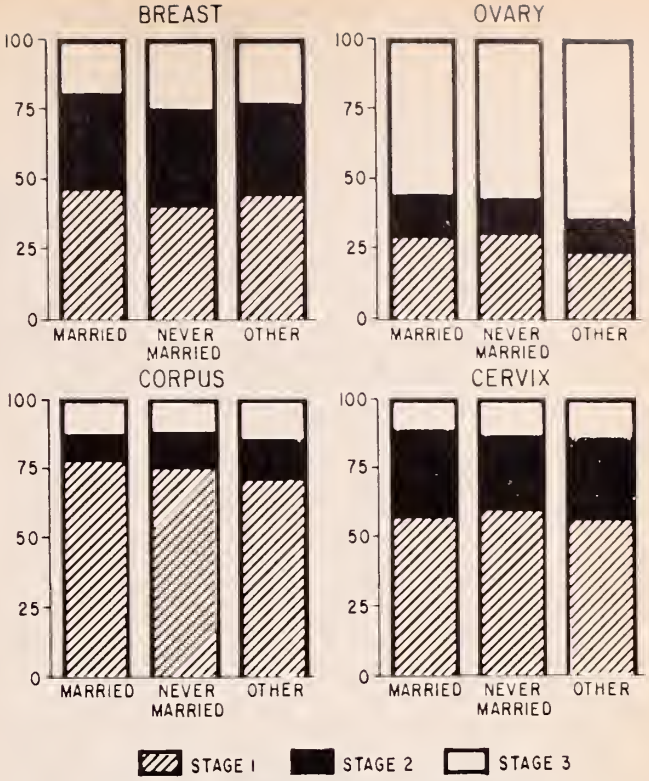

FIGURE 1. Distribution of stage by marital status

DISCUSSION

Is marital status an important variable in early detection of cancer? So far, the limited research on this question has not provided a clear answer. A recent extensive study by Goodwin et al[4] explored the effects of marital status on the diagnosis, treatment, and survival of 27,779 cancer patients with a variety of cancer sites. Combining all sites, the unmarried fared worse than their married counterparts. Unmarried persons were more likely to be diagnosed with advanced stage (OR = 1.19 for non-local versus local; 95% confidence limit 1.12–1.25). After adjusting for stage, unmarried persons were more likely to be untreated for cancer. Finally, after adjustment for treatment and stage, unmarried persons had poorer survival. These results were irrespective of race, with dichotomous categorization of marital status as married versus unmarried.

TABLE I. Distribution of Marital Status by Cancer Site

	Never Married	Married	Other	Unknown	Total
Prostate	273	1,843	655	57	2,828
	(9.7%)	(65.2%)	(23.0%)	(2.0%)	(100%)
Breast	674	2,328	1,951	116	5,069
	(13.3%)	(45.9%)	(38.5%)	(2.3%)	(100%)
Ovary	127	378	311	22	838
	(15.2%)	(45.1%)	(37.1%)	(2.6%)	(100%)
Corpus	160	506	371	41	1,078
	(14.8%)	(46.9%)	(34.4%)	(3.8%)	(100%)
Cervix	150	314	337	64	865
	(17.3%)	(36.3%)	(39.0%)	(7.4%)	(100%)
Colon					
males	226	1,519	386	39	2,170
	(10.4%)	(70.0%)	(17.8%)	(1.8%)	(100%)
females	297	859	1,156	45	2,357
	(12.6%)	(36.4%)	(49.0%)	(1.9%)	(100%)
Rectum					
males	120	699	152	17	988
	(12.1%)	(70.7%)	(15.4%)	(1.7%)	(100%)
females	101	313	438	25	877
	(11.5%)	(35.7%)	(49.9%)	(2.9%)	(100%)

Findings by Goodwin et al[4] on the relationship between marital status and stage of diagnosis for all sites combined, however, does not apply to some specific sites. Unmarried status was a risk factor for diagnosis of advanced cancers of the rectum, breast, cervix, ovary, bladder, and kidney, as well as melanoma; but not for cancers of the uterus, stomach, lung, colon, or prostate. Goodwin et al noted that their results applied basically to whites and Hispanics because there were few blacks in the population.

The present study, although including fewer sites, had a more detailed categorization of marital status and stage and included data on blacks. The results showed that in the Brooklyn population, unmarried persons were not at greater risk of diagnosis at advanced stages for cancers of the cervix, corpus, ovary, colon, and rectum.

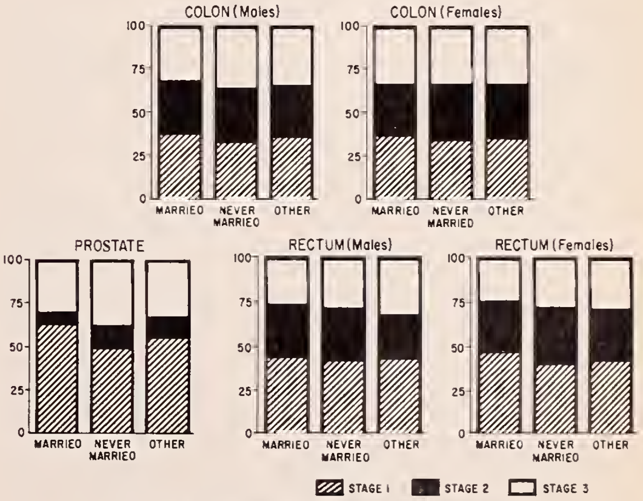

FIGURE 2. Distribution of stage by marital status

TABLE II. Summary Results of Loglinear Analysis Study of Effect of Marital Status on Stage of Diagnosis

Site of Cancer	χ^2	Degrees of Freedom	p value[†]	Goodness of Fit[§]	Odds Ratio[‡]
Prostate					
One	3.18	8	0.922		
Two	25.22	12	0.014		
Two-one	21.04	4	0.0	+[§]	1.7
Breast					
One	9.54	12	0.643		
Two	22.89	16	0.112		
Two-one	13.35	4	0.010	+	1.2
Colon					
One	27.52	20	0.121		
Two	32.64	24	0.112		
Two-one	5.12	4	0.275	–	1.0
Ovary					
One	9.01	8	0.342		
Two	10.67	12	0.558		
Two-one	1.66	4	0.798	–	0.9
Corpus					
One	10.19	8	0.252		
Two	13.10	12	0.362		
Two-one	2.19	4	0.701	–	1.0
Rectum					
One	17.50	20	0.620		
Two	24.26	24	0.447		
Two-one	6.76	4	0.149	–	1.0
Cervix					
One	23.03	8	0.003		
Two	26.10	12	0.010		
Two-one	2.99	4	0.560	–	0.9

[*] The age grouping varies due to the number of cases available for analysis. We have used four age categories for breast and ovary: 20–39 years, 40–59 years, 60–74 years, and 75 years and older. For colon, corpus, prostate, and rectum we have combined the first two and for cervix the last two categories.

[†] The first *p*-value is for the best reduced model; the second *p*-value is for the reduced model excluding the interaction of marital status and stage; the third *p*-value is for the interaction of marital status and stage.

[‡] For purpose of comparison, odds ratios are calculated for dichotomies of married versus never-married for local versus non-local disease.

[§] + indicates significant; – indicates not significant.

TABLE III. Summary Results of Loglinear Analysis Study of Effect of Marital Status on Stage of Diagnosis by Race

Cancer Race*	χ^2	Degrees of Freedom	p value[†]	Goodness of Fit[‖]	Odds Ratio[‡]
Prostate/whites[§]					
One	4.63	8	0.797		
Two	28.43	12	0.005		
Two-one	23.80	4	0.000	+	1.8
Prostate/blacks					
One	3.22	8	0.920		
Two	3.38	12	0.992		
Two-one	0.16	4	0.997	–	1.0
Colon/whites					
One	23.53	20	0.263		
Two	25.79	24	0.364		
Two-one	2.26	4	0.688	–	1.0
Colon/blacks					
One	19.63	20	0.481		
Two	23.18	24	0.509		
Two-one	3.55	4	0.470	–	1.0
Breast/whites					
One	11.13	12	0.518		
Two	20.98	16	0.179		
Two-one	9.85	4	0.043	+	1.2
Breast/blacks					
One	11.24	12	0.508		
Two	12.83	16	0.685		
Two-one	1.59	4	0.811	–	1.0
Ovary/whites					
One	6.96	8	0.540		
Two	10.34	12	0.586		
Two-one	3.38	4	0.496	–	1.1
Ovary/blacks					
One	6.78	8	0.560		
Two	10.03	12	0.613		
Two-one	3.25	4	0.517	–	1.1
Corpus/whites[¶]					
One	3.66	4	0.454		
Two	6.94	8	0.544		
Two-one	3.28	4	0.520	–	1.2
Corpus/blacks					
One	1.81	4	0.771		
Two	4.01	8	0.856		
Two-one	2.2	4	0.699	–	1.1
Rectum/whites[#]					
One	3.26	4	0.515		
Two	6.23	8	0.622		
Two-one	2.97	4	0.563	–	1.0
Rectum/blacks					
One	3.64	4	0.458		
Two	5.84	8	0.665		
Two-one	2.20	4	0.699	–	1.1
Cervix/whites					
One	12.88	4	0.012		
Two	14.79	8	0.063		
Two-one	1.91	4	0.752	–	1.0
Cervix/blacks					
One	2.93	4	0.570		
Two	4.72	8	0.787		
Two-one	1.82	4	0.769	–	1.0

[*] The age grouping varies due to number of cases available for analysis. We have used four age categories for breast and cervix: 20–39 years, 40–59 years, 60–74 years, and 75 years and older. For colon, ovary, and prostate, we have used three age groupings; combining the first two categories. For corpus, rectum, and cervix, we have combined the first and the last two age groups.

[†] The first *p* value is for the best reduced model; the second *p*-value is for the reduced model excluding the interaction of marital status and stage; the third *p*-value is for the interaction of marital status and stage.

[‡] For purpose of comparison, odds ratios are calculated for dichotomies of married to never-married for local versus non-local disease.

[§] The race category whites includes Hispanics.

[‖] + indicates significant; – indicates not significant.

[¶] The odds ratios reported for the site are not adjusted for age because of small numbers.

[#] For rectal cancer, due to relatively small number of cases we did not control for gender.

Discrepancies in studies of marital status and stage of diagnosis in cancer can result from differences in definitions, staging techniques, and characteristics of the populations under investigation. One problem with using marital status as a predictive variable is that the concept of a nuclear family varies over time and across social classes and ethnic groups. This problem is more accentuated in a varied population, such as in Brooklyn where many ethnic groups reside. Married persons may also be said to enjoy more social and economic advantages. It may be that they are encouraged to seek help sooner or that they have better access to the health care system. This is corroborated by studies suggesting that early detection of cancer depends on factors such as access to a family physician, medical check up, knowledge of cancer, screening programs, and lack of disabilities.[8,9]

Another problem in using marital status as a predictive variable is selection bias. In a major study, Lilienfeld et al[10] have found that for all neoplasms except leukemia, there is a higher mortality for single, widowed, and divorced persons than for the married. However, ratios of age-adjusted death rates by marital status for cancer of the prostate were lower among single males among both whites and blacks. Furthermore, for cancer of the breast, the death rate for widowed white females was similar to that of the married.

Such findings led to the formulation of a complex hypothesis regarding marital status and cancer. High mortality rates were said to reflect high incidence rates. The latter was thought to be higher among non-married persons, reflecting selective factors preventing those predisposed to illness or death from marrying, or in the case of widowed or divorced persons, from remarrying. The presumption was that observed mortality patterns might reflect more about the kind of people who enter the various marital states than about any intrinsic influences those states have on disease risk.

The selection factors associated with marital status seem less important in studies of incidence of cancer. Thus, Ernster et al[3]

TABLE IV. Distribution of Marital Status by Race for Cancers of Breast and Prostate

	White No.	Percentage	Black No.	Percentage
Breast				
Never married	479	12.0	188	20.3
Married	2,002	50.0	324	35.0
Widowed, Divorced, or Separated	1,525	38.0	413	44.7
Total	4,006		925	
	$\chi^2 = 82.77$			
	$p = 0.001$			
	Degrees of freedom = 2			
Prostate				
Never married	118	5.6	309	47.2
Married	1,452	69.4	88	13.5
Widowed, Divorced, or Separated	452	21.6	257	39.3
Total	2,092		654	
	$\chi^2 = 877.1$			
	$p = 0.001$			
	Degrees of freedom = 2			

found excess cancer rates across most cancer sites and age groups for single black males, consistently high rates for cancer of the lung and bronchus in divorced white males and in single black females, low rates for the hormone-dependent reproductive tumors (prostate, breast, uterine, corpus, and ovary) in separated white males and females, and high rates for cervical cancer among separated white women. Marital status patterns, where found, frequently differed between whites and blacks and between males and females. Somewhat contrary to earlier studies, married persons did not stand out sharply as a lower risk group with respect to cancer incidence.

It is surprising that the results of marital status studies differ for incidence and mortality, since mortality is influenced by stage of disease at diagnosis, treatment, disease duration, reporting, and so forth.

Marital status, therefore, usually serves as a surrogate for a series of economic, social, cultural, and behavioral variables. As such, the significance of marital status for early diagnosis of cancer should be evaluated in the light of the specific study population. Thus, the present study showed a significant difference among the blacks and whites in marital status for cancers of the breast and prostate (Table IV).

Irrespective of the nature of the association of marital status and stage at diagnosis of cancer, the current data indicate that unmarried whites are at greater risk of late diagnosis compared with married whites for two very common cancers, breast and prostate. This should be taken into account in screening strategies to promote preventive measures against cancer.

References

1. Ortmeyer CF: Variation in mortality, morbidity, and health care by marital status, in Erhardt LL, Berlin JE (eds): *Mortality and Morbidity in the United States,* Cambridge, Harvard University Press, 1974, pp 159–184.
2. Berkson J: Mortality and marital status: Reflections on the derivation of etiology from statistics. *Am J Public Health* 1962; 52:1318–1329.
3. Ernster VL, Sacks ST, Selvin S, et al: Cancer incidence by marital status: US third national cancer survery. *J Nat Cancer Inst* 1979; 63:567–585.
4. Goodwin JS, Hunt WC, Key CR, et al: The effect of marital status on stage, treatment, and survival of cancer patients. *JAMA* 1987; 21:3125–3130.
5. Davies JM: Is testicular cancer incidence related to marital status? *Int J Cancer* 1981; 28:721–724.
6. Susser M: Widowhood: A life stress or a stressful life event? *Am J Public Health* 1981; 71:793.
7. Wilkinson L: SYSTAT: The system of statistics. Evanston, IL, SYSTAT, Inc, 1986.
8. Samet JM, Hunt WC, Goodwin JS: Determinants of cancer stage. A population-based study of elderly new Mexicans. *Cancer* 1990; 66:1302–1037.
9. Mor V, Masterson-Allen S, Goldberg R, et al: Pre-diagnostic symptom recognition and help seeking among cancer patients. *J Community Health* 1990; 15:253–266.
10. Lilienfeld AM, Levin ML, Kessler II: Mortality and marital status, in *Cancer in the United States,* Cambridge, MA, Harvard University Press, 1972.

National Practitioner Data Bank

JAMES ROBB

In 1986 the Health Care Quality Improvement Act provided the legislative authority necessary to establish the National Practitioner Data Bank. The basic aim of the Data Bank is to accumulate and dispense information on the professional conduct and competency of physicians, dentists, and other health care practitioners, and as a consequence, improve the quality of health care by identifying practitioners who engage in incompetent and unprofessional behavior. After a long period of decision and substantial delays, in January 1989 a five year, $15.9 million contract was awarded to Unisys Corporation to establish and operate the Data Bank in conjunction with officials from the Department of Health and Human Services (DHHS), Health Resources and Services Administration (HRSA).

The Data Bank opened on September 1, 1990, in spite of a report from the United State General Accounting Office (GAO) that questioned the Data Bank's ability to protect the confidentiality of information in the system. In addition, the GAO questioned the uncertain nature of the ultimate cost of the project and the ability of the HRSA to manage effectively such a complex project.

Organized medicine has been deeply involved in the evolution of the Data Bank and has provided an ongoing critique of its operation, planning and policies. Historically, carriers providing malpractice insurance in New York State are mandated to report *all* claims and lawsuits to the New York State Department of Health on a very detailed form known as Department of Health 787 form. This form is completed on all cases without regard to whether or not a payment is made. Out of sight, out of mind is the adage that seems to apply. To appreciate and understand practitioner concerns about the Data Bank it is necessary to review briefly the major reporting provisions contained in this federal program.

REPORTING REQUIREMENTS

Basically, four groups are responsible for reports to the Data Bank. Medical malpractice insurers, hospitals, and other health care entities, state medical and dental boards, and professional societies involved in peer review. In addition, self-insured physician, osteopath, dentist, or self-insured entity, including a hospital, as well as state and local governments which make a payment in satisfaction of a judgment or settlement of a malpractice claim, must report the information to the Data Bank on the approved form. In the case of medical malpractice payments, reports must be sent simultaneously to the Data Bank and to the appropriate State Licensing Board in the state in which the malpractice claim occurred. In New York State, a duplicate report is sent to the New York State Education Department, Office of Professional Discipline. With regard to defining "Health Care Entities," hospitals are included in this group, as are Health Maintenance Organizations (HMOs) licensed by a state or determined to be qualified as such by the US DHHS. Also included are any group or prepaid medical or dental practice that provides health care services and engages in professional review activity through a formal peer review process for the purpose of furthering quality health care. Professional societies that are composed of physicians, dentists, or other health care practitioners at national, state, or local levels, that have a formal peer review program for the purposes of furthering quality health care, and that are engaged in professional review activity, are defined as health care entities. In addition, federal groups such as the Veterans Administration and the Department of Defense are responsible for reporting to the Data Bank with regard to actions taken on cases involving health care provided to armed services members and families.

WHAT MUST BE REPORTED

It is important to note that the reporting requirement in all cases is prospective only and involves actions taking place on or after September 1, 1990. The Data Bank collects and releases to eligible parties four major categories or reportable actions as they relate to the professional competence and conduct of health care practitioners. The major reportable actions are medical malpractice indemnity payment, adverse licensure actions taken by State Medical and Dental Boards on the basis of professional competence or conduct, adverse actions on clinical privileges, and adverse actions on professional society memberships.

Regarding medical malpractice payments, any insurer or entity that makes an indemnity payment on behalf of a practitioner as a result of a *written claim* must file a report with the Data Bank. This includes payments made by virtue of a self-insurance plan. Only indemnity payments are reportable, and expenses associated with the defense of a case are not included in reporting requirements. A waiver of debt is not considered a payment and as such is not reportable to the Data Bank. At the present time there is no minimum threshold for reporting payments on malpractice claims to the Data Bank. However, a number of groups including the American Medical Association and the Physician Insurers Association of America have been working with the Data Bank by providing to the federal government statistical information, which will hopefully result in the establishment of a reasonable financial threshold for the reporting of malpractice claims. This would eliminate the reporting of a large number of small settlements.

Hospitals are required to report actions affecting clinical privileges including actions taken against interns and residents. Professional review actions taken by a hospital or health care entity that adversely affect the clinical privileges of a physician, dentist, or osteopath for a period of longer than 30 days must be reported to the Data Bank. The adverse actions against physician privileges that must be reported include reduction, restriction, suspension, revocation of dental or clinical privileges on the basis of the practitioner's professional competence or conduct. Acceptance of a surrender of clinical privileges by a physician or a dentist is also reportable if the individual is under investigation on a matter relating to possible incompetence or improper professional conduct, or in return for not conducting an investigation. Professional society actions that are reportable are actions resulting from a formal peer review process within the professional society based on a practitioner's professional competence or conduct in relation to the care and treatment of patients. An adverse action taken by a professional society as defined by the Data Bank means reducing, restricting, suspending, revoking, or denying membership; it does not include censures, reprimands, or admonishments.

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WHEN MUST ACTIONS BE REPORTED

A report on a medical malpractice payment or adverse licensure action must be submitted to the Data Bank within 30 days of the date of payment or of the date that the licensure or disciplinary action was signed by an authorized official. Reports on adverse actions on clinical privileges and society memberships must be reported to the applicable State Medical or Dental Board within 15 days after the adverse action report assigned by an authorized official. The State Board then has an additional 15 days in which to report the action to the National Practitioner Data Bank.

REPORTING PROCESS

Once a report of either a payment of adverse action is received by the Data Bank, the form is reviewed to determine if all required information has been submitted. Incomplete forms are immediately returned to the reporting entity. Complete forms receive a unique document number from the Data Bank and are entered into the Data Bank's computer system. To enhance the accuracy of information in the Data Bank, each report is held in suspense for 30 calender days after receipt. During this time the information contained in the report is not released to any party other than the practitioner who is the subject of the report and the reporting entity. This is done by way of a computer-generated report known as a Practitioner Notification Document. This suspense period is designed to allow for a review of the information by both the health care practitioner and the reporting entity in order to correct any misinformation or to review any disputes. The reporting forms do not contain any personal or demographic information on the patient. Most of the form is dedicated to identifying the practitioner involved, describing the incident that gave rise to the claim and information concerning the adjudicative body and financial information on the case. Adverse action reports basically use a similar reporting format with most of the form dedicated to a description of the act or omissions that resulted in the adverse action taken against the health care practitioner.

The law requires, as part of the reporting format, a "brief description of the acts of omissions and injuries or illnesses on which the action or claim was based." Also required is a "malpractice claims description code." The federal government has adapted these codes from the Harvard Risk Management Foundation Allegations of Negligence. Although the regulations in the enabling legislation state that "a payment in settlement of a medical malpractice action or claims shall not be construed as creating a presumption that medical malpractice has occurred," some practitioners have been concerned over the general accusatory nature of these descriptive codes. In addition, practitioners have raised questions about the accuracy and objectivity of the information provided in that portion of the report designed to describe the acts of omissions. It appears that the great majority of reports have been accepted as submitted and have not been disputed by the health care practitioner. Insurance companies in general have recognized the possibility of a legitimate dispute and have been working with the health care practitioner to resolve any questions to the satisfaction of all parties. There is a formal, albeit cumbersome, dispute resolution process mandated and detailed in the practitioner notification document sent out by the Data Bank. One of the sources of conflict that has recently been experienced seems to arise from a misunderstanding on the part of the practitioner concerning the nature of information contained in the portion of the report describing the acts or omissions. It should be kept in mind that the information portion is designed to describe the acts of omission giving rise to the claims only. It is not to be used as a form for blanket denials or to raise questions concerning the legitimacy of a plaintiff's assertions in a medical malpractice claim. Above all, the practitioner should follow the instructions carefully whenever notification documents are received if he/she intends to dispute the report. The practitioner must stipulate in writing on the notification document the basis of his/her dispute and also contact the reporting entity to discuss the situation. If a reporting entity refuses to modify its report, the health care practitioner may request that the Secretary of the DHHS review information submitted by both parties. It will be up to the Secretary to determine whether or not the report is accurate. If the Secretary determines that the report is accurate as filed, the physician will be permitted to place a brief statement regarding the dispute in the file, and all entities entitled to receive a report will receive not only the report, but also any dissenting statement by the health care practitioner. Regarding medical malpractice payment reports, most carriers are concerned about this situation and have taken steps to minimize the number of reports that will enter into "disputed" status. Some carriers are using senior personnel within the Claims Department to complete this sensitive part of the form.

REQUESTING INFORMATION

An insurance carrier cannot request information from the Data Bank. Practitioners can receive copies of their complete file at any time, free of charge, by submitting a Request for Information Form to the Data Bank. Health care practitioners are not automatically notified when a report is released on them in response to an inquiry but may periodically obtain this information by requesting a copy of their Data Bank file. Practitioners are only notified when they are the subject of a report *received by* the Data Bank. HMOs and group medical practices that qualify as entities under the Data Bank plan because of their professional and formal peer review activity can query the Data Bank. Hospitals must query the Data Bank when a physician, dentist, or other qualified health care practitioner applies for a position on the medical staff or applies for a change in clinical privileges. Hospitals must also query the Data Bank when recredentialing members of its medical staff. The initial inquiry must be done no later than two years after the opening of the Data Bank and every two years thereafter.

Under certain specific circumstances, a plaintiff or a plaintiff's attorney is permitted to obtain information from the Data Bank. For the data to be released, a medical malpractice claim must be filed against a hospital or health care entity in a state or federal court or other adjudicative body, and in addition, the specific physician, dentist, or other health care practitioner for whom the information is sought must also be named in the action. Plaintiffs must submit evidence to the DHHS demonstrating that the hospital defendant failed to make a mandatory inquiry to the Data Bank regarding the practitioner against whom the plaintiff filed the action. This evidence is not available to the plaintiff through the Data Bank itself, but the plaintiff must substantiate through the litigation discovery process that the hospital has failed to request information on the practitioner from the Data Bank. It is contemplated that in the future, information accumulated by the Data Bank may be released in its aggregate form to the public. The information on individual practitioners would of course not appear in such a report.

CURRENT STATUS OF OPERATIONS

As of early February 1991, the Data Bank had received 9,818 malpractice payment and adverse action reports and 305,126 inquiries. The Data Bank was experiencing significant delay in processing these. In addition, more than 300 disputes had been filed with the Data Bank, which is actively working on establishing a workable and coherent dispute resolution process. Because of the report by the GAO regarding deficiencies in the operation of the Data Bank, it is also expected that funding for the current fiscal year may be held up until such time as the deficiencies have been addressed and corrective action undertaken. It would appear from the data submitted to date that the establishment of a reporting threshold for small claim payments would be a significant step in reducing the number of reports submitted to the Data Bank and the consequent backlog.

REFERENCE

1. Title IV, Public Health Law 99-660, The Health Care Improvement Act of 1986.

REVIEW ARTICLE

Update in epilepsy

Part I: Diagnosis and treatment of epilepsy

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Epilepsy is a disorder characterized by recurrent seizures. It is fairly common, with an incidence in the United States estimated to be 48.7 per 100,000 per year and a prevalence of 6.0 per 1,000.[1] However, this is likely a minimum estimate because of the inherent difficulties in epidemiological studies,[2] and 1% may be a more appropriate estimate of prevalence in the general population. The onset of epilepsy is most common in childhood.[3] The diagnostic and therapeutic principles outlined in this review apply to adults as well as children. Seizures in the neonatal period are beyond the scope of this article, and they are usually managed by a neonatologist.

PATHOPHYSIOLOGY AND CLASSIFICATION

A seizure occurs when neurons of the cerebral cortex become "irritable" and fire in repeated bursts.[4] When these bursts involve a large aggregate of neurons, a clinically detectable seizure occurs. This usually requires recruitment of many normal neurons into the abnormal firing pattern. The electrical activity occurring during a seizure is usually evident on an electroencephalogram (EEG) taken at the same time. This EEG activity is the hallmark of a seizure and differentiates it from other paroxysmal events. Portions of this activity, the "interictal discharches," can be recorded between seizures. The clinical manifestations of a seizure reflect that area of the brain involved in the abnormal electrical discharges, so that an electrical discharge in the right motor cortex will result in epileptic movements in the left extremities, whereas discharges within the temporal lobe may result in psychosensory phenomena or impairment of consciousness. These are focal (partial) seizures. They may spread to become secondarily generalized; that is, they may involve both sides of the brain. In contrast, the electrical activity in primary generalized seizures involves both sides of the brain simultaneously at onset. The origin of these seizures is not clear. Generalized seizures may be convulsive or nonconvulsive. The classification of epileptic seizures[5] is shown in Table I. In the treatment of childhood epilepsy, the classification of epilepsies and epileptic syndromes[6] is useful for its therapeutic and prognostic implications. This classification is shown in Table II.

DIAGNOSIS AND ETIOLOGY

The diagnosis of epilepsy is established by a clinical history of recurring stereotyped episodes in a pattern appropriate to the function of a local brain area (partial seizures), or to one of the generalized seizure disorders shown in Table I. Usually a seizure lasts from seconds to a few minutes and may be followed by a period of confusion lasting 10–20 minutes. The diagnosis is confirmed by an EEG recording of epileptiform activity, both between and during seizures. The differential diagnosis includes conditions secondary to cardiovascular events, transient ischemic attacks, narcolepsy, psychiatric disorders such as panic attacks, fugue states, pseudoseizures, and episodes of vestibular disorder. Cardiac Holter electrocardiogram (EKG) evaluation, and audiologic examinations may be indicated. In cases where the clinical picture is not typical or poorly reported and the EEG not confirmatory, 24-hour closed-circuit television/electroencephalogram (CCTV/EEG) monitoring may be necessary. CCTV/EEG monitoring has been of most value in selecting patients for surgical therapy and in excluding pseudoseizures.

Once the diagnosis of epilepsy is made, further investigation is necessary to determine its cause. In about one third of patients the cause can be determined.[1,7,8] "Genetic" and idiopathic epilepsy account for the rest. Demonstrable causes of epilepsy include brain lesions secondary to trauma, vascular disease, brain abscess, encephalitis, neoplasm, and metabolic disorders. Perinatal injury and febrile seizures may also account for epilepsy in children. Magnetic resonance imaging (MRI) is superior to computed tomography (CT) scanning in demonstrating structural epileptogenic lesions such as gliomas, hamartomas, arteriovenous malformations (AVMs), and mesial temporal sclerosis.[9] CT scan, however, will demonstrate calcified lesions, which may not be evidenced on MRI.[10] Appropriate laboratory data, including serum chemistries, are examined to rule out metabolic causes of seizures including hyperglycemia, uremia, water intoxication, and hypocalcemia. A urinary metabolic genetic screen is helpful in revealing some rare causes of childhood epilepsy such as phenylketonuria.

TREATMENT

For many patients epilepsy is a relatively benign condition, and readily responds to medical management. However, in a minority it is a serious illness with increased morbidity and mortality,[11,12] as well as increased emotional and psychiatric problems,[13] difficulty with education, employment, and driving. Death is more likely to occur in patients with poorly controlled generalized seizures than in nonepileptics of the same age. The causes due to both accidents caused by seizures and, in a smaller number of patients, from the seizures themselves, possibly by inducing a cardiac arrhythmia.[12,14] There is some evidence that psychosocial difficulties may be correlated with a lifetime number of generalized tonic-clonic seizures or episodes of status epilepticus.[15] Thus, complete control of seizures is the goal of treatment. Patients with generalized seizures, especially primary generalized tonic-clonic convulsions,

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Supported by NIH grant NS17111 and sponsored in part with a grant by New Medico Health Care System.

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TABLE I. Classification of Epileptic Seizures

Nomenclature of the International Classification of Epileptic Seizures	Traditional Nomenclature
I. Partial seizures (seizures beginning locally)	
A. Simple partial seizures (without impaired consciousness)	
1. With motor symptoms	Focal motor seizures, Jacksonian seizures
2. With somatosensory or special sensory symptoms	Focal sensory seizures
3. With autonomic symptoms	
4. With psychic symptoms	Temporal lobe seizures
B. Complex partial seizures (with impaired consciousness)	
1. With impaired consciousness only	(Often confused with absence, or petit mal seizures*)
2. With automatisms	Psychomotor seizures, temporal lobe seizures
C. Partial seizures with secondarily generalization	Focal seizures, such as Jacksonian seizures, progressing to grand mal (major motor) seizures
II. Generalized seizures	
A. Absence and atypical absence seizures	Petit mal seizures
B. Myoclonic seizures	Myoclonic seizures, minor motor seizures
C. Clonic seizures	Clonic seizures
D. Tonic seizures	Tonic seizures
E. Tonic-clonic seizures	Grand mal seizures, major motor seizures
F. Atonic (astatic) seizures	Atonic seizures, minor motor seizures, "drop attacks"
III. Unclassified epileptic seizures	

Modified from ILAE: Proposal for Revised Clinical and Electroencephalographic Classification of Epileptic Seizures, 1981.

*An EEG is required to distinguish complex partial seizures with impaired consciousness only from absence seizures.

TABLE II. Classification of Epilepsies and Epileptic Syndromes

Nomenclature of the International Classification	Some Common Epilepsies and Epileptic Syndromes
I. Lateralization-related (focal, local, partial) epilepsies and syndromes	
A. Idiopathic (with age-related onset)	Benign childhood epilepsy with centrotemporal spike Childhood epilepsy with occipital paroxysms
B. Symptomatic	Chronic progressive epilepsia partialis continua of childhood (Kojewnikow's syndrome) Temporal lobe epilepsies Frontal lobe epilepsies Parietal lobe epilepsies Occipital lobe epilepsies
C. Cryptogenic (lack of etiologic evidence)	
II. Generalized epilepsies and syndromes	
A. Idiopathic (with age-related onset)	Benign myoclinic epilepsy in infancy Childhood absence epilepsy (Pyknolepsy) Juvenile absence epilepsy Juvenile myoclonic epilepsy Epilepsies with grand mal (GTCS) seizures on awakening
B. Cryptogenic or symptomatic (age-related)	West syndrome (infantile spasms) Lennox-Gastaut Syndrome
C. Symptomatic (age-related)	Phacomatoses Phenylketonuria
III. Epilepsies and syndromes undetermined whether focal or generalized	
A. With both generalized and focal seizures	Neonatal seizures Severe myoclonic epilepsy in infancy Acquired epileptic aphasia (Landau-Kleffner syndrome)
B. Without unequivocal generalized or focal features	Grand mal (GTCS) during sleep
IV. Special syndromes	Febrile convulsion Isolated seizures or isolated status epilepticus Seizures occurring only with an acute metabolic or toxic event

Modified from ILAE: Proposal for Revised Classification of Epilepsies and Epileptic Syndromes 1989.

are more likely to achieve remission with medical treatment than those with complex partial seizures (CPS).[16,17] Also difficult to control are patients with myoclonic seizures [18] and multiple seizure types.[19] Initial management of seizure patients includes both treatment of the seizures themselves with antiepileptic drugs (AEDs) and treatment of any underlying cause.

Generally AED treatment is started after the second seizure. However, in adults, there is a tendency to start treatment after the first seizure because of the considerable impact on employment, education, and driving. The risk of recurrence after the first seizure in all age groups is reported to be between 29% and 71% (mean 50%) at a 3-year follow-up.[20–23] Recurrence usually occurs within the first year. After the second seizure, the risk of recurrence is about 80%.[24]

Approximately 70%–75% of patients will have their seizures controlled by AEDs.[16,25] After 2–4 seizure-free years, AEDs may be slowly discontinued. At this point, there is a reported range of 8%–66% (mean 20%) recurrence rate[26] depending on the methodology used. Remission is more likely in patients with idiopathic epilepsy, normal neurologic examination, normal EEG, early onset and generalized seizures.[26] Treatment options for patients not controlled on AED therapy are considered in Part III of this series.

AED selection depends on the seizure type. The AEDs for the particular seizure type are listed in Table III. In making the choice of AEDs, it is important to distinguish between some types of CPS and absence seizures ("petit mal"), both of which result in impairment of consciousness only but require different types of AEDs. Toxicity is a major concern in using AEDs. Although seizures are potentially an intermittent threat to life or well being, adverse reactions may be a continuous burden. The three most common types of AED side effects are idiosyncrasy, metabolic derangement, and dose-related toxicity. Idiosyncratic reactions include skin rash, hepatic dysfunction, and bone marrow suppression. These usually occur in the early phase of AED administration and can sometimes be fatal. Treatment includes immediate recognition and withdrawal of the offending AED and protecting against seizures with another medication. However, in most cases, the changes in the liver enzymes or blood cell count are minor and asymptomatic, and AEDs can be continued but with careful observation. Metabolic side effects include folate deficiency, hypocalcemia, and gingival hypertrophy. Dose-related toxicity may be manifested by diplopia, ataxia, somnolence, and decreased cognitive abilities.

Monotherapy in highest tolerated doses (ie, a dose that will control seizures without producing toxicity) has been shown to be superior to polytherapy.[27–29] Although there may be additive anticonvulsant effects with polytherapy, additional side effects and drug interactions are likely to occur. Therefore, using one drug to the point where seizure control is achieved or toxicity ensues is the beginning of treatment. If that AED does not control seizures, a second AED will be introduced and the first one gradually discontinued. This crossover is accomplished with particular concern for the AED half-lives. The pharmacokinetic characteris-

TABLE III. Drugs of Choice in the Treatment of Epilepsy

Group 1: Partial (focal) seizures and/or generalized tonic-clonic (convulsive) seizures
- Phenytoin (Dilantin®)
- Carbamazepine (Tegretol®)
- Primidone (Mysoline®)
- Phenobarbital (Luminol®)
- Sodium valproate (Depakote®)

Group 2: Primary generalized seizures
- A) Absence (petit mal)
  - Ethosuximide (Zarontin®)
  - Sodium valproate (Depakote®)

  If GTC are associated with absence, a drug from Group I must be given in addition to ethosuximide; however, valproate alone may control both types of seizures.
- B) Myoclonic C) Tonic D) Atonic E) Clonic
  - Sodium valproate (Depakote®)
  - Clonazepam (Klonopin®)

The above drugs may be used as monotherapy. Clorazepate (Tranxene®) may be added to any of the above drugs to enhance their antiepileptic effect, but cannot control seizures when used alone. Adrenocorticotrophic hormone and/or ketogenic diet may be used with other AEDs for treatment of myoclonic, tonic, and/or clonic seizures.

tics and side effects of each AED are listed in Table IV.[30] In some instances, seizures can be controlled by the combination of two or three AEDs.

PHARMACOKINETICS OF AEDs

Pharmacokinetic characteristics of the AEDs provide the basis for drug management. The half-life reflects the rate of drug metabolism and elimination.[31,32] It may be altered by drug interactions (Table V). Some drugs may decrease the metabolism of the AED and therefore increase the half-life or vice versa. AEDs also affect the metabolism of other drugs including antibiotics and anticoagulants.[33] Medical conditions such as renal failure[34,35] or hepatic failure[36] may also alter the AED half-life. Administration of the AEDs should be at intervals no longer than one half-life.

Approximately five to six times the half life is required to reach steady state; and, by the same token, it takes about five to six times the half-life for the drug to be completely eliminated from the body. Evaluation of the level achieved by a particular drug dosage will be apparent at time of steady state. A drug with a long half-life such as phenytoin may require a week or more to reach its steady state, whereas a drug with a short half-life, like carbamazepine, may take only three days.

The therapeutic range is that range below which the drug is usually ineffective and above which it produces toxicity. It varies from patient to patient. Some patients tolerate very high serum drug levels without clinical toxicity, whereas others may become toxic at lower levels. Intermittent toxicity may be experienced during high peak drug levels therefore requiring administration of the drug at smaller doses, but more frequent intervals.

Blood levels of AEDs may be altered by interference with absorption, as well as drug interactions. Drug levels are obtained to promote compliance, determine drug efficacy, establish an appropriate dose after a drug change, and determine which AED in polytherapy is responsible for toxicity. When multiple drugs are used, their additive effects may result in toxicity even when the serum level for each individual drug is within the therapeutic range. Seizure frequency may increase with high serum drug levels, a condition recognized as paradoxical intoxication.[37]

Each AED is protein-bound to a different extent which determines the amount of free drug available. Serum drug levels, as usually measured, reflect the total concentration of the drug, whereas brain levels are usually proportional to the free drug level. The degree of protein binding can change with certain drug interactions. Some drugs such as phenytoin are about 90% bound to proteins. Coadministration of valproic acid or aspirin will result in an increase in phenytoin free level because they compete with phenytoin for protein binding. In altered metabolic states such as uremia[34,35] and burn [38] in which there is low albumin concentration, the free level of phenytoin or other protein bound AEDs will be increased. While the total serum drug level determination is most commonly used in clinical practice, the free level is likely to be more useful in these conditions. Free level of AEDs can be

TABLE IV. Pharmacokinetic Characteristics and Side Effects of Antiepileptic Drugs

Drug	Usual Adult Dosage (mg/day)	Usual Therapeutic Range	Mean Half-life	Some Toxic and Dose Related Side Effects
Carbamazepine (Tegretol®)	800–1,400	6–12 μg/mL	12 hr	Nystagmus, ataxia, rash, blood dyscrasia*
Clonazepam (Klonopin®)	Highly individualized (0.03–0.3 mg/kg/day)	5–50 ng/mL	24 hr	Drowsiness, exacerbation of childhood hyperactivity, withdrawal seizures and status epilepticus if dosage not reduced gradually
Clorazepate (Tranxene®)	30–60	Metabolite: desmethyldiazepam 1–2 μg/mL	36 hr	Few side effects, some sleepiness
Ethosuximide (Zarontin®)	1,000–1,200	40–100 μg/mL	30 hr in children; 60 hr in adults	Gastric distress, nausea dizziness, drowsiness
Phenobarbital	60–240	10–40 μg/mL	4 d	Mental changes, withdrawal seizures if stopped abruptly
Phenytoin (Dilantin®)	200–600	10–20 μg/mL	24 hr	Mental dullness, ataxia, diplopia, gingival hypertrophy, acne, hirsutism
Primidone (Mysoline®)	500–1,500	Primidone, 5–10 μg/mL; metabolite: phenobarbital 10–30 μg/mL	4 d	Emotional and mental changes (including depression, irritability), impotence, withdrawal seizures if stopped abruptly
Valproate sodium (Depakote®)	1,000–3,000	50–100 μg/mL	8 hr	Transient nausea, weight gain, alopecia, fine tremor, potential bleeding problems, liver damage†

Adapted from Ojemann LM, Ojemann GA: *Am Fam Physician* August; 1984 with permission.

*Frequently check hematocrit and white blood cell count.

†Should be avoided in children (especially under age 2) who are on polytherapy; frequently check liver function in all patients.

TABLE V. Some Commonly Encountered Drug Interactions

- Drugs that increase phenytoin level
  - Chloramphenicol (Chloromycetin®)
  - Chlorpromazine (Thorazine®)
  - Dicumarol
  - Disulfiram (Antabuse®)
  - Isoniazid (INH®)
  - Methuximide
  - Phenobarbital*
- Drugs that increase phenobarbital level
  - Valproic acid (Depakene®)—increases phenobarbital level by 33%
  - Dicumarol
- Drugs that decrease phenytoin level
  - Phenylbutazone (Azolid®, Butazolidin®)
  - Carbamazepine (Tegretol®)
  - Phenobarbital*
- Drugs that decrease total phenytoin level but increase free phenytoin level, which augments the effects of phenytoin
- Valproic acid
  - Salicylates
- Drugs that decrease valproic acid effects
  - Phenytoin
  - Phenobarbital
  - Primidone
- Effects of certain antiepileptic drugs on other drugs
  - Phenytoin, carbamazepine, and barbiturates decrease the effects of the following drugs
    - Chloramphenicol
    - Dicumarol
    - Digitoxin
    - Doxycycline (Vibramycin®)
    - Oral contraceptives
    - Phenylbutazone
    - Warfarin (Coumadin®, Panwarfin®)
    - Dexamethosone
    - Quinidine

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*Phenobarbital may decrease or increase phenytoin level.

determined in the dializable portion of the serum, in cerebral spinal fluid, or saliva. Because of the multiple considerations described above, AED management must be tailored to the individual patient. Generic substitution of brand name drugs is generally to be avoided because of the variance of bioavailability of these medications.[39]

Acknowledgments. The authors thank Ms Lee Dulin for her patient secretarial assistance, especially with manuscript revisions, and Ms Jill Andrews for obtaining for obtaining reference articles in a timely fashion.

REFERENCES

1. Hauser WA, Kurland LT: The Epidemiology of Epilepsy in Rochester, Minnesota, 1035 through 1967. *Epilepsia* 1975; 16:1–66.
2. Zielinski JJ: Epidemiology and medical-social problems of epilepsy in Warsaw. Psychoneurological Institute, Warsaw 1974.
3. Hauser WA, Hesdorffer DC: Incidence and Prevalence, in *Epilepsy: Frequency, Causes and Consequences,* Maryland, Epilepsy Foundation of America, 1990; pp 1–51.
4. Engle J Jr: Basic mechanisms of epilepsy, in *Seizures and Epilepsy,* Philadelphia, FA Davis Company, 1989; pp 71–111.
5. Commission on classification and terminology of the ILAE. Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia* 1981; 22:489–501.
6. Commission on classification and terminology of the ILAE. Proposal for revised classification of epilepsies and epileptic syndromes. *Epilepsia* 1989; 30:389–399.
7. deGraaf AS: Epidemiological aspects of epilepsy in northern Norway. *Epilepsia* 1974; 15:291–299.
8. Granieri E, Rosati G, Tola R, et al: A descriptive study of epilepsy in the District of Copparo, Italy, 1964–1978. *Epilepsia* 1983; 24:502–514.
9. Laster DW, Penry JK, Moody DM, et al: Chronic seizure disorders: Contribution of MR imaging when CT is normal. *AJNR* 1985; 6:177–180.
10. Schorner W, Meencke HJ, Felix R: Temporal-lobe epilepsy: Comparison of CT and MR imaging. *AJR* 1987; 149:1231–1239.
11. Leestma JE, Walczak T, Hughes JR, et al: A prospective study on sudden unexpected death in epilepsy. *Ann Neurol* 1989; 26:195–203.
12. Schraeder PL, Lathers CM: Paroxysmal autonomic dysfunction, epileptogenic activity and sudden death. *Epilepsy Res* 1989; 3:55–62.
13. Dodrill CB, Batzel LW: Interictal behavioral features of patients with epilepsy. *Epilepsia* 1986; 27 Suppl 2 :S64–S76.
14. Wasterlain CG: Mortality and morbidity from serial seizures. An experimental study. *Epilepsia* 1974; 15:155–176.
15. Dodrill CB: Correlates of generalized tonic-clonic seizures with intellectual, neuropsychological, emotional and social function in patients with epilepsy. *Epilepsia* 1986; 27:399–411.
16. Annegers JF, Hauser WA, Elveback LR: Remission of seizures and relapse in patients with epilepsy. *Epilepsia* 1979; 20:729–737.
17. Juul-Jensen P, Foldspang A: Natural history of epileptic seizures. *Epilepsia* 1983; 24:297–312.
18. Huttenlocher PR, Hapke RJ: A follow-up study of intractable seizures in childhood. *Ann Neurol* 1990; 28:699–705.
19. Brorson LO, Wranne L: Long-term prognosis in childhood epilepsy: Survival and seizure prognosis. *Epilepsia* 1987; 28:324–330.
20. Camfield PR, Camfield CS, Dooley JM, et al: Epilepsy after a first unprovoked seizure in childhood. *Neurology* 1985; 35:1657–1660.
21. Hopkins A, Garman A, Clarke C: The first seizure in adult life. Value of clinical features, electroencephalography, and computerised tomographic scanning in prediction of seizure recurrence. *Lancet* 1988; 1:721–726.
22. Annegers JF, Shirts SB, Hauser WA, et al: Risk of recurrence after an initial unprovoked seizure. *Epilepsia* 1986; 27(1):43–50.
23. Hauser WA, Rich SS, Annegers JF, et al: Seizure recurrence after a 1st unprovoked seizure: An extended follow-up. *Neurology* 1990; 40:1163–1170.
24. Shinnar S, Berg AT, Moshe SL, et al: Risk of seizure recurrence following a first unprovoked seizure in childhood: A prospective study. *Pediatrics* 1990 85:1076–1085.
25. Reynolds EH: Early treatment and prognosis of epilepsy. *Epilepsia* 1987; 28(2):97–106.
26. Dean JC, Penry JK: Discontinuation of antiepileptic drugs. In *Antiepileptic Drugs.* Edited by RH Levy, FE Dreifuss, RH Mattson, BS Medrum, JK Penry. New York, Raven Press, 1989; pp 133–142.
27. Lesser RP, Pippenger CE, Luders H, et al: High dose monotherapy in treatment of intractable seizures. *Neurology* 1984; 34:707–711
28. Reynolds EH, Shorvon SD: Monotherapy or polytherapy for epilepsy? *Epilepsia* 1981; 22:1–10.
29. Schmidt D: Reduction of two-drug therapy in intractable epilepsy. *Epilepsia* 1983; 24:368–376.
30. Ojemann LM, Ojemann GA: Treatment of Epilepsy. *Am Fam Physician* 1984; 30:113–128.
31. Wilder BJ: Seizure Disorders. *A Pharmacological Approach to Treatment.* New York, Raven Press, 1981
32. Levy RH, Dreifuss FE, Mattson RH, et al: *Antiepileptic Drugs.* New York, Raven Press, 1989.
33. Pitlick WH: *Antiepileptic Drug Interactions.* New York, Demos Publications, 1989.
34. Letteri JM, Mellk H, Louis S, et al: Diphenylhydantoin metabolism in uremia. *N Engl J Med* 1971: 285:648–652.
35. Odar-Cederlof I, Borga O: Kinetics of diphenylhydantoin in uremic patients: Consequences of decreased plasma protein binding. *Eur J Clin Pharmacol* 1974; 7:31–37.
36. Wilkinson GR: Influence of hepatic diseases on pharmacokinetics, in *Applied Pharmacokinetics: Principles of Therapeutic Drug Monitoring,* WE Evans, JJ Schentag, WJ Jusko (eds). San Francisco, Applied Therapeutics, 1980; pp 57–75.
37. Troupin AS, Ojemann LM: Paradoxical intoxication—a complication of anticonvulsant administration. *Epilepsia* 1975; 16:753–758.
38. Wilensky AJ, Friel PN: Reduced serum phenytoin levels after severe burns. *Epilepsia* 1987; 28:590.
39. Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology: Assessment—Generic substitution for antiepileptic medication. *Neurology* 1990; 40:1641–1643

Eosinophilic globules resembling Mallory bodies in a renal cell carcinoma

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Eosinophilic globules are the light microscopic manifestations of aggregates of cytoplasmic products or organelles seen after hematoxylin and eosin staining. They are a hallmark of α_1-antitrypsin deficiency in the liver and in yolk sac tumors, but have also been described in several types of carcinoma.[1] Eosinophilic material may accumulate in the cytoplasm as a result of cellular injury, with the most well known instance being Mallory hyalin in alcohol-related liver disease.[2,3]

Rare renal cell carcinomas containing eosinophilic globules have been previously documented.[4,5] The few case reports in the literature describe both periodic acid-Schiff (PAS) positive and negative globules, while ultrastructural studies of the globules have been characteristic of Mallory hyalin.

In the present case, we discuss the histologic and cytologic evaluation and significance of eosinophilic globules in a renal cell carcinoma.

CASE REPORT

A 70-year-old woman was admitted in August 1990 to The Mount Sinai Hospital for hematuria and back pain. Five months earlier, she presented with hematuria and was treated with a course of antibiotics. Her physical examination at that time was unremarkable. The hematuria recurred in the late spring and was associated with diffuse back pain. In July, an intravenous pyelogram revealed questionable bilateral renal masses. Pelvic and abdominal computed tomography (CT) scans revealed a solid mass in the right kidney and multiple bilateral cysts. The patient underwent a right radical nephrectomy. Her postoperative course was uneventful, and she was clinically free of disease 6 months after her surgery.

Representative sections of the renal tumor were fixed in 10% neutral buffered formaldehyde solution. Tissue blocks were processed in paraffin, and sections were stained with hematoxylin-eosin, periodic acid-Schiff with (DPAS) and without (PAS) diastase digestion, trichrome, methyl green pyronine, Luxol fast blue, and phosphotungstic acid and hematoxylin (PTAH).

Immunohistochemical studies were performed using the avidin-biotin-peroxidase complex method with appropriately diluted antisera to α-fetoprotein (Dako Patts, Carpinteria, CA), low-molecular-weight keratin (CAM 5.2, Becton Dickinson, San Jose, CA), high-molecular-weight keratin (keratin), α_1-antitrypsin (Dako), desmin (Biogenex), lysozyme (Dako), chromogranin (Biogenex, San Ramon, CA), S-100 protein (Dako), neurofilament (Dako), and muscle common antigen (HHF35, Enzo Diagnostics, Long Island, NY). The immunohistochemical method used has already been described in detail.[6-8] Trypsin digestion was used for all staining, except for low-molecular-weight keratin, in which pepsin digestion was used.[6-8] Specificity controls were performed by replacing the primary antibodies with phosphate-buffered saline at a pH of 7.4 or with normal sera from the same species as the primary antibodies. Positive controls were run for each of the antibodies during the staining procedures.

Bench aspirates were performed at the time of the pathology dissection. Slides were either immediately fixed in alcohol and stained with hematoxylin-eosin or air dried and stained with Diff Quik (modified Giemsa stain).

Tissue for electron microscopy was immersion fixed in 3% glutaraldehyde with 0.2 M sodium cacodylate buffer at a pH of 7.4, post-fixed in 1% osmium tetroxide, dehydrated, and embedded in epoxy resin (Epon). Thick sections were stained with methylene blue and azure II. Smaller areas were selected for ultrastructural study. Thin sections were stained with uranyl acetate-lead citrate and examined under the electron microscope (model JEM 100CX).

Pathologic Findings. A right nephrectomy specimen weighed 650 g and measured 19 cm in greatest diameter. The kidney was extensively replaced by a markedly necrotic white fleshy tumor that attenuated the renal capsule and invaded the renal pelvis. The renal vein was devoid of tumor. The remaining renal parenchyma contained multiple urinary retention cysts.

Light microscopy of the renal neoplasm disclosed sheets of disaggregated tumor cells showing marked pleomorphism and atypia (Fig 1). Individual cells were spindle to polygonal in shape, and numerous tumor giant cells were noted. The striking histologic feature was the randomly distributed, eosinophilic, cytoplasmic globules. They averaged 5 μm in diameter, were round to oblong in shape, and many appeared to be surrounded by a thin, clear halo. The cytoplasm was otherwise amphophilic and slightly granular and had a sharp cytoplasmic membrane. Mitoses averaged two per ten high power fields. Nuclei were vesicular with prominent nucleoli.

The globules, tumor nuclei, and remaining cytoplasm failed to react to various histochemical stains (PAS, with and without diastase, methyl green pyronine, and PTAH). The globules and tumor cell cytoplasm appeared red with trichrome stain. On Luxol fast blue staining, only the globules demonstrated a bright blue positive reaction. On immunohistochemical analysis, the globules were negative for all the antibodies employed (α-fetoprotein, low- and high-molecular-weight cytokeratins, α_1-antitrypsin, desmin, lysozyme, chromogranin, S-100 protein, neurofilament, and muscle common antigen). There was cytoplasmic reactivity only for vimentin.

On Diff quick staining, at low magnification, there were loosely cohesive clusters of large polygonal cells (Fig 2). The cytoplasmic globules had a glassy, opaque, magenta ap-

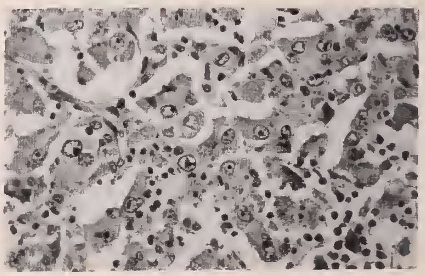

FIGURE 1. Representative histologic section of renal cell carcinoma. The loosely cohesive tumor cells show marked pleomorphism and atypia. The cytoplasm is noteworthy for the randomly distributed globules surrounded by a thin halo. (hematoxylin and eosin stain; original magnification ×200).

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This research was funded by The Hans Shapira Foundation.

pearance. No periglobular halos were visualized. The cytoplasm was otherwise basophilic to amphophilic and slightly filamentous. Fine cytoplasmic vasculization, which was probably an artifactual effect was also focally seen. Nuclei were deeply eosinophilic, whereas the large prominent nucleoli were basophilic. The hematoxylin- and eosin-stained aspirates had an identical appearance to that previously described for the tissue histology; however, no periglobular halos were seen (Fig 2).

At the electron microscopic level, the cells contained large pleomorphic, heterochromatic nuclei with either a single, large nucleolus or a few small nucleoli. The cells had rudimentary desmosomes and many intercellular process interdigitations. The cytoplasms contained relatively few small mitochondria, a significant amount of granular endoplasmic reticulum, Golgi complexes, and many intermediate and microfilaments. There were also numerous lipid vacuoles. In addition, there were nonmembrane-bound inclusions of various sizes (Fig 3). These inclusions consisted of what appear to be fibrillary tangles, consistent with filaments (Fig 3). The tangles were so congested that it was not possible to obtain accurate measurements of individual fibers to identify whether they were 10 nm intermediate or 6 nm actin-type fibers. These inclusions closely resembled Mallory hyalin seen in hepatocytes.

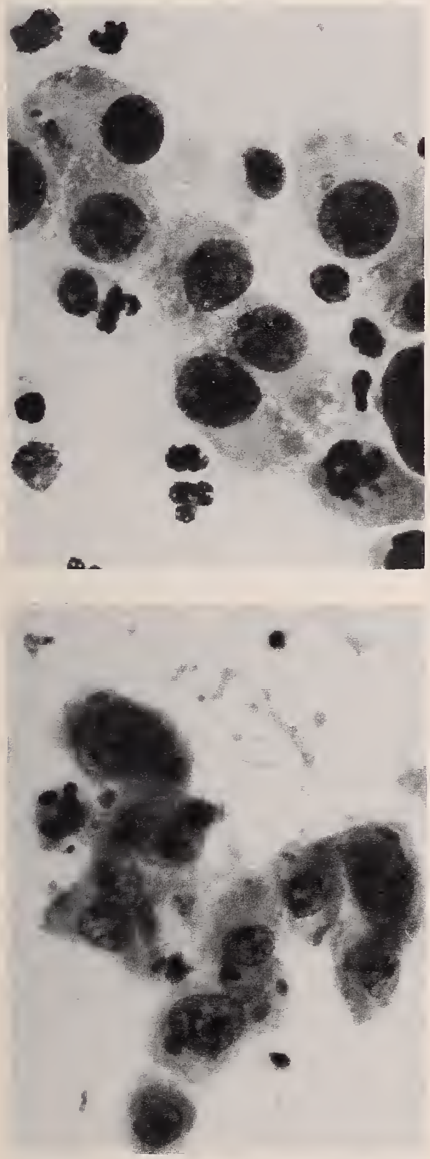

FIGURE 2. Top, cytology of tumor cells after Diff Quick preparation demonstrating opaque cytoplasmic globules. Bottom, cytology of tumor cells after hematoxylin and eosin staining is similar to that seen in the tissue sections, however, no perinuclear halo is apparent (original magnification ×600).

Discussion

Eosinophilic globules may be present in a variety of neoplastic and non-neoplastic tissues. They are the morphologic expression of accumulated cellular secretions, or the sequelae of tissue injury.[1–3] This material can be broadly divided by its reactivity to the DPAS and DAS diastase treatment. Positive staining globules are a hallmark of yolk sac/endodermal sinus tumors and have been observed in other carcinomas such as lung, breast, pancreas, and colon.[11] The globules of yolk sac tumor contain α_1-antitrypsin and α-fetoprotein, as demonstrated by immunohistochemistry.[6] The nature of the inclusions has yet to be understood completely in other malignancies; however, in several cases of lung, liver, and pancreatic carcinomas, α_1-antitrypsin has been identified.[7]

Mallory hyalin, although usually associated with alcohol-related liver disease, may be present in chronic and degenerative hepatic disorders including primary biliary cirrhosis, Wilson's disease, Indian childhood cirrhosis, and benign and malignant hepatic neoplasms.[2,3,7–9] Extrahepatic Mallory hyalin has been reported in pulmonary pathologic conditions, such as radiation pneumonitis, diffuse interstitial fibrosis, organizing bacterial pneumonia, scar adenocarcinoma, and alveolar lining cells associated with asbestosis.[10–12] Mallory hyalin lacks PAS positivity and usually have a reddish color on trichrome staining.

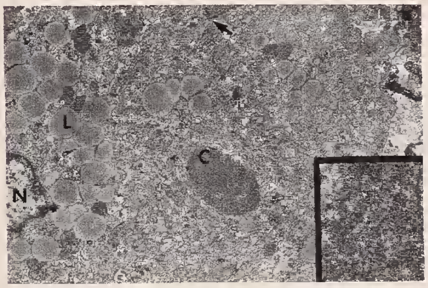

FIGURE 3. Transmission electron micrograph of tumor cells shows nucleated cell (N) containing many lipid vacuoles (L). An adjacent cell exhibits desmosomes (arrow). The most unusual finding is the large cytoplasmic inclusion (C). This inclusion at higher magnification (inset) shows that no enclosing membrane is present and the contents consist of a fibrillary network. (uranyl acetate and lead citrate stain; original magnification ×3,300, inset ×66,000).

Yokoo et al[3] describe three ultrastructural morphologic variants of Mallory hyalin.[3] In the early stages, parallel arrays of filaments are in a random orientation. The more advanced Mallory bodies are electron dense, granular aggregates surrounded by a ring of filaments. Mallory bodies seem to represent accumulations of filaments that normally occur in the cell, which aggregate as a result of cellular injury. In regard to the liver, the electrophoretic mobilities and antigenic determinants of the Mallory hyalin is similar to those of the prekeratin class of intermediate filaments found in hepatocytes.[2] High-molecular-weight intermediate filaments, similar to epidermal cytokeratins, can also be present.

Scattered reports in the surgical pathology literature have described eosinophilic globules in renal cell carcinoma.[4,5] The first case reported by Datta[4] contained PAS-positive globules and were shown ultrastructurally to be dense aggregates of fine granules with no limiting membrane. Strands of rough endoplasmic reticulum were seen loosely applied to their surface, and it was postulated that the granules may have derived from these organelles. No immunologic studies were performed on Datta's case.

Mallory-like bodies in a renal cell carcinoma were subsequently reported by Jagirdar et al.[5] The globules were PAS-negative and immunologically reactive only to anti-Mallory hyalin antibody. On ultrastructural analysis, electron dense, non-membrane-bound globules were seen in close proximity to the nuclei and were composed of finely fibrillar but predominantly granular structures.

Descriptions of eosinophilic globules are primarily limited to tissue histology. Cytoplasmic eosinophilic globules that have been documented in the cytology literature are almost exclusively derived from hepatopathology.[13,14] The PAS-positive inclusions of hepatomas and Mallory bodies stained by the Papanicolaou technique resemble red blood cells superimposed on hepatocytes, whereas on Giemsa stained preparations they will appear blue. The cytologic hepatic globules, like the tissue counterparts, frequently will have immunohistochemical reactivity to α-fetoprotein and α_1-antitrypsin or, on electron microscopy, will appear as giant mitochondria.

Renal tubular cells, like other epithelia, contain as part of their cytoskeleton, intermediate keratin filaments.[15,16] These structures will usually persist in renal cell carcinoma[15–17] and will be expressed by immunohistochemical reactivity to keratin antibodies.[18] A high proportion of renal cell carcinomas also exhibit coexpression for the mesenchymal antigen vimentin, a feature seen in other carcinomas, such as those of the endometrium, lung, ovary, and thyroid.[18–24] Rarely, poorly differentiated carcinomas will only demonstrate vimentin positivity.[18,19]

In summary, we present a poorly differentiated renal cell carcinoma containing eosinophilic globules. After extensive histochemical, immunohistochemical, and ultrastructural studies, the globules most closely resembled late-stage Mallory bodies. They most likely derived from the indigenous cytoskeletal filaments present in an epithelial cell. Although the presence of eosinophilic globules in renal cell carcinoma appears to be an infrequent occurrence, these tumors should be added to the list of neoplasms that may contain these structures.

Acknowledgments. The authors thank Norman Katz and Glenn Maffei for their photographic assistance, Gina Georgescu and Dr David Wolfe for supplying the Luxol fast blue stain, and Marva Barbee for her secretarial assistance.

REFERENCES

1. Wolfe HJ, Palmer PE: alpha-1-antitrypsin. Its immunohistochemical localization and significance in diagnostic pathology, in DeLellis RA (ed): *Diagnostic Immunocytochemistry* New York, Masson, 1991.
2. French SW: Present understanding of the development of Mallory's body. *Arch Pathol Lab Med* 1983; 107:445–450.
3. Yokoo H, Minick OT, Batti F, et al: Morphologic variants of alcoholic hyalin. *Am J Pathol* 1972; 1: 25–31.
4. Datta BN: Hyaline intracytoplasmic globules in renal carcinoma. *Arch Pathol Lab Med* 1977; 101: 391.
5. Jagirdar J, Irie T, French SW, et al: Globular Mallory-like bodies in renal cell carcinoma: Report of a case and review of cytoplasmic eosinophilic globules. *Hum Pathol* 1985; 16:949–952.
6. Thung SN: The development of proliferating ductular structures in liver disease. *Arch Pathol Lab Med* 1990; 114:407–411.
7. Gerber MA, Thung SN, Shen SC, et al: Phenotypic characterization of proliferation: Comparison of antigenic expression by proliferating epithelial cells in fetal liver, massive hepatic necrosis and nodular transformation of the liver. *Am J Pathol* 1983; 110:70–74.
8. Lai Y-S, Thung SN, Gerber MA, et al: Expression of cytokeratin in normal and diseased livers and in primary liver carcinomas. *Arch Pathol Lab Med* 1989; 113:134–138.
9. Palmer PE, Safaii H, Wolfe HJ: Alpha-1-antitrypsin and alpha fetoprotein. Protein markers in endodermal sinus (yolk sac) tumors. *Am J Clin Pathol* 1976; 65:575–582.
10. Palmer PE, Wolfe HJ: Alpha-1-antitrypsin deposition in primary hepatic carcinomas. *Arch Pathol Lab Med* 1976; 100:232–236.
11. Thung SN, Gerber MA, Sarno E, Popper H: Distribution of 5 antigens in hepatoacellular carcinoma. *Lab Invest* 1979; 41:101–105.
12. Palmer PE, Wolfe HJ: Alpha-1-antitrypsin deposition in primary carcinomas. *Arch Pathol Lab Med* 1976; 100:232–236.
13. Craig JR, Peters RL: Fibrolamellar carcinoma of the liver: A tumor of adolescents and young adults with distinctive clinicopathologic features. *Cancer* 1980; 46:372–379.
14. Kuhn C, Kuo TT: Cytoplasmic hyalin asbestosis: A reaction of injured alveolar epithelium. *Arch Pathol Lab Med* 1973; 95:190–194.
15. Warnock ML, Press M, Churg A: Further observations on cytoplasmic hyaline in the lung. *Hum Pathol* 1980; 11:59–65.
16. Michel RP, Limacher JJ, Kimoff RJ: Mallory bodies in scar adenocarcioma of the lung. *Hum Pathol* 1982; 13:81–85.
17. Koss LG, Woyke S, Olszewski W: *Aspiration Biopsy: Cytologic Interpretation and Histologic Bases.* New York, Igaku-Shoin, 1984.
18. Frias-Hidvegi D: Guides to clinical aspirations biopsy. *Liver and Pancreas.* New York, Igaku-Shoin, 1988.
19. Silva E, Mackay B: Ultrastructural features of renal tumors. *Cancer Bull* 1979; 3:6–13.
20. Farrow GM: Diseases of the kidney, in Murphy W (ed): *Urological Pathology* Philadelphia, WB Saunders, 1989.
21. Bonsib SM, Fischer J, Plattner S, et al: Sarcomatoid renal tumors. Clinicopathologic correlations of three cases. *Cancer* 1987; 59:527–532.
22. Reutner VE: Antigen expression in renal cell carcinoma. Correlation with histological grade, pathological stage and development of distant metastasis. *Eur Urol* 1990; 18:4–5.
23. Herman CJ, Moesker O, Kant A, et al: Is renal cell (Grawitz) tumor a carcinoma? Evidence from analysis of intermediate filament types. *Virchows Arch (Cell Pathol)* 1983; 44:73–83.
24. Holthofer H, Miettinen A, Paasivuo R, et al: Cellular origin and differentiation of renal carcinomas. A fluorescence microscopic study with kidney specific antibodies, anti-intermediate filament antibodies and lectins. *Lab Invest* 1983; 49:317–326.
25. Miettinen M, Franssila K, Lehto VP, et al: Expression of intermediate filament proteins in thyroid gland and thyroid tumors. *Lab Invest* 1984; 50: 262–270.
26. Dabbs DJ, Geisinger KR, Norris HT: Intermediate filaments in endometrial and endocervical carcinomas. The diagnostic utility of vimentin patterns. *Am J Surg Pathol* 1986; 10:568–576.
27. Schroder S, Dockhorn-Dvworniczak B, Kastendieck H, et al: Intermediate filament expression in thyroid gland carcinomas. *Virchows Arch (Pathol Anat)* 1986; 409:751–766.
28. Azumi N, Battifora N: The distribution of vimentin and keratin in epithelial and non-epithelial neoplasms. A comprehensive and immunohistochemical study of formalin and alcohol fixed tumors. *Am J Clin Pathol* 1987; 88:286–296.

AIDS-related *Pneumocystis* pneumonia complicated by diffuse pneumatocele formation

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***Pneumocystis carinii* pneumonia** (PCP) is the most common opportunistic lung infection in the acquired immunodeficiency syndrome (AIDS).[1] Typically, the chest radiograph shows diffuse lung shadowing, which progresses from a reticulonodular to a ground glass pattern as the pneumonia worsens. However, atypical presentations, including asymmetric or focal consolidation, nodular opacities (sometimes with cavitation), spontaneous pneumothorax, pleural effusion, lymphadenopathy, and single or multiple cyst-like lucencies, are reported with increasing frequency. The last, often described nonspecifically as "cysts," are present in 10% or more of cases.[1-3] We present a patient with PCP in whom air cysts rapidly developed throughout the lungs and subsequently resolved, consistent with the definition of pneumatocele.[4]

CASE REPORT

A 27-year-old, homosexual man presented with a dry cough and 5 kg weight loss over the previous 1 to 2 months, with recent fever and dyspnea on exertion. His medical history was unremarkable. Examination disclosed a thin man with tachypnea (24/min), tachycardia (110/min), and fever (40°C). He had oral thrush, bilateral cervical and inguinal lymphadenopathy, bronchovesicular breath sounds, and crackles mainly over the left lung; no skin lesions were present. Laboratory abnormalities included low albumin (24 g/L), hyponatremia (130 mmol/L), and mild elevations of serum transaminases, lactic dehydrogenase, and angiotensin-converting enzyme. Arterial blood gas analysis on room air revealed pH 7.50; Pco_2 30 mm Hg; and Po_2 68 mm Hg. A chest roentgenogram demonstrated a diffuse, coarse reticulonodular pattern and innumerable thin-walled air cysts (pneumatoceles), which occupied the majority of all lobes (Fig 1). A film taken elsewhere from 11 days earlier showed widespread airspace consolidation but no pneumatoceles. Pulmonary function tests demonstrated a severe restrictive pattern (vital capacity, 47% of predicted) with normal airflow but impaired diffusion (48% of predicted).

Bronchoalveolar lavage and transbronchial biopsy on day 6 both confirmed PCP. The

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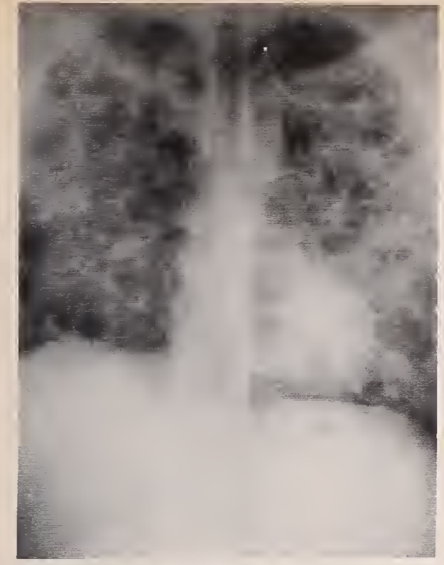

FIGURE 1. Roentgenogram at outset of therapy showing diffuse, non-homogeneous lung consolidation and numerous pneumatoceles involving all lobes.

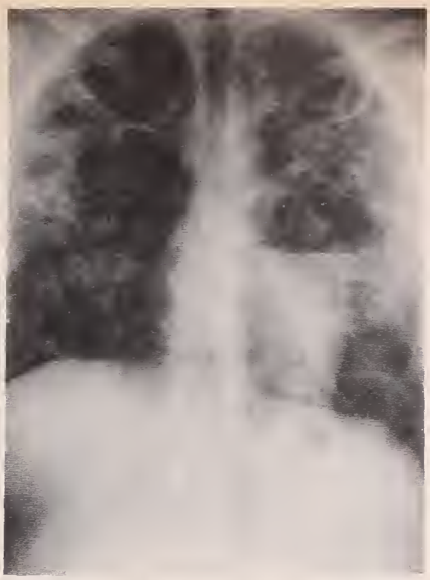

FIGURE 2. Roentgenogram 16 days later showing partial resolution of diffuse pneumonitis but persistence of pneumatoceles, one of which now contains fluid.

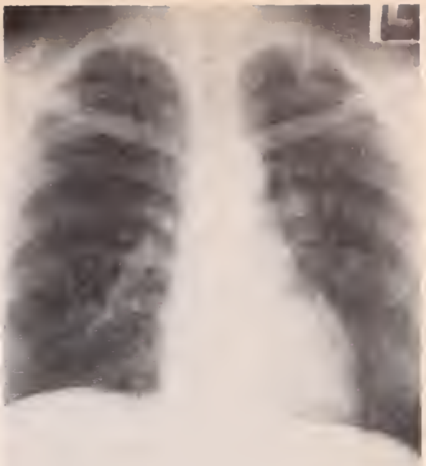

FIGURE 3. Seven months later only a few pneumatoceles remain in left mid-lung and right upper lobe, much diminished in size.

patient's hospital course was marked by a left pneumothorax requiring chest tube drainage, and by the development of an air-fluid level in one pneumatocele (Fig 2), accompanied by renewed fever. The patient's condition improved on intravenous therapy with cotrimoxazole (sulfamethoxazole, 1.6 g; trimethoprim, 320 mg) every 6 hours for 1 week, followed by pentamidine (240 mg daily for 2 weeks), as well as clindamycin (600 mg every 6 hours for 2 weeks) for bacterial superinfection of the pneumatocele. Follow-up chest roentgenograms showed progressive resolution of the pneumatoceles; only a few thin-walled lesions remained seven months later (Fig 3).

Discussion

Pneumatoceles dominated the radiographic picture in this patient and were far more numerous and diffuse than in previous reports of such lesions in PCP.[2,3,5,6] On the basis of radiographic findings, the differential diagnosis also included chronic sarcoidosis or other granulomatous diseases, idiopathic bullous disease, sequelae of diffuse alveolar damage, and widespread bronchiectasis or bronchiolitis. However, cyst-like lesions are usually solitary or few in number in these settings. In contrast to the honeycomb effect seen in end-stage lung disease of varying causes, the air cysts in PCP are typically thin walled and greater than 1 cm in size. They develop rapidly and then usually shrink away over weeks to months.[2,3,5] Their nature remains controversial, due in part to limited histologic correlation and in part to variable use of radiographic terms (cavity, cyst, pneumatocele, bulla).[4,6,7]

Among possible mechanisms for air cyst formation, excavation of consolidated lung parenchyma by liquefactive necrosis, as in tuberculosis or lung abscess, seems unlikely in most cases, based on radiologic and pathologic grounds. Alternatively, an inflammatory breach in the wall of a bronchiole or alveolus may result in interstitial emphysema, which, combined with a check-valve effect, could produce a localized pocket of air under positive pressure. The cysts of PCP are probably analogous to the pneumatoceles classically associated with bacterial pneumonia in children and believed to be a manifestation of interstitial emphysema.[8] Less likely, they might represent terminal airspaces that have ballooned under the influence of parenchymal damage and bronchiolar obstruction.

Recent reports indicate that small foci of necrosis may underlie pulmonary interstitial emphysema in PCP, resulting in pneumatoceles and/or pneumothorax[6,9,10] Necrosis can be a direct effect of the inflammatory response to PCP, but in some cases may reflect vascular involvement with secondary infarction.[6,9] Given the rapidity with which pneumatoceles can appear, as well as their usual resolution with therapy, it is likely that they dilate because of either air-trapping (check-valve mechanism) or the elastic recoil of the surrounding lung. The gradual restoration of lung architecture in this patient supports such a mechanism and argues against tissue necrosis (cavitation) as the main cause of the gas-containing spaces.

Thus, the variable host response to PCP occasionally results in parenchymal pneumatocele formation, for which focal lung necrosis may be the initiating factor. This complication, like pneumothorax, may be more frequent in patients with AIDS who are receiving aerosolized pentamidine, possibly because protracted, necrotizing pneumonitis occurs in this setting.[10–12]

References

1. Murray JF, Mills J: Pulmonary infectious complications of human immunodeficiency virus infection. *Am Rev Respir Dis* 1990; 141:1582–1598.
2. DeLorenzo LJ, Huang CT, Maguire GP, et al: Roentgenographic patterns of *Pneumocystis carinii* pneumonia in 104 patients with AIDS. *Chest* 1987; 91:323–327.
3. Sandhu JS: Pulmonary cysts associated with *Pneumocystis carinii* pneumonia in patients with AIDS. *Radiology* 1989; 173:33–35.
4. Tuddenham WJ: Glossary of terms for thoracic radiology: Recommendations of the nomenclature committee of the Fleischner Society. *AJR* 1984; 143:509–517.
5. Gurney JW, Bates FT: Pulmonary cystic disease: Comparison of *Pneumocystis carinii* pneumatoceles and bullous emphysema due to intravenous drug abuse. *Radiology* 1989; 173:27–31.
6. Feuerstein IM, Archer A, Pluda JM, et al: Thin-walled cavities, cysts, and pneumothorax in *Pneumocystis carinii* pneumonia: Further observations with histological correlation. *Radiology* 1990; 174:697–701.
7. Fraser RG, Pare JAP, Pare PD, et al: *Diagnosis of Diseases of the Chest,* 3rd ed. Philadelphia, WB Saunders, 1988, pp 608–611, 659–663.
8. Efferen LS, Hendler JM: Pneumatoceles. *NY State J Med* 1991; 91:311–314.
9. Liu YC, Tomashefski JF, Jr, Tomford JW, et al: Necrotizing *Pneumocystis carinii* vasculitis associated with lung necrosis and cavitation in a patient with acquired immunodeficiency syndrome. *Arch Pathol Lab Med* 1989; 113:494–497.
10. Beers MF, Sohn M, Swartz M: Recurrent pneumothorax in AIDS patients with pneumocystis pneumonia. A clinicopathologic report of three cases and review of the literature. *Chest* 1990; 98:266–270.
11. Jules-Elysee KM, Stover DE, Zaman MB, et al: Aerosolized pentamidine: Effect on diagnosis and presentation of *Pneumocystis carinii* pneumonia. *Ann Intern Med* 1990; 112:750–757.
12. Sepkowitz KA, Telzak EE, Gold JWM, et al: Pneumothorax in AIDS. *Ann Intern Med* 1991; 114: 455–459.

Neonatal gastrointestinal complications of maternal cocaine abuse

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The incidence of cocaine use has increased dramatically over the last decade, particularly in large, urban centers.[1] There also has been an alarming increase in use during pregnancy, with up to 17% of pregnant women reportedly using cocaine at least once during gestation.[1] Adverse pregnancy outcome has recently been attributed to cocaine use.[1] In utero cocaine exposure has been associated with cerebral infarction and intracerebral hemorrhage,[2] urogenital anomalies,[3] congenital cardiac defects,[4] arterial thrombosis and hypertension,[5] and necrotizing enterocolitis.[6] We report here on two newborns with gastrointestinal disorders associated with in utero cocaine exposure.

CASE REPORTS

Case 1. A 2,050-g female infant was born at home to a 25-year-old, gravida 3 para 1011 mother at 33-weeks gestation by an unassisted vaginal delivery. The mother's history was remarkable for use of "crack" cocaine and marijuana during pregnancy. The specific doses and routes of administration were not known. However, her last episode of crack use was on the day of delivery. There was no known use of cigarettes, alcohol, or other drugs.

The infant was found to be in good condition at a local hospital, with the exception of the left hand which was hypoplastic. Weight, length, and head circumference were appropriate for gestational age. Urine toxicology of both mother and baby was positive for cocaine only. At 7-hours of age, the baby developed bilious vomiting and abdominal distention. An abdominal radiograph revealed dilated loops of bowel. The baby was kept NPO, given intravenous fluid, and transferred to North Shore University Hospital.

The physical examination revealed stable vital signs. The abdomen was soft, nontender, and distended with visible loops of bowel. Bowel sounds were normal, and there were no masses or hepatosplenomegaly. The remainder of the examination was normal. A surgical consultant considered diagnoses of intestinal volvulus, malrotation of the bowel, and intestinal atresia. During surgery, seven widely separated areas of atresia within the jejunum were noted. Multiple resections with end-to-end anastomoses were performed.

The baby recovered well after surgery; however, gastric drainage continued to be bilious and excessive. The infant passed no stools. A contrast enema on day 34 of life revealed an obstruction in the distal sigmoid colon. At surgery, an area of atresia was noted in the sigmoid colon, and a colostomy was created proximal to it. Pathologic examination of the region revealed areas of atresia in the small bowel and sigmoid colon, areas of submucosal fibrosis, and marked stenosis of the bowel lumen. Calcification of the bowel wall and evidence of healed peritonitis was present in one specimen.

Closure of the colostomy was attempted on day 75 of life; however, a perforation was found in the rectum distal to the anastomotic site 2 days later. A new colostomy was created, and the baby was treated with clindamycin and ceftriaxone for 10 days.

An ultrasonogram of the head was negative for hemorrhage at 1 week of life, but a germinal matrix cyst was noted at 2-½ weeks. She recovered without complications.

Case 2. A 2,045-gram, female infant was born at 35–36 weeks gestation via normal spontaneous vaginal delivery at a local hospital to a 29-year-old, gravida 3 para 1011 mother who had had no prenatal care. The mother had smoked one pack of cigarettes per day for 15 years but denied any illicit drug or alcohol use. She presented in labor fully dilated only 20 minutes before delivery. The readings from an external fetal heart rate monitor were within normal limits. Rupture of the membranes occurred 1-½ hours before delivery, and the amniotic fluid was clear but foul smelling. Apgar scores were 9 at 1 minute and 9 at 5 minutes, and meconium was passed in the delivery room.

The infant was transferred to the special care nursery and was begun on feedings at 3-½ hours of age with 10 cc of D10W. She was fed 15–25 cc of infant formula every 3 hours during the first 24 hours. At day 1 of life, the infant vomited bile-stained fluid and passed a bloody stool. An abdominal radiograph revealed dilated loops of bowel without intramural air, bowel wall thickening, portal air, or intestinal perforation. Blood, urine, and cerebrospinal fluid cultures were obtained, and the baby was treated with ampicillin and gentamicin. All cultures were negative. In spite of nasogastric decompression, abdominal distention developed on day 2 of life. A barium enema revealed no passage of contrast beyond the hepatic flexure.

The baby was transferred to North Shore University Hospital, where a physical examination revealed stable vital signs. Weight, length, and head circumference were all at the tenth percentile. The abdomen was soft, distended, tender to palpation, and erythematous in the right lower quadrant. Bowel sounds were normal. No masses or hepatosplenomegally were noted on palpation. The rectal examination was within normal limits, as was the remainder of the physical examination. The baby was given no food by mouth and was given total parenteral nutrition.

A review of the barium enema study performed previously revealed extravasation of barium in the area of the hepatic flexure, and a diagnosis of spontaneous colonic perforation was made. No free air was noted on subsequent abdominal radiographs taken on admission to North Shore University Hospital. Administration of meglumine diatrizoate to the upper gastrointestinal tract revealed normal rotation without leakage of contrast. A surgical consultant recommended conservative treatment with antibiotics, hyperalimentation, and nasogastric decompression, since the perforation appeared to have healed. No surgical procedures were required.

The physical examination findings improved, and the baby began to pass normal stools. Enteral feedings were begun on day 12 of life and were well tolerated. A sweat test to rule out cystic fibrosis and a repeat contrast enema were within normal limits. No other manifestations of ischemia were present in this infant. The mother's urine toxicologic screen was positive for cocaine. A urine toxicologic screen from the infant was not available.

DISCUSSION

Cocaine is an alkaloid with extremely addictive properties and therefore is rapidly becoming a major health problem. Cocaine appears to have multiple mechanisms of action. It blocks the re-uptake of norepinephrine and dopamine at nerve terminals, allowing these transmitters to be present for a longer time and in higher quantities than normal. Elevated plasma cocaine levels achieved by injection of cocaine into gravid ewes have been associated with elevations in norepinephrine and dopamine levels in the ewe, elevation of mean arterial blood pressure in both the ewe and fetus, and a significant decrease in uterine blood flow.[7] Cocaine may exert a direct vasoconstrictive effect on resistance arterioles. The hypertension and vasoconstriction observed in pregnant ewes were not eliminated in spite of pre-treatment with phentolamine, an alpha blocker.[7] However, spasm in rat heart arterioles perfused with cocaine was prevented by the use of

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calcium channel blockers.[8] Cocaine-induced spasm in small arterioles may result in focal endothelial injury, which then disturbs the balance of the prostacyclin-thromboxane system, thus enhancing platelet aggregation and the tendency to form thrombi in these vessels.[9]

All of the above mechanisms result in a decrease in local tissue perfusion and therefore ischemia. Injection of cocaine into the maternal circulation of pregnant ewes resulted in a marked increase in uterine vascular resistance and associated fetal hypoxemia, hypertension, and tachycardia.[10] Hypoxia during critical times of organogenesis and growth could cause a variety of fetal defects.

The cases presented in this report demonstrate evidence of ischemic injury to the developing fetus. The first case, one of a pre-term newborn with multiple small and large bowel atresias, hypoplasia of the left hand, and subependymal germinal matrix cyst, strongly suggests multiple ischemia-related defects.

Cases of multiple intestinal atresias not related to cocaine use, particularly distal to the duodenum, have been described in the literature. These atresias are believed to be secondary to in utero vascular accidents.[11,12] Cocaine has been shown to produce intestinal ischemia when ingested in adults[13] and to produce necrotizing enterocolitis in a term infant.[6] Repeated episodes of cocaine use may result in sufficient intestinal ischemia to produce multiple atresias. Two children with isolated atresia of the large or small bowel born to cocaine-abusing mothers have been reported, but multiple intestinal atresias involving both large and small bowel have not previously been associated with cocaine abuse.[14]

In the second case, the infant developed a spontaneous intestinal perforation without known risk factors for intestinal ischemia other than exposure to cocaine one to two days before delivery. The spontaneous perforation may have been due to a localized area of bowel ischemia shortly before delivery which caused a weakening in the bowel wall. The ischemia could have been cocaine-related via fetal hypoxia, shunting of blood away from the gastrointestinal tract, or local vasoconstriction and thrombus formation. Similar mechanisms have been reported for spontaneous gastrointestinal perforations.[15]

While it is known that other drugs, including amphetamines and nicotine, cause vasoconstriction, the cases reported here only indicate marijuana or cigarette use. Marijuana has been associated with premature birth and decreased fetal growth, but no specific teratogenic abnormalities have been documented.[16,17] Marijuana may potentiate known teratogens by lowering the threshold for their effects.[18] Similarly, more than one component of cigarette smoke may affect the fetus, resulting in decreased birthweight. However, no proven relationship exists between smoking and specific malformations or gastrointestinal disorders.[19]

These two cases supplement the expanding list of fetal gastrointestinal disorders related to in utero cocaine exposure.

REFERENCES

1. Frank DA, Zuckerman BS, Amaro H, et al: Cocaine use during pregnancy: Prevalence and correlates. *Pediatrics* 1988; 82:888–895.
2. Dixon SD, Bejar R: Echoencephalographic findings in neonates associated with maternal cocaine and methamphetamine use: Incidence and clinical correlates. *J Pediatr* 1989; 115:770–778.
3. Chasnoff IJ, Chisum GM, Kaplan WE: Maternal cocaine use and genitourinary tract malformations. *Teratology* 1988; 37:201–204.
4. Little BB, Snell LM, Klein VR, et al: Cocaine abuse during pregnancy: Maternal and fetal implications. *Obstet Gynecol* 1989; 73:157–160.
5. Reznik VM, Anderson J, Griswold WR, et al: Successful fibrinolytic treatment of arterial thrombosis and hypertension in a cocaine-exposed neonate. *Pediatrics* 1989; 84:735–738.
6. Telsey AM, Merrit A, Dixon SD: Cocaine exposure in a term neonate: Necrotizing enterocolitis as a complication. *Clin Pediatr* 1988; 27:547–550.
7. Moore TR, Sorg J, Miller L, et al: Hemodynamic effects of intravenous cocaine on the pregnant ewe and fetus. *Am J Obstet Gynecol* 1986; 155:883–888.
8. Vitullo JC, Karam R, Mekhail N, et al: Cocaine-induced small vessel spasm in isolated rat hearts. *Am J Pathol* 1989; 135:85–91.
9. Simpson RW, Edwards WD: Pathogenesis of cocaine-induced ischemic heart disease. Autopsy findings in a 21-year-old man. *Arch Pathol Lab Med* 1986; 110:479–484.
10. Woods JR, Plessinger MA, Clark KE: Effect of cocaine on uterine blood flow and fetal oxygenation. *JAMA* 1987; 257:957–961.
11. Louw JH, Barnard CN: Congenital intestinal atresia: Observations on its origin. *Lancet* 1955; 2: 1065–1067.
12. El Shafie M, Rickham PP: Multiple intestinal atresias. *J Pediatr Surg* 1970; 5:655–659.
13. Nalbandian H, Sheth N, Dietrich R, et al: Intestinal ischemia caused by cocaine ingestion: Report of two cases. *Surgery* 1985; 97:374–376.
14. Hoyme HE, Jones KL, Dixon SD, et al: Maternal cocaine use and fetal vascular disruption. *Am J Hum Genet* 1988; 43:A566.
15. Zamir O, Goldberg M, Vdassin R, et al: Idiopathic gastrointestinal perforation in the neonate. *J Pediatr Surg* 1988; 23:335–337.
16. Fried PA, Watkinson B, Willan A: Marijuana use during pregnancy and decreased length of gestation. *Am J Obstet Gynecol* 1984; 150:23–27.
17. Zuckerman B, Frank DA, Hingson R, et al: Effects of maternal marijuana and cocaine use on fetal growth. *N Engl J Med* 1989; 320:762–768.
18. Harclerode J: The effect of marijuana on reproduction and development. *Natl Inst Drug Abuse Res Monogr Ser* 1980; 31:137–166.
19. Werler MM, Pober BR, Holmes LB: Smoking and pregnancy. *Teratology* 1985; 32:473–481.

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Headaches and children: A reply to "An urban primary school health program"

TO THE EDITOR: I read with great interest the article by R.B. Birrer et al, "An urban primary school health program" in the August issue of the *Journal.*[1] What surprised me was that headache and migraine were not among the 20 most common diagnoses. Epidemiological studies[2,3] indicate the incidence of migraine in pre-adolescent children to be about 4%, whereas the incidence of headaches ranges between 20% to 54%. These studies were done abroad, and it can be argued that fewer black and Hispanic children have headaches. However, it is clear that headaches are common in children of all ethnic backgrounds.

I would like to know what the authors think about this discrepancy between the high frequency of headaches and migraine in published epidemiological studies and the absence of this diagnosis in their own study.

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2. Bille B: Migraine in school children. *Acta Paediatr Scand.* (Suppl. 136), 1962; 51:1–151.
3. Sillanpaa MI, Peltonen T: Occurrence of headache amongst school children in a Northern Finnish Community. In F. Sicuteri (ed) *Headache, New Vistas.* Florence, Italy, Biomedical Press, 1977, pp 5–8.

In reply. The point made by Dr Alexander Mauskop regarding headache incidence in children is a good one. Although our research article only reported the top 20 diagnoses (Table 2), the diagnostic category of headache (including migraine headache) achieved a ranking of 24 (0.9%) and is in large part responsible for the second place ranking of nervous system disease (12.4%) in Table 1. Number of Diagnoses by ICD-9-CM Classification.

Lastly, the frequency of this diagnosis may be low compared to other studies because the denominator includes all diagnoses, the majority of which are well-child/routine health care. Interestingly, we have seen only six well-defined cases of migraine in project children to date.

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Rhabdomyolysis associated with pneumococcal pneumonia and sepsis in an abstinent alcohol abuser

TO THE EDITOR: Rhabdomyolysis often occurs in the setting of alcohol intoxication. It is thought to arise from trauma, direct toxic effect or hypophosphatemia.[1] We report here a case of rhabdomyolysis in an alcohol abuser in which none of these factors was present, but in whom severe rhabdomyolysis occurred during a bout of pneumococcal sepsis.

Case Report A 36-year-old man presented to the emergency room with shortness of breath, fever, productive cough, chills and pleuritic chest pain for about four days. The patient had a history of hypertension and consumed a pint of alcohol daily but he stated, and his family confirmed, that he had not had a drink for more than a week before admission. Examination showed a well-built muscular man, with an oral temperature of 102.5°F, BP 140/100 mm Hg, pulse 110, and respiratory rate 26/min. He was diaphoretic, tremulous, and lethargic, but oriented to person and place. There was no evidence of trauma. Auscultation of the chest revealed decreased breath sounds in the left lower lung field posteriorly. An initial laboratory examination showed the blood urea nitrogen (BUN) to be 32 mg/dL and the creatinine 2.8 mg/dL. A chest x-ray revealed a left lower lobe infiltrate, and a sputum gram stain showed gram positive diplococci with numerous WBCs.

Urinalysis showed a pH of 6, 3+ blood, 2–3 red blood cells per HPF, and a positive urine test for myoglobin. He was started on intravenous penicillin G, 1 million units every 4 hours. His respiratory status worsened, and he was intubated. Urine output decreased to less than 500 cc per day on the third day of admission, with BUN rising to 132 mg/dL and a creatinine 10.4 mg/dL. The creatine kinase, which had been 4,800 U/L on admission, rose to 68,700 U/L. Hemoglobin was 9.2 g/dL, hematocrit was 29.1%, WBC was 19,200/mm^3, platelet count was 457,000/mm^3, sodium was 141 meq/L, potassium was 5.5 meq/L, glucose was 126 mg/dL, chloride was 99 meq/L, CO_2 was 10 meq/L, protein was 6.5 gm/dL, albumin was 2.9 gm/dL, calcium was 4.6 mg/dL, phosphate was 13.3 mg/dL, and LDH was 3,137 U/L. On admission, urine toxicology showed metabolites of benzodiazepine but no alcohol or its metabolites, and admission blood cultures grew *Strepococcus pneumoniae.* He was hemodialysed for four weeks after which time his urine output increased and the BUN and creatinine started decreasing. The BUN, creatinine, and creatine kinase subsequently returned to normal.

Discussion Infectious causes of rhabdomyolysis account for less than 5% of reported cases, the majority being related to alcohol use, seizures, and trauma.[1] Associated infectious agents include *S. pneumoniae*[2,3], *Brucella,* and *Legionella.*[4] Studies performed on a rat model have demonstrated direct toxic effects of *S. pneumoniae* on skeletal muscle oxidative, glycolytic, and lysosomal enzyme activity.[5]

It has been suggested that up to 33% of patients with rhabdomyolysis develop acute renal failure and that those patients who have non-traumatic rhabdomyolysis have the best prognosis, with a low mortality rate and recovery of renal function in most survivors.[6] It is possible that long term heavy alcohol use could lead to structural or metabolic changes in muscle that would render it more susceptible to damage by toxic or infectious agents. We therefore wish to suggest that in an alcoholic, even in the absence of acute alcohol intoxication, rhabdomyolysis should be considered a possible etiology of any episode of acute renal failure, especially in the setting of pneumococcal pneumonia or a similar infection.

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1. References Gabow, PA, Kaehny WD, Keller SP: The spectrum of rhabdomyolysis. Medicine (Baltimore) 1982; 61:141–152.

2. Marino PL, Nahass GT, Novick W: Bacteremic pneumococcal pneumonia and myoglobinuric renal failure. *Am J Med* 1986; 80:521–522.

3. Hroncich ME, Rudniger AN: Rhabdomyolysis with pneumococcal pneumonia: A report of two cases. *Am J Med* 1989; 86:467–468.

4. Posner MR, Caudill MA, Brass R, et al: Legionnaires' disease associated with rhabdomyolysis and myoglobinuria. *Arch Intern Med* 1980; 140: 848–850.

5. Friman G, Iiback NG, Beisel WR: Effects of Streptococcus pneumoniae, Salmonella typhymurium and Francisella tularensis infections on oxidative, glycolytic and lysosomal enzyme activity in red and white skeletal muscle in rat. *Scand J Infect Dis* 1984; 16:111–119.

6. Grossman RA, Hamilton RW, Morse BM, et al: Non traumatic rhabdomyolysis and acute renal failure. *N Engl J Med* 1974; 291:807–811.

Enteral nutrition supplied by jejunostomy can result in jejunal infarction

TO THE EDITOR: Over the last few years, there has been a significant increase in the use of immediate post-operative enteral feedings supplied via jejunostomy. Today, few surgeons doubt the important of nutritional support in controlling the catabolic state, maintaining immunocompetence, and preventing the multiple organ system failure syndrome. In spite of the many benefits of this procedure, major complications can occur.

Case Report A 34-year-old construction worker presented to the New Jersey Trauma Center by ambulance after a 25-foot fall at work. He had loss of consciousness at the scene and was unresponsive in the Emergency Room.

Physical examination revealed a well nourished, well developed, comatose male. He had a blood pressure of 130/54, a pulse of 70, a respiratory rate of 20, and a temperature of 98°F. The calvarium was intact except for minor lacerations and a right fronto-parietal contusion. The pupils were 3 mm in diameter and equally reactive to light. He had periorbital ecchymoses but no blood or fluid was noted in the external auditory canals, and the tympanic membranes were intact. The examination of the chest revealed no abnormal findings. The abdomen was mildly distended. The extremities showed no sign of injury. The Glasgow Coma Scale was three.

The patient was resuscitated and conventional radiograms were noted to be within normal limits. A CAT scan of the head revealed a right frontal-parietal intracerebral hemorrhage, with intraventricular blood with no midline shift. A concomitant abdominal CAT scan revealed a splenic fracture and free fluid in the peritoneal cavity. A subarachnoid intracranial pressure monitor was put in place. At exploration, a grade 4 splenic laceration was discovered and treated by hemisplenectomy.

Because of the severity of the cerebral injury, a prolonged coma was anticipated, and a jejunostomy for nutritional support was constructed. A site 40 cm from the ligament of Treitz was selected, and a 14 French red rubber catheter was inserted and secured using a Witzel technique. This was exteriorized in the left upper quadrant. Postoperatively the patient remained in deep coma. Jejunostomy feeding (Traum-Aid) was started on the following day, using a 50% concentration at a rate of 50 cc/hr. Although initially well tolerated, on the third postoperative day the patient developed abdominal distension, diarrhea, and hypotension, and the feeding was stopped. Over the next few hours, the patient continued to do poorly with persistent hypotension and a decrease in hemoglobin from 11 to 8 gm. His abdomen was reexplored, and necrosis of the small bowel was identified. The infarcted area extended for 20 cm proximal and distal to the jejunostomy site, sparing the bowel near the ligament of Treitz. This area was resected, and both ends of the jejunum were closed with a surgical stapler. The next day a "second look" celiotomy revealed no further necrosis, and gastrointestinal continuity was restored. A gastrostomy was put in place and functioned postoperatively without difficulty. The patient recovered fully from his abdominal injuries and was discharged to a long-term care facility with persistent coma.

Discussion There are several, well-known complications associated with the use of jejunostomies for supplying enteral nutrition. Most are minor, involving catheter displacements or occlusion and diarrhea. Others are more serious, and include leakage into the peritoneal cavity with peritonitis, abdominal wall fascitis, fistula formation, and aspiration pneumonia.[1] Pneumatosis intestinalis has also been reported[2–4] and has generally been considered amenable to nonoperative management. Thompson described a case of small bowel necrosis associated with jejunostomy tube feeding in 1983. He points out that intramural gas can be seen in cases of intestinal necrosis and pneumatosis intestinalis and advises caution in treating these patients conservatively.[5] Another five cases were reported by Gaddy et al, where the entire small intestine was affected in three patients, with necrosis confined to the jejunum in two. Hypotension was present in four cases before the discovery of the infarction. The investigators postulate ischemia as the underlying mechanism of infarction, and implicate distention of the bowel wall and increased metabolic demand secondary to the feeding as possible causative factors.[6] Risk factors for mesenteric low flow states were present in all five cases. The overall incidence was 4% with a mortality of 100%.[1]

An additional three cases were reported by Brotman et al. In two instances, necrosis involved only the jejunostomy site, with infarction of a larger segment of jejunum in the remaining patient.[7] In the case reported herein, no risk factors for decreased mesenteric blood flow before injury were present. The patient did not manifest hypotension until just before the discovery of the jejunal necrosis, at which time abdominal distention was also noted. No mechanical or technical cause for the infarction was identified. Although we concur with the previously cited authors[6,7] that the most logical cause would be ischemia, whether the ischemia is due to increased metabolic demands or to decreased blood supply is unknown.

Early nutrition has assumed an important role in modern surgical practice, with jejunostomy being an indispensable route for the delivery of feedings. Surgeons who employ this modality should be aware that necrosis of the small bowel can occur. A mechanical cause for infarction may not be present.

As the application of this procedure becomes even more common, we feel that jejunal infarction will be recognized as a rare but inherent complication. Great caution should be exercised when using jejunostomies for nutritional support in patients who have preexisting risk factors for decreased mesenteric blood flow and for those who have been hypotensive before or during surgery. Close observation during the institution of tube feedings should be mandatory in all cases. Abdominal distention, especially when associated with hypotension should prompt immediate discontinuation of jejunostomy feedings. Early abdominal exploration is essential in patients who remain symptomatic.

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2. Cogbill TH, Wolfson RH, Moore EE, et al: Massive pneumatosis intestinalis, and subcutaneous emphysema: Complication of needle catheter jejunostomy. *J Parent Ent Nut* 1983; 7:171–175.

3. Strain JD, Rudicoff JC, Moore EE, et al: Pneumatosis intestinalis associated with intracatheter jejunostomy feeding. *Am J Radiol* 1982; 139:107–108.

4. Zern RT, Clarke-Pearson DL: Pneumatosis intestinalis associated with enteral feedings by catheter jejunostomy. *Obstet Gynecol* 65:81S–83S.

5. Thompson JS: Pneumatosis intestinalis and needle catheter jejunostomy: A word of caution. (Letter) *J Parent Ent Nut* 1983; 7:495.

6. Gaddy MC, Martin HM, Schwab CW, et al: Small bowel ischemia: A consequence of feeding jejunostomy? *S Med J* 1984; 79:180–182.

7. Brotman S, Marshall W: Complications from needle catheter jejunostomy in post-traumatic surgery. *Con Surg* 1985; 27:52–54.

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Anthony R. Pisani, MD, White Plains. Died September 23, 1991; age 50. Dr Pisani was a 1971 graduate of the Facolta di Medicina e Chirurgia, Universita di Roma-La Sapienza, Rome, Italy. He was a Diplomate of the American Board of Plastic Surgery and a Fellow of the American College of Surgeons. His memberships included the New York State Surgical Society, the American Society of Plastic and Reconstructive Surgery, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Murray Polsky, MD, Elmira. Died October 2, 1991; age 77. Dr Polsky was a 1940 graduate of Louisiana State University School of Medicine in New Orleans. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the Medical Society of the County of Chemung and the Medical Society of the State of New York.

Charles Joseph Preefer, MD, West Palm Beach, Florida. Died October 4, 1991; age 87. Dr Preefer was a 1930 graduate of Tufts University School of Medicine, Boston. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York Society for Clinical Ophthalmology, the American Academy of Ophthalmology and Otolaryngology, the Pan American Association of Ophthalmology, the American Geriatrics Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Albert J. Rosso, MD, Kenmore. Died October 10, 1991; age 70. Dr Rosso was a 1945 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Isabelle D. A. Seismann, MD, Fort Lauderdale, Florida. Died June 1, 1991; age 74. Dr Seismann was a 1942 graduate of New York Medical College. She was a Diplomate of the American Board of Obstetrics and Gynecology, a Fellow of the American College of Obstetricians and Gynecologist, and a Fellow of the American College of Surgeons. Her memberships included the American Medical Women's Association, Medical Society of the County of Kings, and the Medical Society of the State of New York.

Hyman Irving Teperson, MD, Fort Lauderdale, Florida. Died November 1, 1991; age 98. Dr Teperson was a 1916 graduate of the State University of New York Health Science Center at Brooklyn, College of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Schedule of Sessions 27
Officers, Councilors, and Trustees 29
Members of the House 32
Reference Committees 35
Nominating Committee 36
Report of the House Committee on Bylaws 37

HOUSE OF DELEGATES

March 19 through March 22, 1992
Marriott Marquis Hotel
New York City

SCHEDULE OF SESSIONS

Thursday, March 19, 1992
Opening Session . . . . 1:00 p.m.

Friday, March 20, 1992
Reference Committee Hearings . . . . . 8:30 a.m.–5:00 p.m.

Saturday, March 21, 1992
Second Session . . . . 9:00 a.m.
Third Session . . . . 1:30 p.m.

Sunday, March 22, 1992
Closing Session . . . . 9:00 a.m.

A Guide to the 1992 Annual Meeting

Policy-making in the House of Delegates

This section of the New York State Journal of Medicine is a call to the officers, delegates, and members of the Medical Society of the State of New York to attend the 186th Annual Meeting of the Society and the meeting of its House of Delegates; 317 delegates representing 61 county medical societies, 9 district branches, specialty societies, the hospital medical staff section, 12 medical schools, the young physician, resident physician, and the medical student sections will meet at the Marriott Marquis Hotel in New York City on March 19–22, 1992.

The officers, members of the Council and the Board of Trustees, past presidents of the MSSNY, the Commissioner of the New York State Department of Health, the Past President of the American Medical Association from New York, Gerald D. Dorman, MD; Past Executive Vice-President of the Medical Society of the State of New York, George J. Lawrence, Jr., MD; and Past Deputy Executive Vice-President, Robert J. O'Connor, MD; are also official representatives to the House.

Mandated by the Bylaws of the Society, this section lists the names of elected representatives, delegates who are serving on reference committees, and selections of the Nominating Committee for the election of Officers, Councilors, Trustees, and AMA Delegates. Also included are the amendments to the Bylaws which will be considered by the House of Delegates.

Since the House of Delegates is the legislative body of the Society, charged with the general management of the State Society and its affairs, members of the Society are urged to use this information to consult with and advise their representatives. Resolutions introduced by the representatives bring members' concerns to the attention of the entire Society. Representatives not only have the responsibility to present the views of their constituents, but also to inform them of the actions taken by this governing body. The House of Delegates also formulates policy for the Society. No officer, councilor, board, commission, committee, or employee may initiate any policy unless such policy has been expressly approved by the House of Delegates, or by the Council of the Society.

If one were to choose the most important work of the House of Delegates, it would have to be that of the reference committees. Any member of the Medical Society of the State of New York has the right to appear before any reference committee and participate in its deliberations. It is also in the reference committees that people outside of the State Society are asked to give their views. Nonmember physicians, guests, or interested observers who wish to express their opinions may, with permission, come before the reference committee and speak to the business of the committee. I strongly urge members to use this forum to express their views.

Seymour R. Stall, M.D., Speaker
House of Delegates

John H. Carter, M.D., Vice-Speaker
House of Delegates

MEDICAL SOCIETY OF THE STATE OF NEW YORK

Charles N. Aswad, MD

Broome
President

Morton Kurtz, MD

Queens
President-Elect

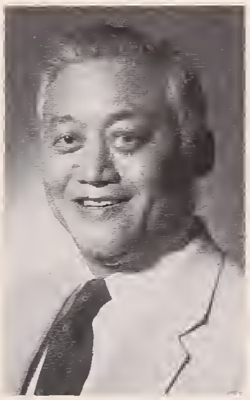
Officers 1991–1992

Robert A. Mayers, MD

Westchester
Past President

Stanley A. Steckler, MD

Suffolk
Vice-President



George Lim, MD

Oneida
Secretary

Joyce M. McChesney, MD

Monroe
Assistant Secretary

James H. Cosgriff, Jr., MD

Erie
Treasurer

Antonio F. LaSorte, MD

Broome
Assistant Treasurer

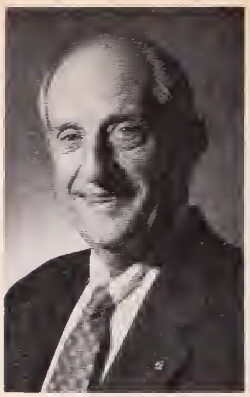
Seymour R. Stall, MD

Dutchess
Speaker

John H. Carter, MD

Albany
Vice-Speaker

Board of Trustees 1991–1992


Allison B. Landolt, MD

Westchester

Chairman

John A. Finkbeiner, MD

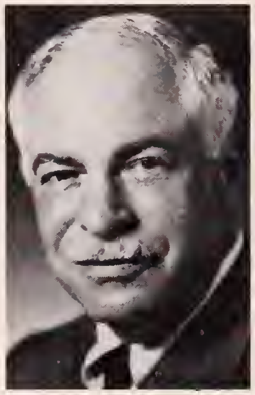
New York

James M. Flanagan, MD

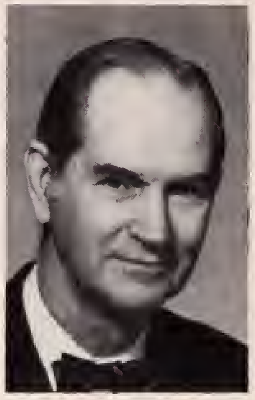
Wayne

Samuel M. Gelfand, MD

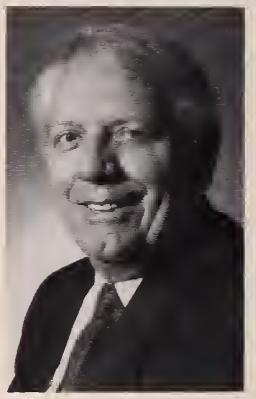
Nassau


M. Theodore Tanenhaus, MD

Kings

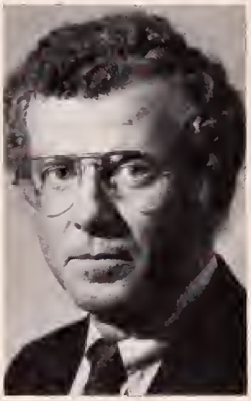

Charles D. Sherman, MD

Monroe

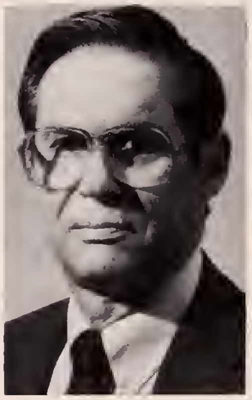

Duane M. Cady, MD

Onondaga

Councilors 1991–1992


Robert E. Fear, MD

Suffolk


Stanley L. Grossman, MD

Orange

Thomas D. Pemrick, MD

Rensselaer

Ralph E. Schlossman, MD

Queens

Randall D. Bloomfield, MD

Kings

William A. Dolan, MD

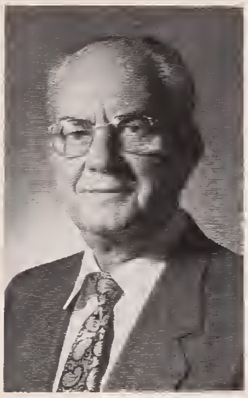
Monroe

Robert M. George, MD

Oneida

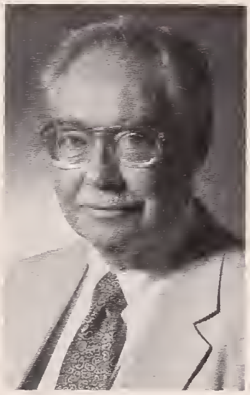
Stuart I. Orsher, MD

New York



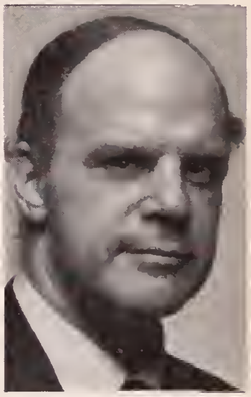
Robert Bruce Bergmann, MD

Nassau



Richard A. Hughes, MD

Warren



Jeffrey A. Ribner, MD

Broome

Anthony P. Santomauro, MD

Erie

Matthew J. Kleinerman

New York

Paul Pipia, MD

New York

1992 HOUSE OF DELEGATES MEMBERS

The following is a list of members of the 1992 House of Delegates of the Medical Society of the State of New York.

Officers 1991–1992
President—Charles N. Aswad, Broome
President–Elect—Morton Kurtz, Queens
Vice–President—Stanley A. Steckler, Suffolk
Secretary—George Lim, Oneida
Assistant Secretary—Joyce M. McChesney, Monroe
Treasurer—James H. Cosgriff, Erie
Assistant Treasurer—Antonio F. LaSorte, Broome
Speaker—Seymour R. Stall, Dutchess
Vice–Speaker—John H. Carter, Albany

Councilors
Term Expires 1992
Robert E. Fear, Suffolk
Stanley L. Grossman, Orange
Thomas D. Pemrick, Rensselaer
Ralph E. Schlossman, Queens

Term Expires 1993
Randall D. Bloomfield, Kings
William A. Dolan, Monroe
Robert M. George, Oneida
Stuart I. Orsher, New York

Term Expires 1994
Robert Bruce Bergmann, Nassau
Richard A. Hughes, Sr, Warren
Jeffrey A. Ribner, Broome
Anthony P. Santomauro, Erie

Student Councilor
Term Expires 1992
Matthew J. Kleinerman, New York

Resident Councilor
Term Expires 1992
Paul Pipia, New York

Trustees
Allison B. Landolt, Westchester, *Chairman*
John A. Finkbeiner, New York
James M. Flanagan, Wayne
Samuel M. Gelfand, Nassau
M. Theodore Tanenhaus, Kings
Charles D. Sherman, Monroe
Duane M. Cady, Onondaga

Past Presidents
1960–1961—Norman S. Moore, Tompkins
1966–1967—James M. Blake, Schenectady
1971–1972—George Himler, New York
1972–1973—Edward Siegel, Clinton
1975–1976—Ralph S. Emerson, Nassau
1976–1977—George L. Collins, Jr, Erie
1978–1979—George T. C. Way, Dutchess
1980–1981—Ralph M. Schwartz, Bronx
1982–1983—Richard D. Eberle, Onondaga
1983–1984—Bernard J. Pisani, New York
1984–1985—Allison B. Landolt, Westchester
1985–1986—Daniel F. O'Keeffe, Warren
1986–1987—John A. Finkbeiner, New York
1987–1988—Samuel M. Gelfand, Nassau
1988–1989—Charles D. Sherman, Monroe
1989–1990—David M. Benford, New York
1990–1991—Robert A. Mayers, Westchester

Past Executive Vice–President
George J. Lawrence, Jr, Queens

Past Deputy Executive Vice–President
Robert J. O'Connor, Richmond

Past President of the American Medical Association
Gerald D. Dorman, New York

President–Elect of the American Medical Association
John L. Clowe, Schenectady

Commissioner, New York State Department of Health

District Delegates
(Elected Delegates of District Branches)
First—Albert Accettola, Jr, Richmond
Second—Melvyn Bruckstein, Suffolk
Third—Albert M. Ellman, Albany
Fourth—
Fifth—
Sixth—Anton A. Vreede, Broome
Seventh—Norman W. Wetterau, Livingston
Eighth—Bruce E. Baker, Genesee
Ninth—Zane A. Miranda, Westchester

Specialty Society Delegates
New York State Society of Allergy and Immunology—
New York State Society of Anesthesiology—Edward C. Sinnott, Nassau
New York State Chapter American College of Cardiology—Joseph Lauterstein, Sullivan
New York State Society of Dermatology—
New York Chapter American College of Emergency Physicians—
New York State Academy of Family Physicians—Elise Korman, Suffolk
New York State Society for Surgery of the Hand—
New York State Society of Internal Medicine—Robert M. Kohn, Erie
New York State Neurosurgical Society—Herbert Oestreich, Westchester
American College of Obstetricians and Gynecologists—Burton Krumholz, Nassau
New York Occupational Medical Association—William Schneider, New York
New York State Ophthalmological Society—Francis Gilroy, Broome
New York State Society of Orthopaedic Surgeons—Leo Arthur Green, Queens
New York State Society of Otolaryngology—Anthony J. Durante, Nassau
New York State Society of Pathologists—Edward Zaino, Nassau
American Academy of Pediatrics–District II—
New York Society of Physical Medicine and Rehabilitation—Irvin Kotkin, Queens
New York Regional Society of Plastic and Reconstructive Surgery—Arthur J. Wise, Nassau
New York State Psychiatric Association—Edward Gordon, Putnam
The New York State Radiological Society (A Chapter of the American College of Radiology—Jerome S. Zwanger, Nassau
New York State Society of Surgeons, Inc—Theodore C. Max, Oneida
New York State Urological Society, Inc—Datta G. Wagle, Erie

Delegates from Component Medical Societies

Albany (5)
Teresa S. Briggs, Albany
Steven M. Parnes, Albany
James H. Puleo, Albany
Nathan P. Reed, Albany
Demostene Romanucci, Latham

Allegany (1)
Paul Kolisch, Friendship

Bronx (10)
John P. Albanese, Bronx
Sana Bloch, Bronx
Marcos A. Charles, Bronx
Dattatraya G. Lanjewar, Pelham Manor
Stanley Luftschein, Rye
Hugo Morales, Bronx
Venkatachala Pathy, Bronx
Raul Pohorille, New Rochelle
Howard S. Schwartz, Bronx
Juana Toporovsky, Bronx

Broome (4)
Jerome Cohen, Johnson City
Louis W. Giordano, Endicott
Theodore R. Poritz, Endwell
Charles Rubin, Johnson City

Cattaraugus (1)
Edward Griffin, Olean

Cayuga (1)
Richard J. Freeman, Auburn
C. Richard Nangle, Auburn

Chautauqua (2)
Nadia Geleil, Jamestown
Bert Rappole, Jamestown

Chemung (2)
Donald L. Brooks, Elmira
John Forrest, Elmira

Chenango (1)
Lawrence J. Rosenblum, Norwich

Clinton (2)

Columbia (2)

Cortland (1)

Delaware (1)
Gary Preiser, Walton

Dutchess (4)
Eleanor C. Kane, Poughkeepsie
Florian M. Cortese, Hyde Park
Reuben Delgado, Fishkill
Jagdish Wadhwa, Poughkeepsie

Erie (9)
Richard J. Buckley, Amherst
Amy P. Early, Williamsville
Edmond J. Gicewicz, Getzville
Allen L. Lesswing, Buffalo
William K. Major, Jr, Buffalo
Nancy H. Nielsen, Orchard Park
Richard M. Peer, Buffalo
H. John Rubinstein, Tonawanda
Franklin Zeplowitz, Buffalo

Essex (1)

Franklin (1)

Fulton (1)
A. J. D'Errico, Gloversville

Genesee (1)
David A. Parker, Batavia

Greene (1)
Karuvath Enu, Hudson

Herkimer (1)
Krishna K. Vadlamudi, Ilion

Jefferson (2)
Louis C. Battista, II, Watertown
David O. VanEenenaam, Watertown

Kings (19)
Joseph C. Amico, Brooklyn
Rubin Berlinerblau, Brooklyn
Norman S. Blackman, Brooklyn
George W. Calvosa, Brooklyn
Duncan W. Clark, Brooklyn
Clifford Cohen, Brooklyn
Vashti R. Curlin, Brooklyn
Paul Finkelstein, Brooklyn
Robert E. Gordon, Valhalla
Julius Holtzman, Garden City
Iraj Iraj, Brooklyn
Monte Malach, Brooklyn
Martin Markowitz, Brooklyn
Rufus A. Nichols, Brooklyn
Hubert S. Pearlman, Brooklyn
Judithann Pryce, Brooklyn
Abdul Rehman, Brooklyn
Veronica C. Santilli, Brooklyn
Marcus Wiener, Brooklyn

Lewis (1)
John C. Herrman, Lowville

Livingston (1)
Sheppard Arluck, Geneseo

Madison (1)
Leonard A. Argentine, Oneida

Monroe (7)
Marilyn R. Brown, Rochester
Thomas Cardillo, Victor
William A. Kern, Jr, Rochester
Edward D. Lewis, Rochester
Mario B. LoMonaco, Rochester
Alexander L. Strasser, Rochester
Edward C. Tanner, Rochester

Montgomery (2)
Feori Pipito, Amsterdam

Nassau (11)
Lee S. Binder, Valley Stream
Patrick F. Carone, Rockville Centre
A. Philip Fontanetta, Mineola
Paul A. Hamlin, Great Neck
Arnold D. Lurie, Rockville Centre
Sharon C. H. Mead, Massapequa
Sidney Mishkin, Fort Salonga
Felix A. Monaco, Rockville Centre
John A. Ostuni, Freeport
E. John Pesiri, Massapequa
Leonard S. Weiss, Levittown

New York (22)
Arnold L. Bachman, New York
Melvin H. Becker, New York
Robert S. Bernstein, New York
Michael B. Brodin, New York
Kenneth H. Brookler, New York
Joseph B. Cleary, New York
Anthony A. Clemendor, New York
Irwin J. Cohen, New York
Hugh C. Davidson, New York
Joseph E. Davis, New York
Ralph L. Engle, New York
Jonas M. Goldstone, New York
George Hyams, New York
Virginia Kanick, New York
Vance Lauderdale, Jr, New York
Samuel H. Madell, New York
Aaron G. Meislin, New York
Alfred F. Moldovan, New York
Donna B. O'Hare, New York
Stuart I. Orsher, New York
Richard N. Pierson, Jr, New York
Clifford N. Spingarn, New York

Niagara (3)
Mary Ann Bishara, Lewiston
James J. Kropelin, Niagara Falls
Robert J. Reszel, Tonawanda

Oneida (4)
Sidney J. Blatt, New Hartford
James D. Dunda, Rome
Kenneth K. Kim, Utica
Steven Z. Kussin, Utica

Onondaga (7)
Armand J. Cincotta, Liverpool
Daniel L. Dombroski, Syracuse
Jack Egnatinsky, Syracuse
John A. Hoepner, Syracuse
Richard A. Konys, Syracuse
Frank J. Piper, Syracuse
Patricia A. Randall, Syracuse

Ontario (2)
Kenneth Steadman, Geneva
Verne Marshall, Geneva

Orange (4)
Stanley F. Brunn, Port Jervis
Clayton R. DeHaan, Middletown
James V. DiLorenzo, Newburgh
Michael A. Parmer, Port Jervis

Orleans (1)
David B. Meza, Albion

Oswego (1)
Michael S. Nupuf, Oswego

Otsego (3)
Rodman D. Carter, Cooperstown
John O. Lusins, Oneonta
Jonathan Sastic, Oneonta

Putnam (2)
Edward L. Socolow, Mt. Kisco
Suresh Chandani, Cold Spring

Queens (16)
Marvin L. Blumberg, Rego Park
Daniel Chansky, Forest Hills
John W. V. Cordice, Jr, St. Albans
Daniel G. Deckler, Richmond Hill
Alexander M. De la Garza, Flushing
Fred S. Fensterer, Glendale
Fred N. Flatau, Flushing
Lorraine M. Giordano, Jamaica Estates
Victor Guarneri, Bayside
Nicetas H. Kuo, Great Neck
Maxwell J. Marder, Great Neck
Jean P. Papps, Jamaica
Frank Ratner, Flushing
Hilda Ratner, Beechurst
Sheldon D. Sax, Flushing
Ezra A. Wolff, Manhasset

Rensselaer (2)
Arsenio G. Agopovich, Watervliet
William A. Whyland, Troy

Richmond (4)
Robert Bonvino, Staten Island
Zoltan Brody, Staten Island
Tano S. Carbonaro, Staten Island
Mark Jarrett, Staten Island

Rockland (4)

St. Lawrence (1)
Ravinder Agarwal, Ogdensburg

Saratoga (1)
Allen T. Jones, Saratoga

Schenectady (3)
William Busino, Jr, Schenectady
Richard H. Lange, Schenectady
Robert J. Pletman, Schenectady

Schoharie (1)

Schuyler (1)

Seneca (1)
Donald Bidwell, Seneca Falls

Steuben (1)

Suffolk (11)
Bruce H. Berlin, Huntington
Stephen I. Braitman, Islip Terrace
Clive D. Caplan, Islip Terrace
Frank R. Collier, Port Jefferson
John G. Egner, West Islip
Kenneth B. Levites, W. Sayville
Philip A. Mantia, Smithtown
Martin Matalon, Bay Shore
Gerard F. Ryan, Patchogue
Robert A. Scher, Huntington
William H. Weir, Jr, Riverhead

Sullivan (1)
Gary M. Phillips, Monticello

Tioga (1)

Tompkins (2)
Hope Perry, Ithaca
Elliot Rubinstein, Ithaca

Ulster (2)
John V. Ioia, Kingston
Fred Horwitz, Ellenville

Warren (3)
Robert Hughes, Queensbury
Robert Reid, Queensbury

Washington (1)
Barney Rubenstein, Cambridge

Wayne (3)
David T. Hannan, Newark
Daniel Koretz, Ontario
Norman Loomis, Ontario

Westchester (9)
Ann C. Cea, Rye Brook
Mark L. Fox, Bronxville
Sanford A. Kaplan, Bronxville
Thomas A. Lansen, Valhalla
Robert C. Mickatavage, Rye
Toni G. Novick, New Rochelle
Stephen O. Schwartz, White Plains
Pellegrino J. Tozzo, New Rochelle
William J. Walsh, Jr, White Plains

Wyoming (1)

Yates (1)
Michael Boquard, Penn Yan

Medical School Delegates (12)
Albany Medical—Henry S. Pohl, Albany
Albert Einstein College of Medicine of Yeshiva University of New York—Melvin Zelefsky, Bronx
Columbia University College of Physicians and Surgeons—Linda D. Lewis, New York
Cornell University Medical College—
Mount Sinai School of Medicine of the City University of New York—Kevin Troy, New York
New York Medical College—Susan Anderson Kline, Westchester
New York University of Medicine—
State University of New York Health Science Center at Brooklyn—Schuyler Kohl,Kings
State University of New York at Buffalo School of Medicine—Dennis Nadler, Erie
State University of New York at Stony Brook School of Medicine—Dorothy Lane, Suffolk
State University of New York Health Science Center at Syracuse—John Bernard Henry, Onondaga
University of Rochester School of Medicine and Dentistry—Philip P. Bonanni, Monroe

Medical Student Delegates (3)
Michal Amir, New York
Gerard J. Dynes, New York
Joseph S. Valenti, Erie

Resident Membership Delegate (1)
Mitchell Miller, Schenectady

Young Physician Delegate (1)
John F. Piper, Onondaga

Hospital Medical Staff Delegate (1)
L. P. Hinterbuchner, Kings

REFERENCE COMMITTEES

Bylaws

Elise Korman, MD, New York State Academy of Family Physicians, *Chairperson*
Armand J. Cincotta, MD, Onondaga
Stanley Oransky, MD, Rockland
John F. Piper, MD, Young Physician Section
Pellegrino J. Tozzo, MD, Westchester
Clayton DeHaan, MD, Orange, (Observer)

Governmental Affairs and Legal Matters

Thomas A. Lansen, MD, Westchester, *Chairman*
John P. Albanese, MD, The Bronx
Edmond J. Gicewicz, MD, Erie
Marilyn Pearl, MD, New York
Kenneth T. Steadman, MD, Ontario
Jerome Cohen, MD, Broome (Observer)

Hospital and Medical Facilities

David B. L. Meza, III, MD, Orleans, *Chairman*
Reuben J. Delgado, MD, Dutchess
Daniel S. Fuleihan, MD, Onondaga
Lorraine M. Giordano, MD, Queens
Ladislav P. Hinterbuchner, MD, Hospital Medical Staff Section
Melvin Zelefsky, MD, Albert Einstein College of Medicine of Yeshiva University of New York (Observer)

Public Health and Education

Robert Bonvino, MD, Richmond, *Chairman*
Bruce E. Baker, MD, Eighth District Branch
Philip P. Bonanni, MD, University of Rochester School of Medicine and Dentistry
John V. W. Cordice, Jr, MD, Queens
Joseph E. Davis, MD, New York
Robert J. Hughes, MD, Warren (Observer)

Reports of Officers and Administrative Matters

Sharon Mead, MD, Nassau, *Chairman*
William A. Busino, Jr, MD, Schenectady
Florian Cortese, MD, Dutchess
Kenneth K. Kim, MD, Oneida
Edward C. Sinnott, MD, New York State Society of Anesthesiology

Socio-Medical Economics

David T. Hannan, MD, Wayne, *Chairman*
Martin Matalon, MD, Suffolk
Raul Pohorille, MD, The Bronx
Edward C. Tanner, MD, Monroe
E. John Pesiri, MD, Nassau
Kenneth H. Brooklen, MD, New York (Observer)

CONVENTION COMMITTEES

Credentials

Louis C. Battista, II, MD, Jefferson, *Chairman*
Michal Amir, Medical Student Section
John S. Forrest, MD, Chemung
C. Richard Nangle, MD, Cayuga
Ezra A. Wolff, MD, Queens
Iraj Iraj, MD, Kings (Observer)

Sergeant at Arms

Albert B. Accettola, MD, First District Branch, *Chairman*
George W. Calvosa, MD, Kings
David A. Parker, MD, Genesee
Steven M. Parnes, MD, Albany
Richard M. Peer, MD, Erie
Corliss Varnum, MD, Oswego (Observer)

Rules and Order of Business

Mario B. LoMonaco, MD, Monroe, *Chairman*
Robert S. Bernstein, MD, New York
Mary Ann Bishara, MD, Niagara
John G. Egner, MD, Suffolk
Arnold D. Lurie, MD, Nassau
Roy H. Lieberman, MD, Rockland (Observer)

Tellers

Judithann Pryce, MD, Kings, *Chairperson*
Teresa S. Briggs, MD, Albany
Suresh Chandani, MD, Putnam
Hope Perry, MD, Tompkins
Norman W. Wetterau, MD, Seventh District Branch
Mitchell Miller, MD, Resident Physician Section (Observer)

NOMINATING COMMITTEE

To the House of Delegates, Ladies and Gentlemen:

In accordance with Article XI, Section 2 of the Bylaws of the Medical Society of the State of New York, the Nominating Committee met at the Society's headquarters, 420 Lakeville Road, Lake Success, New York, on Wednesday, November 6, 1991 at 11:00 a.m.

Members of the Committee present were:

Member-at-Large—**Robert A. Mayers, M.D.**, Westchester *Chairman*
First District Branch—**John A. Finkbeiner, M.D.**, New York
Second District Branch—**Samuel H. Gelfand, M.D.**, Nassau
Third District Branch—**Thomas Pemrick, M. D.**, Rensselaer
Fourth District Branch—**Richard A. Hughes, Sr., M.D.**, Warren
Fifth District Branch—**John C. Herrman, M.D.**, Lewis
Sixth District Branch—**Jeffrey A. Ribner, M.D.**, Broome
Seventh District Branch—**Charles D. Sherman Jr., M.D.**, Monroe
Eighth District Branch—**Bruce E. Baker, M.D**, Genesee
Ninth District Branch—**John A. Ramsdell, M.D.**, Westchester
Member-at-Large—**Antonio F. La Sorte, M.D.**, Broome

After careful consideration of the recommendations submitted by the district branches, county medical societies, and the resident, student and young physician sections in response to a memorandum, dated August 6, 1991, your Committee respectfully nominated the following candidates for election on March 22, 1992:

President—**Morton M. Kurtz, M.D.**, Queens
President-Elect—**Stanley A. Steckler, M.D.**, Suffolk
Vice President—**James H. Cosgriff, Jr., M.D.**, Erie
Secretary—**George Lim, M.D.**, Oneida
Assistant Secretary—**Joyce M. McChesney, M.D.**, Monroe
Treasurer—**Antonio F. LaSorte, M.D.**, Broome
Assistant Treasurer—**Stanley L. Grossman, M.D.**, Orange
Speaker—**Seymour R. Stall, M.D.**, Dutchess
Vice-Speaker—**John H. Carter, M.D.**, Albany
Councilors (four for three years)—**Bruce H. Berlin, M.D.**, Suffolk
—**Albert M. Ellman, M.D.**, Albany
—**Fred N. Flatau, M.D.**, Queens
—**Sanford A. Kaplan, M.D.**, Westchester
Resident Councilor–(one for one year)—****Paul Pipia, M.D.**, New York
Student Councilor–(one for one year)—***Leon Henderson**, New York
Trustees: (one for five years)—**Robert A. Mayers, M.D.**, Westchester

Nomination for one-year term as AMA Student Alternate Delegate
(Term: January 1, 1993–December 31, 1993)—Tanvir Choudhri, *New York.

Nominations for AMA Delegates—(two-year terms–1/1/93 to 12/31/94) and

Nominations for AMA Medical Student Delegate* and Resident Physician Delegate**—(one-year terms–1/1/93 to 12/31/93).

Charles N. Aswad, M.D., Broome
Bruce E. Baker, M.D., Genesee
Randall D. Bloomfield, M.D., Kings
Duane M. Cady, M.D., Onondaga
William A. Dolan, M.D., Monroe
Richard D. Eberle, M.D., Onondaga
Robert E. Fear, M.D., Suffolk
***Dawn Gangi**
Louis W. Giordano, M.D., Broom
Stanley L. Grossman, M.D., Orange
****Lawrence Hakim, M.D.**, Kings
Samuel H. Madell, M.D., New York
Robert A. Mayers, M.D., Westchester
Joyce M. McChesney, M.D., Monroe
Sidney Mishkin, M.D., Nassau
Donna B. O'Hare, M.D., New York
Jeffrey A. Ribner, M.D., Broome
William B. Rosenblatt, M.D., New York
Veroncia Santilli, M.D., Kings
Anthony P. Santomauro, M.D., Erie
Ralph E. Schlossman, M.D., Queens
Ralph M. Schwartz, M.D., Kings
Stanley A. Steckler, M.D., Suffolk
George T.C. Way, M.D., Dutchess

The twelve nominees receiving the largest number of votes will be delegates; the second twelve will be alternates.

Respectfully submitted:
Robert A. Mayers, M.D., *Chairman*

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*Medical Student
**Resident Physician

HOUSE COMMITTEE ON BYLAWS

To the House of Delegates, Ladies and Gentlemen:

The members of the House Committee on Bylaws are as follows:

Stanley F. Brunn, M.D., Chairman	Orange
Rodman D. Carter, M.D.	Otsego
Louis W. Giordano, M.D.	Broome
Robert M. Kohn, M.D.	Erie
Thomas Lansen, M.D.	Westchester
Sidney Mishkin, M.D.	Nassau
Donna B. O'Hare, M.D.	New York
Marilyn Pearl, M.D.	New York
Hilda Ratner, M.D.	Queens
Charles N. Aswad, M.D.	President, ex officio, Broome
George Lim, M.D.	Secretary, ex officio, Oneida
Seymour Stall, M.D.	Speaker, ex officio, Dutchess
Donald F. Foy	Executive Vice-President, ex officio
William R. Egan	Deputy Exec. Vice President, ex officio
Donald R. Moy, Esq.	General Counsel, ex officio

A meeting of the Reference Committee on Bylaws was held at the Hyatt Regency in Buffalo, New York on May 10, 1991. The Reference Committee prepared a transcript of testimony and a summary of its recommendations to the House Committee on Bylaws. The input received from the meeting of the Reference Committee on Bylaws proved to be of great assistance to the House Committee on Bylaws when it met on November 11, 1991.

Three resolutions asking for amendments to the Bylaws and one resolution asking for an amendment to the Principles of Professional Conduct were referred to the Committee for consideration.

Resolution 91-55, Date of Annual County Medical Society Meetings, When Officers Take Office

Introduced by the Fifth and Sixth District Branches

Whereas, Component county medical societies have sound reasons why they seek greater latitude in setting the date of the annual county medical society meeting at any time in the calendar year to meet local circumstances; and

Whereas, Interacting with State legislators has become an ever increasing priority for county medical society officers;and

Whereas, In the past the State Legislature adjourned prior to the Easter and Passover holidays; and

Whereas, The State Legislature now meets annually until at least July; and

Whereas, Changing county medical society officers in the 11th hour of the State legislative session can be a source of confusion in this vital governmental relations activity; therefore be it

Resolved, That the Bylaws of the Medical Society of the State of New York be amended to permit each component county medical society to determine when it wishes to have its annual meeting at any time in the calendar year and to permit each component county medical society to determine the date it wishes its officers to take office;

and be it further

Resolved, That Article XVIII, Section 4, paragraphs 2 and 3 of the Bylaws of the Medical Society of the State of New York be amended to read as follows, (deletions lined through):

"The annual meeting of each component county medical society shall be held ~~between January 1 and July 1 of each year~~ at a time which it shall determine.

All officers of the component county medical society shall enter on their official duties ~~between May 1 and July 1~~ at a date determined by the component county medical society and shall serve for a term of one year or until their successors shall have been duly elected and qualified."

The current requirements that "the annual meeting of each component county medical society shall be held between January 1 and July 1 of each year at a time which it shall determine" and "All officers of the component county medical society shall enter upon their official duties between May 1 and July 1 at a date determined by the county medical society and shall serve for a term of one year or until their successors shall have been duly elected and qualified", were added to the Bylaws of the Medical Society of the State of New York with the adoption of Resolution 70-16 by the House of Delegates in 1971. The whereas portion of Resolution 70-16 cited the following reasons for conformity:

- all successful membership organizations have conformity in their components
- conformity provides for more efficient and economical administration
- the lack of conformity among 61 county medical societies causes unnecessary waste of funds and staff time for the preparation and maintenance of numerous officer and committee rosters
- a degree of conformity in the assumption of office by the officers improves communications between the county medical societies and MSSNY.

The House Committee on Bylaws firmly believes that the reasons for adopting Resolution 70-16 in 1971 remain valid as reasons for leaving Article XVIII, Section 4 paragraphs 2 and 3 of the Bylaws of the Medical Society of the State of New York undisturbed. The members of the Committee believe that paragraphs 2 and 3 improve administration and communication without placing hardship on the component county medical societies. It was further stated that the lack of any conformity in the dates in which officers assume office would place hardship on the Speaker and Vice-Speaker of the House of Delegates in appointing reference committees.

Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 91-55 **NOT BE ADOPTED.**

Resolution 91-56, Non-Voting Councilor Representing the Hospital Medical Staff Section

Introduced by Lewis County Medical Society

Whereas, Many of the regulatory issues confronting the medical profession toady relate to medical practice in the hospital environment; and

Whereas, It is important to have a strong communications link between the MSSNY Council and the Society's hospital medical staff section; and

Whereas, This can best be accomplished through formal hospital medical staff section representation on the Council, and state medical societies have been encouraged by the AMA to provide for such representation; and

Whereas, Many other states have done this formally with mutual benefits to the Council and the HMSS; and

Whereas, The President of the Medical Society of the State of New York (with approval of the Board of Trustees) invited the chairperson of the HMSS to attend Council meetings as a guest this past year which has demonstrated the value of this practice which should now be codified in the MSSNY Bylaws; and

Whereas, The medical student and resident physician sections of the MSSNY are represented on the Council; therefore be it

Resolved, That the Bylaws of the medical society of the State of New York be amended to provide that the chairperson of the hospital medical staff section be a non-voting member of the MSSNY Council; and be it further

Resolved, That Article XII-A, paragraph 1 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions underlined):

"The hospital medical staff section is authorized to be represented in the House of Delegates by one delegate, and on the MSSNY Council by one councilor, who shall be the individual elected by the section as its chairperson. The hospital medical staff section shall represent physicians on the medical staffs of acute care hospitals located in New York State; of psychiatric inpatient facilities maintained by the State of New York or its political subdivisions; and of hospitals with inpatient programs; certified by the New York State Office of Mental Health."

and be it further

Resolved, That Article XI, Section 2, paragraph 2 under the heading "Nominating Committee" be amended to read as follows (additions underlined, deletions lined through):

"In the event that a member of the Nominating Committee who has been selected as the representative of a particular district branch (or his alternate) shall be unable to attend the meeting of this committee, the president shall appoint an at large member, from the district branch whose representative is absent, to represent the interests of that district on the Nominating Committee. It shall be the duty of this committee to propose and nominate members of the Medical Society of the State of New York for all vacancies to be filled at the ensuing annual meeting of the House of Delegates, with the exception of the councilor representing the hospital medical staff section. ~~These~~ Nominating Committee recommendations shall be made to the House of Delegates in the same manner as prescribed in Article X, Section 1, paragraph 2";

and be it further

Resolved, That Article IV, Section 1, paragraphs 1-5 be amended to read as follows (additions underlined, deletions lined through):

"The Council shall be composed of the president, president-elect, vice-president, immediate past-president, secretary, assistant secretary, treasurer, assistant treasurer, speaker, vice-speaker, chairman of the Board of Trustees and 15 councilors elected by the House of Delegates.

"The trustees, the executive vice-president, the deputy executive vice-president, and the general counsel of the Medical Society of the State of New York shall attend all meetings of the Council with voice but without vote. The councilor from the resident physician section shall attend all meetings of the Council with voice and with vote. The councilors from the medical student section and the hospital medical staff section shall attend all meetings of the Council with voice but without vote.

"Four councilors shall be elected annually by the House of Delegates, each for a term of three years. Two councilors, one councilor representing the medical student section and one councilor representing the resident physician section to the Medical Society of the State of New York shall be elected every year by the House of Delegates, each for a term of one year. One councilor representing the hospital medical staff section shall be elected every other year by the House of Delegates for a term of two years. Article IV, Section 1, paragraph 4 is not applicable to the term of office of a resident or student councilor, or a councilor representing the hospital medical staff section. In the event of a vacancy, a councilor shall be elected by the Council to serve until the next meeting of the House of Delegates at which time the House of Delegates shall elect a councilor to fill the unexpired term.

"No person shall serve, consecutively, more than two terms as councilor. An unexpired term shall not be construed as a term of office.

"The councilors shall assume office on election and shall hold office until their successors are duly elected and qualified.

"Councilors, other than the councilors representing the medical student ~~section~~ and ~~the councilor representing~~ the resident physician sections, and the hospital medical staff section shall be assigned to specific county societies as liaison for the Council in accordance with the provisions of Article V, Section 2. Councilors shall be required to disseminate information of Council activity as well as returning information to the Council, and shall report regularly to the Council on their activity. The councilor representing the medical student section must be a student member of the Medical Society of the State of New York; and the councilor representing the resident physician section must be a resident member of the Medical Society of the State of New York."

After thorough deliberation, the House Committee on Bylaws does not believe that the Bylaws need to be amended to provide for a non-voting councilor to represent the interests of hospital medical staffs. The Committee believes that the current composition of the Council is more than adequate to address the issues pertinent to hospital medical staffs.

The Committee notes that it is the custom of the President of the Medical Society of the State of New York to extend an invitation to the chairperson of the hospital medical staff section to attend Council meetings as an active participant with voice. The chairperson of the hospital medical staff section has regularly attended meetings of the Council and has substantially contributed in the deliberations of the Council. The Committee strongly recommends that future presidents of the Medical Society continue the practice of inviting the chairperson of the hospital medical staff section to attend meetings of the Council. It is not believed, however, that it is necessary to amend the Bylaws to designate the chairperson of the hospital medical staff section as a nonvoting councilor.

Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 91-56 **NOT BE ADOPTED.**

Resolution 91-87, Physician Ownership Interest in Facilities To Which They Refer Patients

Introduced by The New York State Radiological Society, Inc.

Whereas, The public and large numbers of physicians have expressed concern about the ethics of physicians referring their patients to facilities in which they have an ownership interest; and

Whereas, Perceived or real conflicts of interest involved in such arrangements reflect unfavorably on the medical profession; therefore be it

Resolved, That Chapter 1 (General Principles) of the Principles of Professional Conduct of the Medical Society of the State of new York be amended by adding a new Section 7 thereto to read as follows (additions underlined):

"Section 7: Physicians should not have a direct or indirect financial interest in diagnostic or therapeutic facilities to which they refer patients (except in rural areas)";

and be it further

Resolved, That Chapter II of the Principles of Professional Conduct be amended by the deletion of Section 6:b) which reads as follows (deletions lined through):

"b) ~~In owning a commercial medical facility for adjunct treatment purposes, a physician must keep the primary benefit of the patient in mind and such ownership should not result in overutilization for profit motives. Such ownership or participation must be approved by the local county medical society.~~"

The position of the American Medical Association's Council on Ethical and Judicial Affairs and the Medical Society of the State of New York is that physician ownership in a commercial venture with potential for abuse **IS NOT IN ITSELF UNETHICAL**. The AMA and MSSNY believe that the potential conflict of interest must be addressed by the following guidelines:

1. The physician has an affirmative ethical obligation to disclose to the patient or referring colleagues his or her ownership interest in the facility or therapy prior to utilization;
2. The physician may not exploit the patient in any way, as by inappropriate or unnecessary utilization;
3. The physician's activities must be in strict conformance with the law;
4. The patient should have free choice either to use the physician's proprietary facility or therapy or to seek the needed medical services elsewhere; and
5. When a physician's commercial interest conflicts so greatly with the patient's interest as to be incompatible, the physician should make alternative arrangements for the care of the patient.

It is strongly believed that physicians who exploit patients and who engage in inappropriate utilization should be sanctioned. The prohibition of all physician ownership-referral arrangements, however, is inappropriate and would adversely affect quality medical care.

Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 91-87 **NOT BE ADOPTED.**

Resolution 91-102, Dues Structure for Retired Physicians

Introduced by Ontario County Medical Society

Whereas, The American Medical Association's House of Delegates in December of 1990 adopted the recommendation of its Board of Trustees that the age limit for fully retired physicians on dues exemptions be reduced to age 65 and that, to be consistent, the age limit for partially retired physicians also be reduced to 65; and

Whereas, The dues structure of the Medical Society of the State of New York and the Ontario County Medical Society does not conform to the AMA's provisions; and

Whereas, The irregularities of dues regulations produce unnecessary hardship for the retiring physician; therefore be it

Resolved, That the Medical Society of the State of New York amend its Bylaws to conform to the dues exemption provision for retired physicians of the American Medical Association;

and be it further

Resolved, That Article II, paragraph 6 of the Bylaws of the Medical Society of the State of New York be amended to read as follows (additions underlined, deletions lined through):

"An active member in good standing who has completely or partially withdrawn from the active practice of medicine and who has been a member in good standing for the ten consecutive years prior to the attainment of the age of ~~sixty-seven~~ sixty-five years or an active member in good standing who continues in the active practice of medicine and who has been a member in good standing for the ten consecutive years prior to the attainment of the age of ~~seventy-two~~ seventy years, or an active member in good standing for ten consecutive years or more who is permanently disabled, may apply for life membership. The House of Delegates or the Council may waive the requirements in a prior case where there appear to be extenuating circumstances. He shall apply for such life membership to the component county medical society of which he is a member. His application shall be governed by the constitution and bylaws of the component county medical society relative to active membership.

Fiscal Implications: Loss of $561,275 of New York State Medical Society revenue, and a similar fiscal impact on county medical societies.

The adoption of this resolution would impose serious adverse financial consequences on MSSNY and the component county medical societies. At a time when critical issues pertaining to health care are being considered by executive, legislative and judicial bodies, and debated before the public and the media, it is more vital than ever that the Medical Society of the State of New York and the county medical societies remain highly active as an advocate for health care rights and quality medical care. The Committee is concerned that the adoption of this resolution would seriously undermine the ability of the organized medical profession to address vital and critical issues.

Accordingly, the House Committee on Bylaws unanimously recommends that Resolution 91-102 **NOT BE ADOPTED**

Your Chairman would like to thank, in addition to the members of the Committee and the physicians and other individuals who testified before it, those members of staff who helped this Committee: Donald R. Moy, Esq., Jay B. Silverman, Esq. and Deborah Gerrity.

Respectfully submitted
Stanley F. Brunn, M.D., *Chairman*

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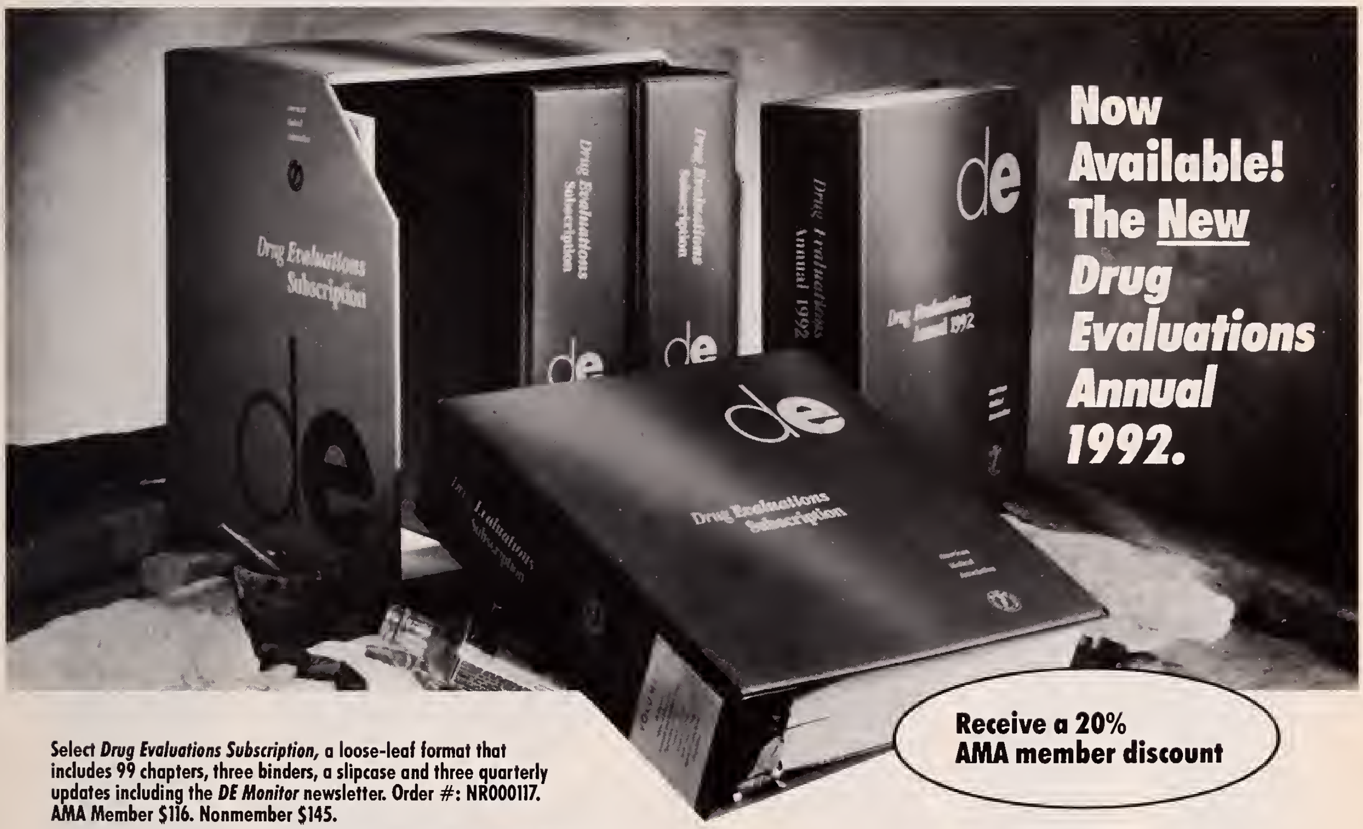

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See accompanying page for prescribing information.

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Contraindication: Known hypersensitivity to the drug. Because cross sensitivity in this class of compounds has been observed, H_2-receptor antagonists, including Axid, should not be administered to patients with a history of hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests—False-positive tests for urobilinogen with Multistix® may occur during therapy.

Drug Interactions—No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

PV 2093 AMP [101591]

Additional information available to the profession on request.

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(Continued from p 3A)

Feb 13–15. **25th Annual Recent Advances in Neurology.** San Francisco.

Feb 23–25. **Fifth Annual Congress of the North American Society of Phlebology on the Advances in Sclerotherapy and the Advances in Surgical and Medical Treatment of Venous Disease.** Grand Hyatt on Union Square, San Francisco. Contact: North American Society of Phlebology, 204 N El Camino Real, #E-234, Encinitas, CA 92024. Tel: (619) 942-8380.

Feb 27–Mar 1. **The American Society of Outpatient Surgeons 13th Annual Meeting.** Hyatt Islandia Hotel, San Diego. Contact: American Society of Outpatient Surgeons, 3960 Park Blvd, Suite E, San Diego, CA 92103. Tel: (619) 692-9918.

Feb 29–Mar 1. **Endoscopy Update 1992: The Southern California Society for Gastrointestinal Endoscopy Symposium.** Century Plaza Hotel, Los Angeles. Contact: Joyce M. Fried, Brain Research Institute, University of California, Center for the Health Sciences, Los Angeles, CA 90024-1761.

COLORADO

Feb 2–9. **Ninth Annual Symposium on Office Based Sports Medicine.** Tamarron Resort, Purgatory Ski Area, Durango. Contact: Extended Programs in Medical Education, University of California, Room LS-105, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Feb 17–20. **Advances in Sleep Medicine.** 17 ½ Cat I Credits. Breckenridge Hilton. Contact: Office of Continuing Medical Education, Albert Einstein College of Medicine, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

FLORIDA

Feb 3–7. **Second Annual MRI: Present and Future.** Ritz-Carlton Resort Hotel, Naples. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

Feb 3–6. **Fourth Annual International Symposium on Vascular Diagnosis and Intervention.** Fontainebleu Hilton, Miami Beach. Contact: Education Coordinator, Miami Vascular Institute, Baptist Hospital, 8900 N Kendall

(Continued on p 16A)

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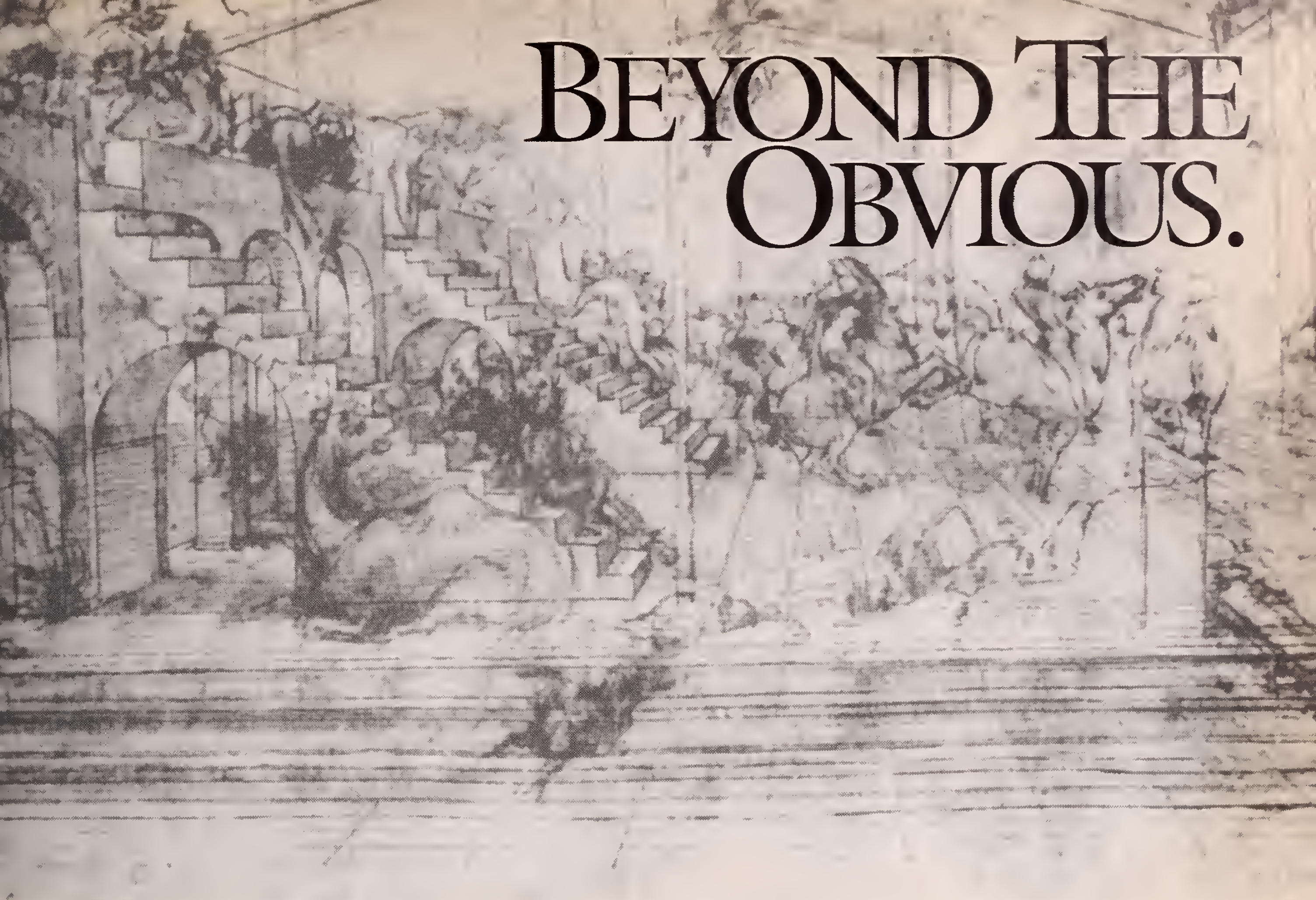

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MISCELLANEOUS—CONT'D

BONE UP ON OSTEOPOROSIS. A state of the art approach to diagnosis and management including case studies. Friday, March 20, 1992. Yale University School of Medicine. For registration information: Postgraduate & Continuing Medical Education, Yale University School of Medicine, 333 Cedar St., New Haven, CT 06510. Tel: (203) 785-4578.

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MISCELLANEOUS—CONT'D

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#5

(*Continued from p 11A*)

Dr, Miami, FL 33176. Tel: (305) 598-5992.

Feb 10–12. **15th Annual Gold Coast OB/GYN Conference.** 11 Cat I Credits.

Feb 10–14. **Controversies and Recent Advances in Surgical Pathology.** Disney's Contemporary Resort, Orlando. Contact: Education Division, Armed Forces Institute of Pathology, Washington, DC 20306-6000. Tel: (301) 4272-5231.

Feb 15–16. **Current Corneal Concepts.** 11 Cat I Credits. Good Samaritan Medical Center, West Palm Beach. Contact: Laura J. Keech, CME Coordinator, Good Samaritan Medical Center, PO Box 3166, West Palm Beach, FL 33402-4308. Tel: (407) 650-6236.

HAWAII

Feb 24–28. **Diagnostic Imaging Update: Body Imaging, Mammography, MRI and Interventional.** 23 ½ Cat I Credits. Ritz-Carlton, Mauna Lani. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773. ☐

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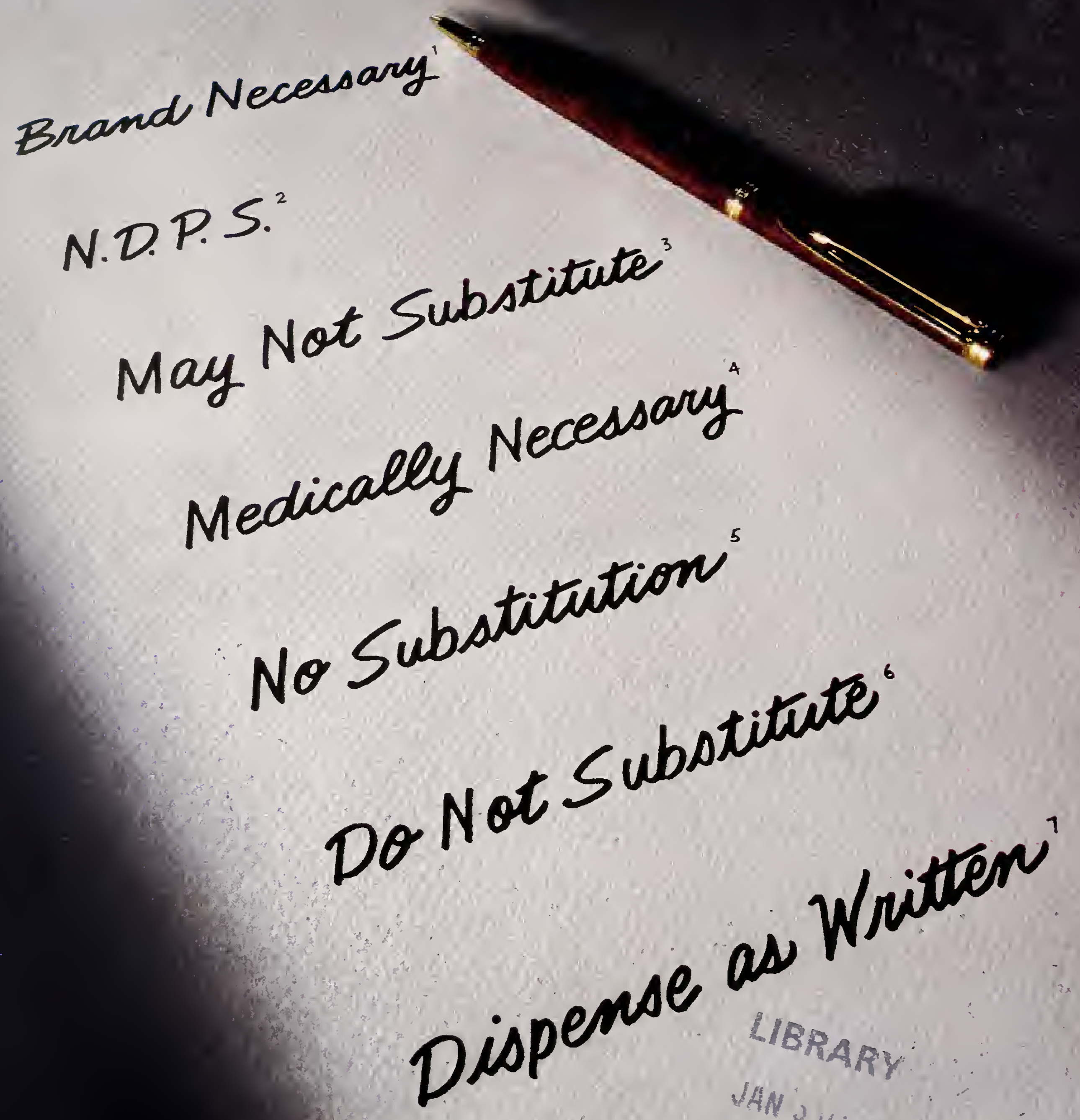

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New York State Journal of Medicine

MEDICAL SOCIETY OF THE STATE OF NEW YORK
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VOL 92
NO 2
FEBRUARY
1992

MSSNY UNIVERSAL HEALTH PLAN: A PROPOSAL FOR IMPROVING THE US HEALTH CARE SYSTEM

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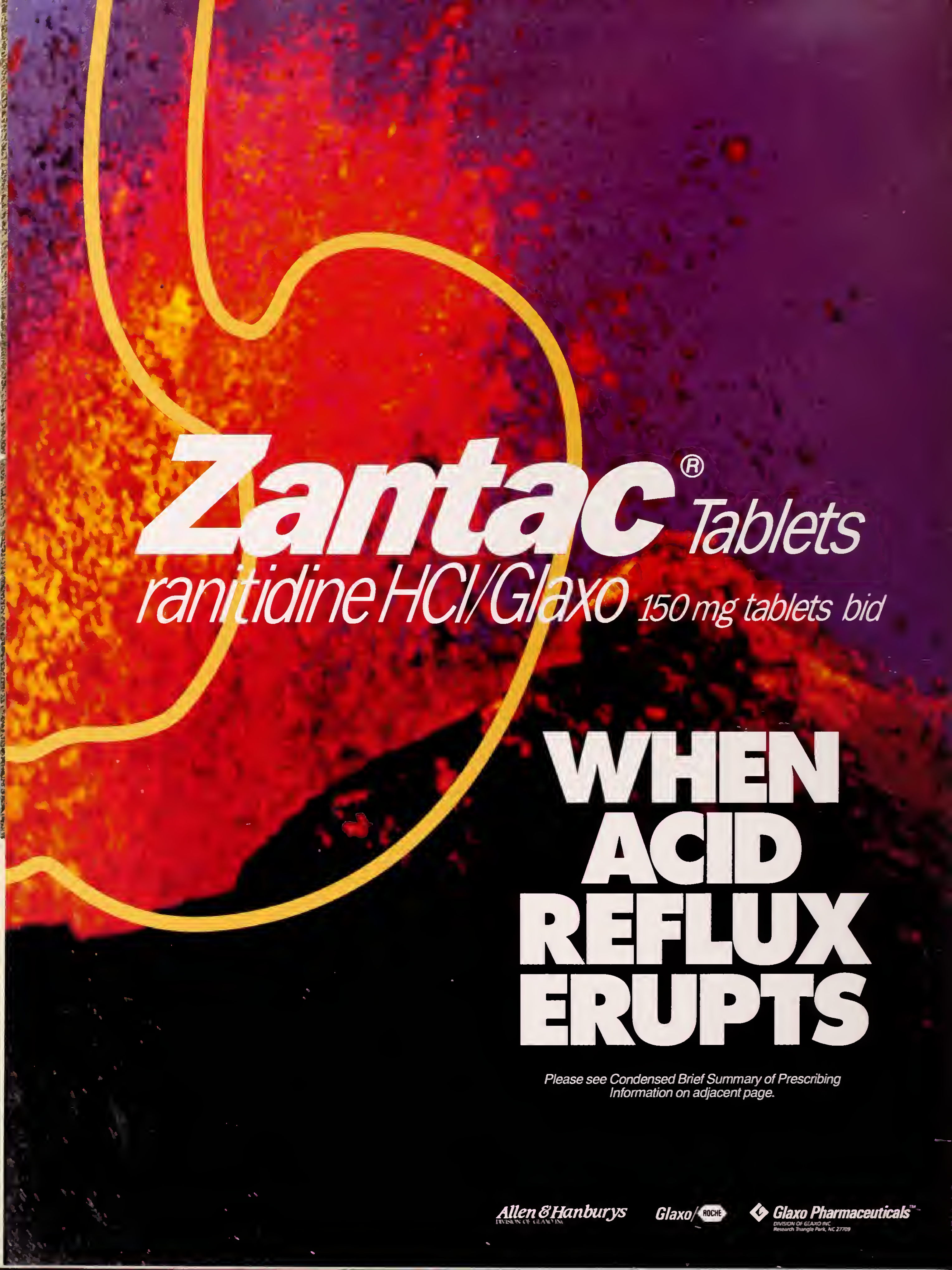

Zantac® Tablets

ranitidine HCl/Glaxo 150 mg tablets bid

WHEN ACID REFLUX ERUPTS

Please see Condensed Brief Summary of Prescribing Information on adjacent page.

Allen & Hanburys
DIVISION OF GLAXO INC

Glaxo/ROCHE

Glaxo Pharmaceuticals™
DIVISION OF GLAXO INC
Research Triangle Park, NC 27709

Zantac® 150 Tablets
(ranitidine hydrochloride)
Zantac® 300 Tablets
(ranitidine hydrochloride)
Zantac® Syrup
(ranitidine hydrochloride)

CONDENSED BRIEF SUMMARY

The following is a brief summary only. Before prescribing, see complete prescribing information in Zantac® product labeling.

INDICATIONS AND USAGE: Zantac® is indicated in:

1. Short-term treatment of **active duodenal ulcer.** Most patients heal within 4 weeks.
2. **Maintenance therapy** for duodenal ulcer patients at reduced dosage after healing of acute ulcers.
3. The treatment of **pathological hypersecretory conditions** (e.g., Zollinger-Ellison syndrome and systemic mastocytosis).
4. Short-term treatment of **active, benign gastric ulcer.** Most patients heal within 6 weeks and the usefulness of further treatment has not been demonstrated.
5. Treatment of **gastroesophageal reflux disease (GERD).** Symptomatic relief commonly occurs within 1 or 2 weeks after starting therapy and is maintained throughout a 6-week course of therapy.

In active duodenal ulcer; active, benign gastric ulcer; hypersecretory states; and GERD, concomitant antacids should be given as needed for relief of pain.

CONTRAINDICATIONS: Zantac® is contraindicated for patients known to have hypersensitivity to the drug.

PRECAUTIONS: General: 1. Symptomatic response to Zantac® therapy does not preclude the presence of gastric malignancy. 2. Since Zantac is excreted primarily by the kidney, dosage should be adjusted in patients with impaired renal function (see DOSAGE AND ADMINISTRATION). Caution should be observed in patients with hepatic dysfunction since Zantac is metabolized in the liver.

Laboratory Tests: False-positive tests for urine protein with Multistix® may occur during Zantac therapy, and therefore testing with sulfosalicylic acid is recommended.

Drug Interactions: Although recommended doses of Zantac do not inhibit the action of cytochrome P-450 enzymes in the liver, there have been isolated reports of drug interactions that suggest that Zantac may affect the bioavailability of certain drugs by some mechanism as yet unidentified (e.g., a pH-dependent effect on absorption or a change in volume of distribution).

Increased or decreased prothrombin times have been reported during concurrent use of ranitidine and warfarin. However, in human pharmacokinetic studies with dosages of ranitidine up to 400 mg per day, no interaction occurred; ranitidine had no effect on warfarin clearance or prothrombin time. The possibility of an interaction with warfarin at dosages of ranitidine higher than 400 mg per day has not been investigated.

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in rats and rabbits at doses up to 160 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to Zantac. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: Zantac is secreted in human milk. Caution should be exercised when Zantac is administered to a nursing mother.

Pediatric Use: Safety and effectiveness in children have not been established.

ADVERSE REACTIONS: Headache, sometimes severe, seems to be related to Zantac® administration. Constipation, diarrhea, nausea/vomiting, abdominal discomfort/pain, and, rarely, pancreatitis have been reported. There have been rare reports of malaise, dizziness, somnolence, insomnia, vertigo, tachycardia, bradycardia, atrioventricular block, premature ventricular beats, and arthralgias. Rare cases of reversible mental confusion, agitation, depression, and hallucinations have been reported, predominantly in severely ill elderly patients. Rare cases of reversible blurred vision suggestive of a change in accommodation have been reported.

In normal volunteers, SGPT values were increased to at least twice the pretreatment levels in 6 of 12 subjects receiving 100 mg q.i.d. intravenously for 7 days, and in 4 of 24 subjects receiving 50 mg q.i.d. intravenously for 5 days. There have been occasional reports of hepatitis, hepatocellular or hepatocanalicular or mixed, with or without jaundice. In such circumstances, ranitidine should be immediately discontinued. These events are usually reversible, but in exceedingly rare circumstances death has occurred.

Blood count changes (leukopenia, granulocytopenia, thrombocytopenia) have occurred in a few patients. These were usually reversible. Rare cases of agranulocytosis, pancytopenia, sometimes with marrow hypoplasia, and aplastic anemia and exceedingly rare cases of acquired immune hemolytic anemia have been reported.

Although controlled studies have shown no antiandrogenic activity, occasional cases of gynecomastia, impotence, and loss of libido have been reported in male patients receiving Zantac, but the incidence did not differ from that in the general population.

Incidents of rash, including rare cases suggestive of mild erythema multiforme, and, rarely, alopecia, have been reported, as well as rare cases of hypersensitivity reactions (e.g., bronchospasm, fever, rash, eosinophilia), anaphylaxis, angioneurotic edema, and small increases in serum creatinine.

OVERDOSAGE: Information concerning possible overdosage and its treatment appears in the full prescribing information.

DOSAGE AND ADMINISTRATION: (See complete prescribing information in Zantac® product labeling.)

Dosage Adjustment for Patients with Impaired Renal Function: On the basis of experience with a group of subjects with severely impaired renal function treated with Zantac, the recommended dosage in patients with a creatinine clearance less than 50 mL/min is 150 mg or 10 mL (2 teaspoonfuls equivalent to 150 mg of ranitidine) every 24 hours. Should the patient's condition require, the frequency of dosing may be increased to every 12 hours or even further with caution. Hemodialysis reduces the level of circulating ranitidine. Ideally, the dosing schedule should be adjusted so that the timing of a scheduled dose coincides with the end of hemodialysis.

HOW SUPPLIED: Zantac® 300 Tablets (ranitidine hydrochloride equivalent to 300 mg of ranitidine) are yellow, capsule-shaped tablets embossed with "ZANTAC 300" on one side and "Glaxo" on the other. They are available in bottles of 30 (NDC 0173-0393-40) tablets and unit dose packs of 100 (NDC 0173-0393-47) tablets.

Zantac® 150 Tablets (ranitidine hydrochloride equivalent to 150 mg of ranitidine) are white tablets embossed with "ZANTAC 150" on one side and "Glaxo" on the other. They are available in bottles of 60 (NDC 0173-0344-42) and 100 (NDC 0173-0344-09) tablets and unit dose packs of 100 (NDC 0173-0344-47) tablets.

Store between 15° and 30°C (59° and 86°F) in a dry place. Protect from light. Replace cap securely after each opening.

Zantac® Syrup, a clear, peppermint-flavored liquid, contains 16.8 mg of ranitidine hydrochloride equivalent to 15 mg of ranitidine per 1 mL in bottles of 16 fluid ounces (one pint) (NDC 0173-0383-54).

Store between 4° and 25°C (39° and 77°F). Dispense in tight, light-resistant containers as defined in the USP/NF.

September 1991

Glaxo Pharmaceuticals™
DIVISION OF GLAXO INC.
Research Triangle Park, NC 27709

Zantac® Syrup:
Manufactured by Roxane Laboratories, Inc., Columbus, OH 43216
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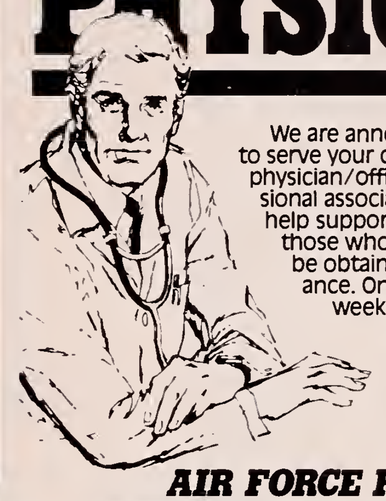

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The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

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Mar 5–7. **Ophthalmology Update.** Marriott. Contact: Office of Continuing Medical Education, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

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Mar 6–7. **Controversies in the Management of Breast Cancer.** Waldorf Astoria. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

Mar 16–20. **Seminar in Advanced Pheumatology.** NYU Medical Center. *Mar 21.* **Fiberoptic Endoscopy Workshop for the Management of the Difficult Airway.** NYU Medical Center Alumni Hall. *Mar 22–27.* **Anesthesiology Comprehensive Review XII.** Waldorf Astoria. *Mar 30–Apr 4.* **Comprehensive Review of Physical Medicine and Rehabilitation.** NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295.

Mar 18–20. **Advances in Cancer/Bone Marrow Transplantation.** New York Hilton. Contact: Office of Continuing Medical Education, Albert Einstein College of Medicine, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

Mar 19–21. **19th Annual Conference Language and Medical Symposia on Dyslexia.** Pennsylvania Hotel. Contact: NY Branch of the Orton Dyslexia Society, 80 Fifth Ave, New York, NY 10011. Tel: (212) 691-1930.

Mar 20–21. **Management of Cerebral Vascular Disease.** Columbia University. Contact: Center for Continuing Education, College of Physicians and Surgeons of Columbia University, 630 W 168th St, New York, NY 10032. Tel: (212) 305-3682. ☐

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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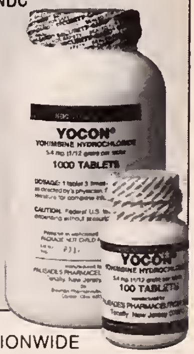

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New York State Journal of Medicine

FEBRUARY 1992 / Volume 92, Number 2

Contents

COMMENTARIES

41 **Cardiac tumors: A current perspective**
ERIC C. HANSON, MD

43 **Tunneled central venous catheter complications and the iatrogenic superior vena cava syndrome**
CARY A. PRESANT, MD

SPECIAL PROPOSAL

45 **Medical Society of the State of New York Universal Health Plan: A proposal for improving the United States health care system**
CHARLES N. ASWAD, MD, MARIO V. MENGHINI

RESEARCH PAPER

49 **Primary cardiac tumors: Diagnosis and management in 14 cases**
RICHARD A. MOGGIO, MD; ANTHONY L. PUCILLO, MD; ANDREW G. SCHECHTER, MD; RICHARD W. POOLEY, MD; MOHAN R. SARABU, MD; GEORGE E. REED, MD

REVIEW ARTICLE

53 **Part II: Special considerations in the treatment of epilepsy**
DAVID PRIMROSE, MD; CHAICHON LOCHARERNKUL, MD; WEBSTER H. PILCHER, MD, PhD; LINDA M. OJEMANN, MD; GEORGE A. OJEMANN, MD

SPECIAL ARTICLE

57 **Island Peer Review Organization educational posters**
RAPHAEL P. NENNER, MD

(continued on p 6A)

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New York State
Journal of Medicine

COMMITTEE REPORT

59 **Illicit drug use should not be legalized**
FRED ROSNER, MD; JONATHAN WOLPAW, MD; PHILIP H. SECHZER, MD; PETER ROGATZ, MD; HERMAN M. RISEMBERG, MD; FERNANDO V. ONA, MD; PATRICIA J. NUMANN, MD; ROBERT LOWENSTEIN, MD; LAURENCE LOEB, MD; ALLISON B. LANDOLT, MD; PIETER KARK, MD; VIRGINIA KANICK, MD; JOHN B. HENRY, MD; ABRAHAM L. HALPERN, MD; PETER B. FARNSWORTH, MD; ERIC J. CASSELL, MD; ANTHONY BUSCAGLIA, MD; ALLEN J. BENNETT, MD

ESSAY REVIEW

63 **The public health crisis in New York City**
PASCAL JAMES IMPERATO, MD

CASE REPORTS

65 **Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome**
GARY RICHMOND, MD; SANDRA HANDWERGER, MD; NORMAN SCHOENFELD, MD; WILFREDO TALAVERA, MD

67 **The strongyloides hyperinfection syndrome**
SYED A. JAMIL, MD, EILEEN HILTON, MD

69 **Gastrocolic fistula complicating a benign gastric ulcer**
MICHAEL S. BUTENSKY, MD, DAVID B. FALKENSTEIN, MD

LETTERS TO THE EDITOR

71 **Needle localization and biopsy in non-palpable lesions of the breast: An eight-year experience**
RAYMOND D. LARAJA, MD; ROBERT E. ROTHENBERG, MD; LAWRENCE HERBERT, MD; EDITH H. PRYCE, MD

71 **Phenytoin and smoking**
BHARAT S. SHAH, MD, HAROLD RATNER, MD

72 **Hospice care**
MARTIN R. LIEBOWITZ, MD

73 LEADS FROM EPIDEMIOLOGY NEWS

75 BOOK REVIEWS

83 BOOKS RECEIVED

84 OBITUARY LISTINGS

3A MEDICAL MEETINGS AND LECTURES

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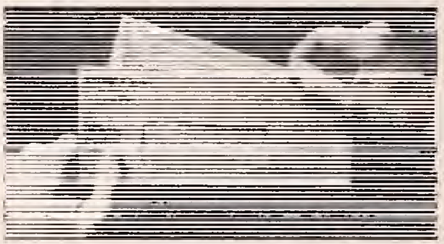

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COMMENTARIES

Cardiac tumors: A current perspective

Although cardiac tumors are rare, every practitioner may well be confronted with one over the course of his career. Unfortunately, most cardiac tumors are metastatic malignancies, which are exceedingly more common than primary cardiac tumors.[1] Usually, the discovery of a cardiac malignancy is only further evidence of generalized carcinomatosis in an extremely ill and dying patient. Generally, the diagnosis is made during postmortem examination and is only of intellectual interest. In most patients diagnosed during their lives, treatment is only palliative.

In the adult population, nearly two-thirds of primary cardiac tumors are benign, over half being myxomas, with a smaller number of lipomas. Angiosarcomas, rhabdomyosarcomas, mesotheliomas, and fibrosarcomas comprise the spectrum of the very rare primary cardiac malignancies.[1]

Apart from a metastatic cardiac tumor, one is most likely to encounter a myxoma. The typical patient diagnosed with this condition is a middle-aged female presenting to her physician or referred to a cardiologist with vague complaints, such as atypical chest pains, palpitations, or intermittent shortness of breath with few if any clinical findings.[2] The literature is filled with examples of unusual presentations of myxomas, such as devastating constitutional complaints (eg, fevers, night sweats, and weight loss); primary tumor emboli; severe right-sided heart failure mimicking mitral valve stenosis; and syncope. However, clinical events or debilitating symptoms usually appear late in the natural history of the tumor.[1–5] In large collected series, the majority of patients are moderately or severely symptomatic, since most were seen before the widespread use of modern diagnostic imaging and were diagnosed late.[3–5] Today, myxomas are frequently discovered in the asymptomatic or nearly asymptomatic patient who is essentially free of clinical findings. The universal application of first m-mode and now two-dimensional echocardiography in the evaluation of cardiac disease has led to the extremely timely discovery of these tumors.[2] Once discovered and confirmed by further studies, patients are promptly sent for surgery.

Because of the low mortality and morbidity of modern cardiac surgical techniques, the treatment for myxomas is quick, safe, and effective. Tumors nearly always appear on the left side of the atrial septum near the fossa ovalis and are easily explored through the left atrium. There, they are sharply excised or cauterized from the inner atrial septum. More extensive resection or reconstruction of the heart is unnecessary and unwarranted in most situations.[2] Myxomas are not malignant, do not spread, and infrequently recur with proper surgery.[2–5] Unfortunately, again, the literature is filled with fascinating reports of anomalous myxomas appearing in the right atrium or other chambers of the heart ($<10\%$), on or near heart valves, within chordae tendineae, in multiple locations, or recurring after surgery.[2–5] Such examples are of academic interest and are so exceedingly rare that they should not lead us to proceed with an intricate evaluation, or more important, a elaborate and more dangerous surgical treatment.

Papillary fibroelastomas and lipomas are the other benign tumors of the heart seen less commonly. They are of less clinical significance because they are usually discovered incidently at post mortem examination or at an unrelated surgery and rarely require any type of intervention or treatment.[1]

Primary cardiac malignancies on the other hand have a far different presentation and natural history.[1,6] Generally, they cannot be treated by surgery, although this is not always the case. Angiosarcomas usually envelop the right side of the heart. Their extensive size and pericardial involvement tend to make them inoperable and most patients die within a year of developing symptoms. Rhabdomyosarcomas, which are composed of malignant cells from striated muscle, can occur singly or in multiples anywhere throughout the heart. Some of these tumors are amenable to resection, but the prognosis is generally poor, even though less than long-term survivals have been reported. Fibrosarcomas, which also arise in single or multiple sites, can be resected. Prognosis depends on malignant behavior and the propensity to metastasize. Mesotheliomas usually envelop the pericardium diffusely but generally do not invade the heart. Although they may be very extensive, they can sometimes be resected.[6]

In spite of treatment, the recurrence rate for all primary cardiac malignancies is high, and long-term survival is generally poor. For those tumors that cannot be resected and have not metastasized, cardiac transplantation has been reported.[6,7]

In this month's issue of the *Journal*, Moggio et al present an interesting group of 14 patients with primary cardiac tumors: three malignancies and 11 myxomas. Each malignancy represents a different tumor histology. Two were merely biopsied, and the third tumor was partially resected.[8] Their experience is no different from that reported in the literature; no patient was resected for cure, and the medium-term survival was poor.[6]

Among those patients with myxomas, five presented with minor symptoms, two with atrial fibrillation, and four with embolic events. The large proportion of patients with emboli is unusual among modern series.[2–4] Most patients received expeditious surgery, some with associated coronary bypass grafting and/or mitral valve repair/replacement, and all have done well in sharp contrast with those patients with primary cardiac malignancies.

One patient required additional mitral valve replacement because of an iatrogenic injury to his mitral valve. Such surgical complications have been reported[5] and, I believe, will not occur if one avoids an aggressive surgical technique. Left atrial and not bi-atrial exploration is preferable in most situations, and resection of the tumor with a portion of intra-atrial septum and septal patching is unnecessary.[2]

This article, however, is important in that it describes and emphasizes the clinical use of preoperative magnetic resonance imaging (MRI) in the diagnosis and surgical management of cardiac tumors.[8] Other groups have found MRI scanning to be beneficial in differentiating between benign and malignant masses.[9,10] Cardiac tumors differ in their location of origin, structure, mobility, invasiveness, and multicentricity.[10–12] For instance, an intramyocardial lipoma may be suspected by its location and its consistency (fatty tissue vs. fibrous or calcified tissue) and may be followed without surgical intervention.[13,14] Strictly speaking, however, MRI cannot differentiate most solid tumors by composition alone, and other clinical data may be needed.[9,10] Nevertheless, the operability of malignant tumors can be assessed, and the exact location for biopsy can be determined.[9,10] After surgery, MRI can detect an early recurrence of a tumor, or if a patient receives chemotherapy, the result can be more easily quantitated.[15,16]

Although MRI has definite advantages in diagnosing and managing cardiac malignancies, transesophageal echocardiography (TEE), which is not mentioned in the article by Moggio et al, has been an especially helpful tool in managing myxomas.[17,18] TEE is more frequently available in most institutions, conveys most of the necessary information about this most common tumor, and has been our method of choice for more fully elucidating their position and size.[17,18] Frequently, it can distinguish left atrial clot from tumor, which is the other major alternative diagnosis when a left atrial mass is discovered.[19]

One is consistently impressed by TEE's real-time portrayal of myxomas. Most are extremely mobile, easily prolapse partially or totally through the mitral valve, and clearly illustrate the need for removal before their attaining large size and causing mitral valve obstruction or embolization. In our institution, TEE is performed just before surgery but can be done earlier if any diagnostic questions are raised. Unfortunately, TEE is of less value in assessing the broad spectrum of invasive malignant tumors, although there are reports to the contrary.[20,21]

With the availability of two-dimensional echocardiography, MRI, and TEE, intra-cardiac structures and masses have become more quickly, easily, and clearly defined for the physician. Diagnostic imaging methods will soon accurately determine the location and predict the histology of the vast majority of cardiac neoplasms. Earlier diagnosis and treatment of cardiac tumors, as well as other conditions, has become the norm, and this trend will only accelerate as present techniques are perfected and others are introduced.

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REFERENCES

1. McAllister HA: Primary tumors of the heart and pericardium. *Curr Probl Cardiol* 1979; 4:1–51.
2. Hanson EC, Gill CC, Razavi M, et al: The surgical treatment of atrial myxomas: Clinical experience and late results in 33 patients. *J Thorac Cardiovasc Surg* 1985; 89:298–303.
3. St John Sutton MG, Mevcier LA, Guilliani ER, et al: Atrial myxomas: A review of clinical experience in 40 patients. *Mayo Clinic Proc* 1980; 55:371–376.
4. Dein JR, Frist WH, Stinson EB, et al: Primary cardiac neoplasms: Early and late results of surgical treatment in 42 patients. *J Thorac Cardiovasc Surg* 1987; 93:502–511.
5. Larsson S, Lepore V, Kennergren C: Atrial myxomas: Results of 25 years experience and review of the literature. *Surgery* 1989; 105:695–698.
6. McAllister MC: Surgical treatment of cardiac tumors. *Card Surg St Art Rev* 1990; 4:437–464.
7. Aravot DJ, Banner NR, Madden B, et al: Primary cardiac tumors: Is there a place for cardiac transplantation? *Eur J Cardiothoric Surg* 1989; 3:521–524.
8. Moggio RA, Pucillo AL, Schechter AG, et al: Primary cardiac tumors: Diagnosis and management in 14 cases. *NY State J Med* 1992; 92:49–52.
9. Lund JT, Ehman RL, Julsrud PR, et al: Cardiac Masses: Assessment by MR imaging. *AJR* 1989; 152:469–473.
10. Ehman RL, Julsrud PR: Magnetic resonance imaging of the heart: Current status. *Mayo Clin Proc* 1989; 64:1134–1146.
11. Bavakos JA, Brown JJ, Higgins CB: MR imaging of secondary cardiac and paracardial lesions. *AJR* 1989; 153:47–50.
12. Watanabe AT, Teitelbaum GP, Henderson RW, et al: Magnetic resonance imaging of cardiac sarcomas. *J Thorac Imaging* 1989; 4:90–92.
13. Hananouchi GI, Goff WB: Cardiac lipoma: Six year follow-up with MRI characteristics, and a review of the literature. *Mag Res Imaging* 1990; 8:825–828.
14. Fisher MS, Edmonds PR: Lipomatous hypertrophy of the intra atrial septum: Diagnosis by magnetic resonance imaging. *J Compute Tomography* 1988; 12:267–269.
15. Bernier P, Patrax B, Duchateau C, et al: Primary left atrial angiosarcoma: Follow-up by magnetic resonance imaging. *Mag Res Imaging* 1990; 8:651–655.
16. Szucs RA, Rehr RB, Yanovich S, et al: Magnetic resonance imaging of cardiac rhabdoryosarcoma. Quantifying the response to chemotherapy. *Cancer* 1991; 67:2066–2070.
17. Vargas-Barron J, Romero-Cardenas A, Villegas M, et al: Transthoracic and transesophageal echocardiographic diagnosis of myxomas in four cardiac cavities. *A Heart J* 1991; 121:931–933.
18. Obeid AI, Marvasti M, Parker F, et al: Comparison of transthoracic and transesophageal echocardiography in the diagnosis of left atrial myxoma. *Am J Card* 1989; 63:1006–1008.
19. Aschenberg W, Schlutes M, Kremer P, et al: Transesophageal two-dimensional echocardiography for the detection of left atrial appendage thrombus. *JACC* 1986; 7:163–166.
20. Schwinger ME, Katz E, Rotterdan H, et al: Right atrial papillary fibroelastoma: Diagnosis by transthoracic and transesophgeal echocardiography and percutaneous transvenous biopsy. *Am Heart J* 1989; 118:1047–1050.
21. Schrem S, Colvin SB, Weinreb JC, et al: Metastatic cardiac liposarcoma: Diagnosis by transesophageal echocardiography and magnetic resonance imaging. *J Am Soc Echocardiography* 1990; 3:149–153.

Tunneled central venous catheter complications and the iatrogenic superior vena cava syndrome

Tunneled central venous long-term catheters have become common in the last decade. Multiple factors have favored the introduction of this technology. Patients prefer catheters because of increased convenience, avoidance of repeated venipuncture, and enhanced quality of life. Physicians favor catheter use because of frequently poor peripheral venous access, avoidance of sclerosing peripheral venous extravasation of vesicant medication, and the ability to administer hyperosmolar medications (such as high glucose concentration hyperalimentation fluids). Managed care systems and utilization reviewers value the use of central catheters since the ambulatory administration of antibiotics, nutritional support fluids, chemotherapy, and opioid analgesics permit earlier discharge of patients, and decrease the frequency of hospitalization.

Patients who most often have central venous catheters inserted are preoperative and postoperative (for nutritional support and antibiotics) or suffering from leukemia, lymphoma, solid tumors, or acquired immunodeficiency syndrome (AIDS). In our institution, 10% of surgical patients, 25% of leukemia and lymphoma patients, 25% of solid tumor patients, and 5% of AIDS patients have such catheters.

There has been considerable improvement in catheter technology over time. Hickman catheters, Broviac catheters, Leonard catheters, Groshong catheters, Hohn catheters, Quinton catheters, and subcutaneous infusion portals have permitted easier catheter introduction and resulted in a less frequent need for flushing, decreased infection rate, and avoidance of heparinization. Because of their frequency of use in hospitals and offices, consideration of the management of catheter-associated complications is important.

Complications of these catheters are frequent.[1] The most frequent complications are consequent to catheter placement: malposition, bleeding, and local symptoms. After healing of the introduction site, the most common complication is infection, occurring in about 15% of patients. Organisms causing these infections are most commonly *Staphylococcus epidermidis*, *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Escherichia coli*, and *Candida albicans*. Prompt diagnosis and treatment is paramount and almost always requires inpatient therapy. In our experience, although catheters may often be saved with intensive antibiotic treatment for the staphylococcus bacteremias, other systemic infections and tunnel site infections usually mandate catheter removal and systemic antibiotics. The ease of modern catheter insertion and removal should influence the prudent physician to tend to remove catheters in the presence of infection, rather than try to preserve an infection-associated catheter in place.

The second most common complication is clot formation. Clots may occur in the axillary vein, subclavian vein, or superior vena cava.[2] Prompt anticoagulation is necessary using heparin, and consideration should be given to thrombolytic therapy in severe cases. Catheter removal alone is insufficient to reverse catheter-associated thrombosis. With full therapeutic anticoagulation, in fact, catheter removal can sometimes be avoided.[2,3]

Once anticoagulation has been initiated, the etiologies of clotting complications observed with these catheters should be considered in evaluation and management. Equally frequently, as exemplified by the two patients reported in this issue of the *Journal*,[4] clots may be associated with infection. Catheter removal and appropriate systemic antibiotic treatment must be initiated immediately. Superior vena cava thrombosis and/or obstruction should raise the possibility of mediastinal tumor; this should be evaluated by computed axial tomography or magnetic resonance imaging and confirmed by biopsy if a mass is found. Radiotherapy and/or chemotherapy should immediately be added as appropriate to the tumor histology.[5] Lastly, the underlying disease may itself lead to a hypercoagulable state associated with the thrombotic complication.[6] Evaluation should be performed with appropriate coagulation tests. A hematology consultation is usually needed to confirm such a suspected hypercoagulable state, since prior anticoagulation with heparin usually has made the interpretation of the laboratory tests more difficult.

In this issue of the *Journal*,[4] Richmond et al present a scholarly review of two cases of superior vena cava syndrome associated with the use of central venous catheters in AIDS patients. The authors discuss diagnosis and management with particular emphasis on the unique problems of catheters in HIV-infected individuals.

The cases teach two additional lessons. First, the occurrence of bacterial and thrombotic complications three and seven days after catheter insertion illustrates the need for meticulous attention to postoperative care and should prompt the prudent physician to consider prophylactic antibiotic coverage perioperatively in immunocompromised individuals. Second, the appearance of superior vena cava syndrome was delayed until 24 and 72 hours after catheter removal and initiation of antibiotics. This indicates the seriousness of suppurative superior vena cava phlebitis, and its thrombogenic potential. Consideration should be given to short-term anticoagulation after catheter removal to prevent the morbidity and mortality of superior vena cava syndrome, as illustrated by four and six week courses of antibiotics and heparin in the case reports.

How can these complications be minimized? The patient, family, physician, and nurse all must be aware of the principles of catheter management. Meticulous catheter care by health professionals, and the patient is needed to minimize infections.[7] This includes cleansing, dressing changes, and appropriate interval flushing with aseptic technique. Although it has recently been proposed that occlusive dressings be avoided,[8] such a technique may be inappropriate for immunosuppressed individuals such as those with leukemia, lymphoma, solid tumors, transplants, and HIV-infection. Lastly, it has recently been reported in a small randomized prospective trial that monthly urokinase flushing reduces the occlusive complications (presumably thrombotic) in patients with central venous ports by 75%, and reduces infectious complications by 90%.[9] If confirmed, this urokinase technique may substantially increase the safety of these catheters.

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1. Moosa HH, Julian TB, Rosenfeld CS, et al: Complications of indwelling central venous catheters in bone marrow transplant recipients. *Surg Gynecol Obst* 1991 172:275–279.
2. Bertrand M, Presant CA, Klein L, et al: Iatrogenic superior vena cava syndrome. A new entity. *Cancer* 1984; 54:376–378.
3. Gould J, Skinner W, Carloss H: Groshong catheter-related deep venous thrombosis: Risk factors and analysis of long-term function in affected catheters. *Proc Amer Soc Clin Oncol* 1991; 10:340.
4. Richmond G, Handwerger S, Schoenfeld N, et al: Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome. *NY State J Med* 1991; 2:65–66.
5. Perez CA, Presant CA, Van Amburg AL: Management of superior vena cava syndrome. *Semin Oncol* 1978; 5:123–134.
6. Sack GH Jr, Levin J, Bell WR: Trousseau's syndrome and other manifestations of chronic disseminated coagulopathy in patients with neoplasms: Clinical, pathophysiologic, and therapeutic features. *Medicine* 1977; 56:1–37.
7. Wilson JM, Schaeffer N, Nolan KR, et al: Silicone catheters: Patient teaching pamphlet. *NITA* 1984; 7:169–172.
8. Ausman R, Martin R, Cole J, et al: "No Dressing" technique for central vein catheter care. *Proc Amer Soc Clin Oncol* 1991; 10:327.
9. Fraschini G, Becker M, Bruso P, et al: Comparative trial of urokinase vs heparin as prophylaxis for central venous ports. *Proc Amer Soc Clin Oncol* 1991; 10:337.

FROM THE LIBRARY

PRIMARY TUMOR OF THE HEART

COPLIN (Text Book of Pathology, p.503) states that about forty instances of primary cardiac neoplasms have been reported.

The condition is extremely rare.

Of the primary tumors occuring in this organ carcinoma, myxoma, lipoma, fibroma, myoma and their combinations are the only types that have been so far recorded, and of these fibroma is the most frequent.

Of the congenital tumors of the heart there have been collected from the literature eleven cases of rhabdomyoma, and one case further, the latest reported as far as ascertained, of Wohlbach *(Journal of Medical Research.* 1907. xvi, 495).

The interesting observation of an apparent relationship between congenital cardiac tumors and certain cerebral conditions, notably a sclerosis of gliomatous type, may have some bearing upon the case reported in this article. That embryonic muscle tumors of the heart wall are associated with sclerosis of the cerebral cortex was a fact first observed by von Recklinghausen and Virchow, and later described by Knox and Schorer (Abbott, in Osler's *Modern Medicine*).

Wohlbach's case was that of a child with hydrocephalus and spina bifida, and there was an ovoid nodule in the inter-ventricular septum and papillary muscle, which upon microscopical examination proved to be a rhabdomyoma. Wohlbach explains the combination as depending upon fetal mal-nutrition leading to vascular degeneration. There is no assurance that the tumor in the case reported here is of congenital origin, any more than may be the belief that all neoplasms are of embryological genesis, but the association of this tumor with the cerebral condition also found, may be suggestive of some relationship in the light of the above mentioned observation.

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Medical Society of the State of New York Universal Health Plan: A proposal for improving the United States health care system

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Executive Summary. Although the United States health care system is the best in the world, its successes are tempered by disturbing trends that continue to deteriorate and jeopardize access to quality health care. The continued and uncontrolled growth in health care costs makes it increasingly difficult to afford the level of care expected by our citizens. Furthermore, as difficult as it may be to conceive, over 33 million Americans are without health insurance as the result of unemployment, low-paying jobs, or health care coverage deemed to be unaffordable. Meanwhile, concomitant with the proliferation of critical health-related problems that are societal in nature and beyond the control of Medicine (eg, AIDS, drug abuse, alcohol abuse, poverty, homelessness, victims of violence), the nation's health care expenditure has risen from $12.7 billion, or about $1 billion per month in 1950, to $600 billion in 1989, or $1.5 billion per day. In fact, the cost of health care provided to Americans in 1990 reportedly reached $675 billion, roughly approaching $2 billion a day and approximately $2,500 per year for each man, woman, and child in the United States. In fact, latest federal governmental reports indicate $738 billion spent for health care in 1991, with $817 billion estimated for 1992.

With mounting pressures emanating from state and federal proposals stressing increased employer funding as a key element, a financially beleaguered business sector, which has experienced a critical escalation in costs for employee health care benefits, in turn advocates a nationalized health insurance initiative financed by the government.

Therefore, in view of the (a) inadequacies of current national health care schemes in other countries (including Canada); (b) waste and inefficiency associated with the administration of current health insurance programs encompassing Medicaid and Medicare; and (c) perceived inadequacies inherent in current state and federal health care initiatives, this report proposes an appropriate benefits package for the United States population. The proposed package is predicated on benefit structures carefully developed under the Health Policy Agenda (HPA) for the American People.

Consequently, the Medical Society of the State of New York (MSSNY) advocates the following:

- An appropriate and equitably coordinated Universal Health Plan (UHP), which could be administered through the existing private and voluntary health insurance industry, with (a) free-market competition emerging as the stabilizing factor within the industry; and (b) current health insurance oversight mechanisms kept intact and enhanced (with no costs necessary for establishment of new administrative oversight entities);
- Dissolution of the Medicaid and Medicare programs, thus eliminating the gross inefficiencies, related costs, and complexities associated with these bureaucratically controlled processes;
- An appropriate benefits package under the proposed system for the United States population predicated on the HPA for the American People Subcommittee on Basic Benefits proposal;
- Required purchase of such an appropriate benefits package by all Americans (not possessing voluntarily provided employer-based coverage) through *a tax credit mechanism*, with any additional coverage procured as desired at the expense of purchasers;
- Amendment of the personal income tax code to allow for the recommended tax-credit mechanism based on sliding income scales to provide relief to individuals above 200% of the poverty level (up to a prescribed income ceiling limitation) concerning out-of-pocket expenses for purchase of an established appropriate benefits package from an insurer of choice;
- Provision of federal vouchers for individuals below 200% of the poverty level, negotiable only for purchasing an established appropriate benefits program from an insurer of choice;
- Long-term care coverage financed through (a) the initial premium cost of the HPA basic package, which includes such services; (b) encouraged purchase of riders for extended long-term coverage by individuals above 200% of the poverty level, with tax credits provided based on a sliding income scale; and (c) supplementary federal voucher support to the more needy individuals with incomes below 200% of the poverty level;
- Shared risk selection among participating private and voluntary health insurers through the required acceptance of high-risk individuals in direct proportion to the number of low-risks enrolled;
- A cap on the portion of the premium dollar a health insurer may use for administrative expenses at the risk of absorbing any costs in excess of the prescribed limitation;
- Provision of an option for individuals to purchase untaxed Health Individual Retirement Accounts (HIRAs) to supplement health care costs for unexpected expenses (such as costs associated with catastrophic illnesses);
- Inclusion of deductibles and co-insurance features in any appropriate benefits program for individuals above 200% of the poverty level to ensure affordable premiums and control use of services;
- Inclusion of frequency of services parameters (with meaningful physician input concerning their development) to certify the quality and cost-effectiveness of the advocated program;
- A physician-controlled utilization- and quality-assurance process at state levels, complemented by similar activities at local levels to ensure the quality, necessity, and the appropriateness of the services provided;
- Vigorous Internal Revenue Service (IRS) pursuit of known tax-evaders as well as IRS establishment of an effective monitoring process to detect and penalize individuals avoiding taxation

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- while thriving in the "underground economy" (and who would be subject to exposure on entering the new health care system), with all such recouped monies contributed to funding the new system;
- Consideration by the congress of the foregoing concepts and objectives including congressional determination of the affordability (ie, annually assessed cost) of an insurance package predicated on the HPA basic benefits structure.

IMPROVING THE UNITED STATES HEALTH CARE SYSTEM

The United States population benefits from a health care system that is the best in the world, with an increased life expectancy for Americans of 69.7 years in 1960 to 74.7 years in 1985.[1] Furthermore, life expectancy in this country is anticipated to continue increasing into the next century. In fact, the following are the results of our pluralistic system:

- Innovative developments in clinical and health care delivery are being emulated by other industrialized nations;
- Availability of state-of-the-art technology encompassing CT and PET scanners, ultrasound, magnetic resonance imaging, and new medical procedures, such as organ transplants and kidney dialysis has greatly improved diagnosis and treatment;
- Constant introduction of new drugs has significantly improved treatment regimens;
- Consumers may choose from many thoroughly trained physicians practicing within a multiplicity of settings.

Yet, these successes are tempered by the continuing and disturbing trends that jeopardize access to health care and the high quality of care expected by the citizens of this country. Concomitant with the proliferation of critical health-related problems that are societal in nature and beyond the control of Medicine (eg, AIDS, drug abuse, alcohol abuse, poverty, homelessness, victims of violence), these concerns evolve from the following:

- The sustained, marked, and seemingly uncontrollable growth in health care costs emanating from numerous factors, inclusive of societal behavior, increased life expectancy, technological advancements, etc, that make it increasingly difficult to afford the level of care Americans have come to expect;
- The irreconcilable fact that too many people across the country are underinsured or have no health insurance at all.

Although the United States has experienced major improvement in the health status of its citizens, many questions have arisen as to whether the increased health care spending is yielding commensurate benefits in terms of improved health or quality of life. Many millions of people are constrained from equal participation in the United States health care system since they (a) are unemployed, (b) have low-paying jobs and simply cannot afford health insurance, or (c) have inadequate coverage.

As implausible as it may seem in this country, the following is true:

- Approximately 33 million Americans (ie, 13% of the United States population) are uninsured, of which more than 70% are either employed or the dependents of employees (ie, approximately 24 million of the total 33 million uninsured)[2]
- 70 million are underinsured;[1]
- 23 million are covered by Medicaid and are not guaranteed access to medical treatment or equity in the quality of the medical care received.[1]

Consequently, our health care system has created a moral dilemma with significant implications of a separation between rich and poor and young and old concerning access to health care services.

To underscore the problem, testimony at congressional hearings[1] on the future of health care in America identified the following:

- The United States population is aging, with 12% of the current total 65 years or older. (NOTE: It is estimated that by the year 2050 almost one in four people will attain age 65, and of that total, 20% will be aged 85 and over compared with 10% today);
- In 1950, the nation spent $12.7 billion on health care, with spending increasing to over $550 billion in 1988, and $600 billion in 1989, ie, almost $2,200 per person or $7,000 per family. (In fact, the cost of health care provided to Americans in 1990 reportedly reached $675 billion,[3] approaching, roughly, a staggering $2 billion a day and approximately $2,500 per year for each man, woman, and child in the United States). Latest federal government reports indicate that $738 billion was expended in 1991, with over $817 billion projected for 1992;
- The United States share of the GNP devoted to health care rose from 4.4% in 1950 to 11.5% in 1988, more than any other industrialized nation (at 6%–9%) and projected to grow to 15% by the end of the century;
- The United States health care system is the most bureaucratic in the world with paperwork adding an approximate $20 surcharge for each $100 spent on health care (ie, 20% of the nation's total expenditure for health care; eg, $147 billion calculated on the basis of the reported 1990 national expenditure for health care of $738 billion);
- Skyrocketing medical liability premiums in recent years have not only added to the cost of health care, (they comprise 16% of the increase in physician fees and add another $10 billion per year in defensive medicine), but have also had a severe impact on the availability of certain physician specialties (ie, obstetrics and family practice).

The following recommendations emanated from the aformentioned Congressional hearings:

- The establishment of a national health insurance program, preferably modeled after the Canadian approach. (MSSNY NOTE: The American Medical Association (AMA) and a growing number of legislators and media have assessed this system as perhaps more conducive to Canadians but not the United States, particularly because of our public expectation of unlimited access to health care. Furthermore, although Canada and other countries with national health schemes spend less per capita than the United States, their citizens (a) receive less care, (b) have restricted access to technology, and (c) are taxed at a higher rate than Americans. Consequently, the Canadian system with too few access points to health care is less responsive to consumers than the admittedly often duplicative United States system, with rationing of care emphasized to constrain demand and stabilize health care costs, and waiting lists for most elective surgeries being commonplace. In fact, in the last three decades,[3] the cost of health care in Canada is reported to have risen at a rate equal to or greater than in the United States. Consequently, the evidence seems to indicate that a "Canadian solution" would not be effective in controlling our greatest threat—out-of-control health care inflation.);
- The reform of the Medicare program with emphasis on substantial mitigation of a complicated, unwieldy, and costly program and simplification of its current complex reimbursement system. (NOTE: A revolutionary and extremely complex Medicare Physician Payment Reform predicated on the Resource Based Relative Value Scale reimbursement system became effective January 1, 1992.);
- A change in the focus of the health care system from sickness to wellness care, with health insurance coverage for preventive care services meaningfully encouraged;
- The development of alternative methods for resolving medical liability cases;
- Greater attention in the research area to the diseases afflicting the elderly (ie, arthritis, dementia, incontinence);
- Public and professional education toward changing attitudes concerning responsibilities in the maintenance of health, with emphasis on patient acceptance of significant responsibility for management of their own health, thus eventually mitigating health care costs.

Although aware that the Congress is currently addressing the foregoing problems and issues associated with the current health care system, the physician community remains apprehensive concerning a number of these recommendations and the remedial measures the federal bureaucracy may eventually wish to employ to reform the system. Consequently, there is a growing sentiment among the physician community that a Universal Health Care[4] Program through the private sector, replacing the current health care system inclusive of Medicare, Medicaid, and other health insurance programs, may be a viable solution. This sentiment generally embodies the belief that any additions to current federal and state health insurance programs are unaffordable and would only compound and perpetuate existing problems. The belief is that the substitution of a UHP through the private sector, with (a) enhanced utilization controls; and (b) enhanced individual cost-participation, thus stimulating meaningful individual cost-awareness and sensible economies, would be more cost-efficient and equitable for everyone—particularly since it would purportedly reduce the enormous waste and inefficiency associated with the current health care system.

Concomitantly, it should be noted that:

- The business sector, which has reportedly been responsible for providing 64.3% of health insurance coverage[5] at considerable and increasing expense, favors a nationalized health insurance system funded by the federal government to relieve the growing employer financial role in the health insurance process. In fact, the US Chamber of Commerce has advanced an initiative for National Health Insurance;
- The AMA has embarked on promotion of its Health Access America initiative, which provides a set of principles essentially calling for substantial reform of the Medicare and Medicaid programs; increase in employer-provided insurance for the two thirds of the uninsured who are employed; tax credits to small business; risk pools for the uninsured; catastrophic care coverage; and state-level risk pools for the remainder of the uninsured population;
- The Heritage Foundation[6] strategy for a national health system would reform the basic tax treatment for health care insurance, thus focusing on assistance to individuals predicated on economic considerations. (It is noteworthy that the Foundation's strategy contains marked similarities to the MSSNY proposal for Improving the United States Health Care System.)
- With access to health care becoming an increasingly critical issue at the congressional level, over 31 universal health-type bills have been introduced to date. Currently, some of the more publicized such initiatives are: HR1300, The Universal Health Care Act of 1991 introduced by Representative Marty Russo (D-IL), Vice-Chairman of the Ways and Means Subcommittee on Health. The proposal espouses a single-payor national health care plan that would replace private health insurance programs, Medicare, Medicaid, and veteran's health programs with a single program administered by the Department of Health and Human Services. A 6% payroll tax on employers and an increase in the corporate income tax for businesses with more than $75,000 in profits would be used to finance the program, which would include a wide range of benefits, without application of co-payments or deductibles; Health America, HR2530/S.1227, spearheaded by Senator George Mitchell, espouses basic health insurance coverage for all Americans through (a) a "play-or-pay" approach wherein employers either provide health insurance for workers or pay a payroll tax toward this new public program; and (b) a federal/state insurance program called "Americare" to replace Medicaid; The Health Insurance Reform and Cost Control Act of 1991 (HR3626) introduced by Representative Dan Rostenkowski, Chairman Committee on Ways and Means, is an *incremental* proposal designed to "jump-start" the legislative process as well as stimulate debate on health care reform, particularly since the administration has not taken a firm stand on this issue. The bill primarily provides for (a) cost containment predicated on the new Medicare physician payment methodology; and (b) a federal/state system of regulating insurers in the provision of coverage to small groups on a required community-rated basis.

With this nationwide crisis proliferating, it is not surprising that some larger states have been endeavoring to cope with increased health care costs, diminishing federal contributions to Medicare and Medicaid programs resulting in a cost-shifting to other sectors, and the growing number of uninsureds who are significantly contributing to the financial drain in state health care delivery systems. The following are examples:

- Massachusetts attempted to respond to this problem through its Health Security Act ("Health Care For All"), which essentially collapsed because the state's economy, lack of support from small business, and the state's governor who asked the legislature to repeal the provision.
- California with its 5.2 million uninsureds (25% of the state's population) endeavored to come to grips with the problem through several initiatives including a Department of Health sponsored Universal Health Care Act as well as through a California Medical Association (CMA) sponsored basic benefits initiative for access to health care. The CMA eventually suspended efforts to place its initiative on a 1992 ballot and challenged critics to develop a meaningful solution to the state's health care crisis.
- In New York, where an estimated $50 billion is spent on health care of which over $11.5 billion is expended for the State Medicaid Program covering 2.3 million recipients (roughly, an average cost of $5,000 per recipient), town meetings on Universal Health Care sponsored by Governor Cuomo and the League of Women Voters were held beginning June 1991 and culminating in November 1991. The meetings focused on four prominent initiatives including (a) the MSSNY UHP, a proposal for improving the United States health care system; (b) the Hospital Association of New York State (HANYS) "Pro-Health" initiative calling for multiple funding sources and the enhancement of the Medicare and Medicaid programs; (c) the Citizen Action of New York Proposal, New York Health, calling for health coverage for everyone in New York State with envisioned financing through elimination of administrative waste under the current multi-payer system; federal, state, and local taxes; employer payroll taxes; and (d) the New York State Department of Health Universal New York Health Care Program (UNY*Care), which contains similarities to the Massachusetts initiative and the Canadian health care system and is anticipated by the state to effectively address the problem of the 2.5 million uninsured, the population at or below 200% of the poverty level, and the remainder of the state community through a three-phase approach. It should also be noted that the managed care concept, which has been receiving significant attention by legislators, insurers, consumers, providers of health care, and the media, is currently being adapted to the New York State Medicaid Program through statewide demonstrations on local levels in an effort to control costs while improving access to health care for recipients. Consistent with a pluralistic system, managed care is expected to be tested and evaluated by the medical community for effectiveness in enhancing patient access to quality care, provided that physicians may continue to participate in this process on a voluntary basis.

In addition to the foregoing considerations, state mandates for health insurance coverage of specific services including home health care, maternity care, pre-admission testing before surgery, emergency medical services in hospital facilities, second surgical opinions, outpatient treatment of alcoholism and drug abuse, and annual mammography screening for patients 50 years or older, although laudable in intent, have increased costs to employers, increased cost sharing for employees, and encouraged large companies to self-insure to avoid these costly state mandates. Purportedly, small- or medium-sized businesses that cannot afford to self-insure, or provide health insurance inclusive of these state-mandated services, either cancel purchased coverage or opt not to provide such coverage at all. This appears to have contributed to a significant number of the uninsured total comprised of employees and their dependents. As a result, this segment of the population seems either unable to afford health insurance, or of necessity, risks financial distress by not purchasing coverage.

However, there is a growing dilemma emerging from differing viewpoints concerning the impact of state mandates. Maternity care, which is the most essential element of any benefits package developed for needy pregnant women, constitutes the most costly component of the New York State mandated benefits (ie, according to Blue Cross Blue Shield data, almost 70% of the premium dollar increase attributed to the mandates applies to maternity benefits).

Consequently, further independent study and assessments are required to reconcile the significantly disparate views relative to state-mandated benefits, with particular focus on maternity services, which are essential to the establishment of an urgently required benefits package addressing the needs of indigent and low-income pregnant women.

Since the spiraling health care costs associated with federal and state health insurance initiatives such as Medicare and Medicaid are expected to rise at significant rates, it appears time to espouse a more significant role for the private sector in the health insurance arena through federally sourced funding.

Consequently, this position paper advocates the following:

- An appropriate and equitably coordinated UHP that could be administered through the existing private and voluntary health insurance industry, with (a) free-market competition emerging as the stabilizing factor within the industry, and (b) current health insurance oversight mechanisms kept intact and enhanced (with no costs necessary for establishment of new administrative oversight entities);
- Dissolution of the Medicaid and Medicare programs, thus eliminating the gross inefficiencies, spiraling costs, and complexities associated with these bureaucratically controlled processes;
- An appropriate benefits package under the proposed system for the United States population predicated on the HPA for the American People Subcommittee on Basic Benefits proposal.[7] (NOTE: The estimated cost of the package would be in the vicinity of $2,500. This is predicated on the calculated cost of a similar AMA-developed benefits package featuring deductibles and coinsurance amounts by a leading actuarial firm as identified in AMA Board of Trustees Report KK (A-90).[8]
- Required purchase of such an appropriate benefits package by all Americans, not possessing voluntary employer-based coverage, through *a tax credit mechanism* (with any additional coverage procured as desired at the expense of purchasers);
- Amendment of the personal income tax code to allow for the recommended tax-credit mechanism based on sliding-income scales to provide relief to individuals above 200% of the poverty level (up to a prescribed income ceiling limitation) concerning out-of-pocket expenses for purchase of an established appropriate benefits package from an insurer of choice;[9]
- Provision of federal vouchers for individuals below 200% of the poverty level, negotiable only for purchasing an established appropriate benefits program from an insurer of choice;
- Long-term care coverage financed through (a) the initial premium cost of the HPA basic package, which includes such services; (b) encouraged purchase of riders for extended long-term coverage by individuals above 200% of the poverty level, with tax credits provided based on a sliding income scale; and (c) supplementary federal voucher support to the more needy individuals with incomes below 200% of the poverty level;
- Shared risk selection among participating private and voluntary health insurers through the required acceptance of high-risk individuals in direct proportion to the number of low-risks enrolled;
- A cap on the portion of the premium dollar a health insurer may use for administrative expenses at the risk of absorbing any costs in excess of the prescribed limitation;
- Provision of an option for individuals to purchase untaxed HIRAs to supplement health care costs for unexpected expenses (such as costs associated with catastrophic illnesses);[9]
- Inclusion of deductibles and co-insurance features in any appropriate benefits program for individuals above 200% of the poverty level to ensure affordable premiums and to control use of services;
- Inclusion of frequency of services parameters (with meaningful physician input concerning their development) to certify the quality and cost-effectiveness of the advocated program;
- A physician-controlled utilization- and quality-assurance process at state levels, complemented by similar activities at local levels to ensure the quality, necessity, and the appropriateness of the services provided;
- Vigorous IRS pursuit of known tax-evaders, as well as IRS establishment of an effective monitoring process to detect and penalize individuals avoiding taxation while thriving in the "underground economy" (and who would be subject to exposure on entering the new health care system), with all such recouped monies contributed to funding the new system.
- Consideration by the Congress of the foregoing concepts and objectives including Congressional determination of the affordability (ie, annually assessed cost) of an insurance package predicated on the HPA benefits structure).

While it may be that no single proposal currently advocated will prevail intact, we must continue to express our views and meaningfully participate in the health care delivery reform process.

REFERENCES

1. Staff Report of the Congressional Subcommittee on Education and Health of the Joint Economic Committee on The Future of Health Care in America, Washington, DC, 1989.
2. Health Access America. The AMA proposal to improve access to affordable, quality health care. Chicago, Illinois, American Medical Association, 1990.
3. Laszewski RL: Accountability: The Missing Link in our Health Care System. Presented at the Public Issues Lecture Series, New England Medical Center, 1991; Boston, Ma. Cardillo TE: Access to Health Care for All Americans, Bulletin of the Monroe County Medical Society, 1989; 55:5.
4. Cure or Cause, The Public Policy Institute of New York State, 1989.
5. Butler SM, Haislmaier E: Critical Issues: A National Health System for America, Washington, DC, The Heritage Foundation, 1989.
6. The Health Policy Agenda (HPA) for the American People, Report of the Subcommittee on Health on Basic Benefits, Chicago, Illinois, American Medical Association, 1988.
7. American Medical Association Board of Trustees Report KK Minimum Benefits in Required Employer Health Insurance, Chicago, Illinois, American Medical Association, 1990.
8. American Medical Association Board of Trustees Report MM Proposal for Financing Health Care of the Elderly, Chicago, Illinois, American Medical Association, 1986.

Primary cardiac tumors

Diagnosis and management in 14 cases

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ABSTRACT. In our experience over a 10-year period at Westchester County Medical Center (Valhalla, NY), we diagnosed 11 left atrial myxomas and three primary cardiac malignancies in ten females and four males, aged 18–74 years. Two-dimensional echocardiography enabled the correct diagnosis of these lesions. Detailed characteristics of the cardiac tumors were provided by magnetic resonance imaging (MRI) scanning in five patients. Two of three malignancies were only biopsied; the myxomas were completely excised. We emphasized removal of tumor and its attachments to the atrial wall, accomplished by left or biatrial incision, full-thickness excision of the area of attachment, and pericardial patch closure of the atrial or free-wall defects. Associated procedures included mitral valve repair (three) and coronary artery bypass grafting (two). There was no mortality associated with the surgery. Two patients with malignancy died three and 36 months after surgery; a third is alive at 30 months after surgery. No patients with myxoma has had recurrence from 16 to 100 months. Preoperative diagnosis of cardiac tumors is enhanced by MRI scanning and may help in planning the surgical procedure. Complete and timely removal of these tumors contributes to long-standing patient survival.

(NY State J Med 1992; 92:49–52)

Primary cardiac tumors remain an unusual pathologic entity, encountered in approximately 0.1% of autopsy cases, and in 1% of cardiac surgical practice.[1] The most common benign tumors are atrial myxomas, but a variety of other benign and malignant lesions may be encountered.[2] Two-dimensional echocardiography[3] and MRI[4–7] techniques have increased diagnostic accuracy and facilitated more precise preoperative planning. We review our experience with 14 cases of cardiac tumor over a 10-year period and emphasize the recent contributions of MRI.

Patients and Methods

Clinical Presentation. Fourteen patients with primary cardiac tumors were treated between July 1979 and June 1989. These patients made up 0.2% of the cardiac surgical practice at Westchester County Medical Center (Valhalla, NY). The four male patients averaged 37 years of age (range 18–48 yr); three had the malignancies, and one had a myxoma. Ten women (all with myxomas) had a median age of 59 years (range, 31–74 yr). Preoperative symptoms and modes of presentation were varied (Table I).

Among myxoma patients, four presented with embolic events: two cerebral, one cerebral and upper extremity, and one lower extremity. Three patients had episodic or vaguely described shortness of breath and dyspnea on exertion, with imitation of mitral valve disease. Other patients experienced syncope (one), palpitations (one), and new onset atrial fibrillation (two). Three patients with primary cardiac malignancies had localized chest pain. One of these also had dyspnea secondary to pericardial effusion causing cardiac tamponade.

Diagnostic Work-up. A combination of modalities was used for preoperative diagnosis of cardiac tumors. All patients but one had two-dimensional echocardiography, with correct diagnoses of cardiac tumors. Two patients with suspected malignancies had computed tomography examinations to delineate further the cardiac masses. The last five patients in the series underwent MRI studies, and the last two had both spin-echo and gradient echo signal acquisition imaging. Thirteen of the 14 patients had cardiac catheterization with assessment of ventricular function, valvular competence, coronary artery anatomy, and visualization of the already detected cardiac tumors.

Surgical Considerations. Indications for surgery in patients with myxomas included the presence of the mass, as well as obstructing, embolizing, or symptomatic tumor masses, presence of cardiac tamponade, the demonstration of dysfunctional mitral valves, and significantly diseased coronary arteries.

Timing of surgery was five days to eight months after clinical presentation with an average of 14 days; surgery generally followed the completion of work-ups. Four patients with embolic episodes underwent surgery between one week and eight months following the events. Concern about recurrence of emboli or life-threatening obstruction to blood flow prompted early operation. Deliberation about survival and quality of life after a cerebral embolus in a 74-year-old patient delayed surgery excessively in one instance.

Median sternotomy was the surgical technique in all patients. Two patients with malignant tumors underwent pericardial and extracardiac exploration only. The remaining 12 patients were placed on cardiopulmonary bypass, and 11 of these had aortic crossclamping and cardioplegic arrest for removal of their tumors. Of the 12 patients, one underwent exploration of the right ventricle and pulmonary artery with incomplete removal of a large tumor mass arising from the left pulmonary artery.

The 11 patients with intra-atrial masses had exploration through right, left, or bi-atriotomy incisions. The sites of origin and the extents of attachments of these tumors varied, influencing the plans of approach and the techniques of removal. Two patients with only single, small myxomas with discrete septal origins were treated by atriotomy, septal and tumor excision, and pericardial patch closure of the septum, one via the right atrium, the other through the left atrium. In all cases in which the exact attachment of the tumor bases could be determined, full-thickness removal of the tumor bases from the septum and from other aspects of the atrium was accomplished.

Three patients underwent mitral valve repair at the time of tumor removal. The one with rheumatic mitral stenosis was treated by commissurotomy. In two patients, mitral regurgitation caused by stretched chordae and myxomatous degeneration of the valve was repaired by measured, asymmetric annuloplasty. Two other patients had single coronary artery bypass grafting to the anterior descending vessel.

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TABLE I. Initial Events in 14 Patients with Primary Cardiac Tumors

Signs and Symptoms	Number
Benign tumors	
Episodic shortness of breath	3
Syncope, palpitations	2
Atrial fibrillation	2
Embolic event	4
Malignant Tumors	
Localized chest pain only	2
Chest pain and pericardial effusion with tamponade	1

RESULTS

The noninvasive diagnostic techniques correctly diagnosed cardiac tumors in all patients. Although echocardiographic techniques correctly demonstrated the cardiac tumors, MRI scanning more precisely defined the location, attachment and, to some extent, the character of the tumor mass its composition and extent of calcification. MRI identified a small area of tumor attachment to the atrial septum in two patients and demonstrated broad tumor bases in two other patients. The firm encapsulation and calcification of two large tumors also were demonstrated by MRI, findings that were not appreciated on either echocardiographic or catheterization studies. Cardiac catheterization revealed mild-to-moderate pulmonary hypertension in six patients. Five of these had large left atrial tumors, and the sixth had an obstructing malignancy in the pulmonary artery demonstrated by atrioventriculography. Three of the patients with pulmonary hypertension had mitral regurgitation or stenosis. Two other patients had significant coronary artery disease affecting a single vessel.

All myxomas originated in the left atrium, and all except one involved the atrial septum in the region of the fossa ovale. Five tumors were attached to both the atrial septum and additional sites in the left atrium.

The 11 tumors inside atrial chambers were characterized by pathologic study as myxomas (Table II). The gross and microscopic appearances were not uniform. Sizes varied from 2 × 2 to 6 × 8 cm.

Only three were well pedunculated with small mobile stalks. Eight were gelatinous in nature and four had firm, glistening, capsulated surfaces. Eight had large areas of thrombus or recent hemorrhage associated with myxomatous changes. Three were multilobulated, three exhibited inflammatory characteristics, and three had evidence of osseous changes. The specimens from the four patients with embolic presentations showed multilobulation with friable soft tumor in a fourth.

Three patients had primary cardiac malignancies (Table II). An 18-year-old man with an extensive mass around the right atrium and superior vena cava had an undifferentiated malignant spindle cell tumor. Rhabdomyosarcoma of the pleomorphic variety originated in the left pulmonary artery of a 38-year-old man. A 45-year-old man with pleural effusion and tamponade had poorly differentiated mesothelial tumor of the right ventricle (Fig 1).

TABLE II. Operative Approaches in 14 Patients with Primary Cardiac Tumors

Management	Number
Malignancies (3)	
Biopsy only	2
Incomplete removal	1
Myxomas (11)	
Left atriotomy only	1
Right atriotomy only	1
Bilateral atriotomy	9
Excision of atrial septum (10)	
Primary repair	1
Pericardial patch	9
Excision of atrial free wall and atrial septum (5)	
Primary repair of wall	4
Patch repair of wall	1

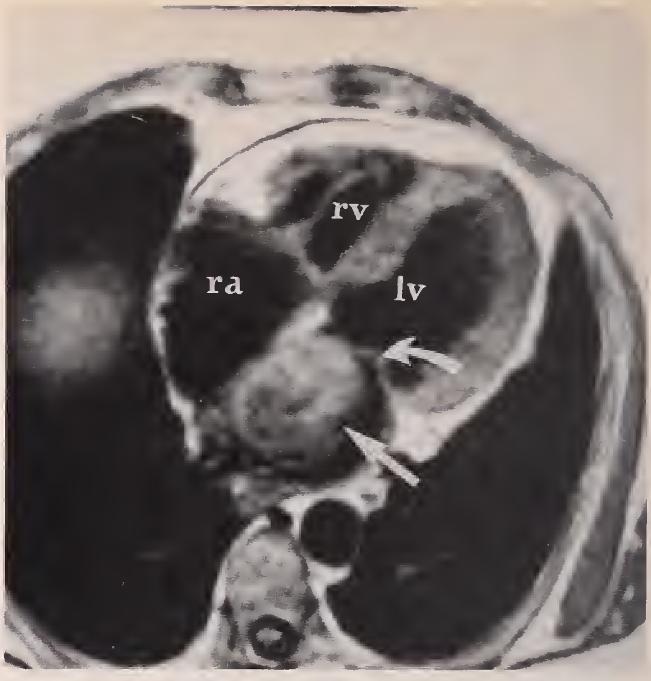

FIGURE 1. Left atrial myxoma. Axial spin-echo image shows well-encapsulated tumor mass within left atrium. Straight arrow, myxoma; curved arrow, mitral valve; lv, left ventricle; rv, right ventricle; ra, right atrium.

All patients survived surgery and left the hospital. In one patient, mitral regurgitation with congestive heart failure developed two days after removal of a large, friable myxoma. Reoperation eight days later demonstrated a tear of the anterior leaflet of the mitral valve and partial dehiscence of the septal patch. Mitral valve replacement and suture repair of the residual atrial defect were effective.

Follow-up. The 38-year-old patient with rhabdomyosarcoma died three months after surgery, and the 18-year-old patient with malignant spindle cell cancer died three years after surgery. The 45-year-old man with a poorly differentiated right ventricular tumor remains active 30 months after diagnosis, with no evidence of increasing tumor.

Of the 11 patients, eight with myxomas were alive and well 60 to 100 months after surgery (mean follow-up 49 months), six with no limitations, and two with residual impairment from their presenting embolic events. No patients with atrial myxoma have evidence of recurrence, but only two have undergone repeat noninvasive scans. One patient has metastatic colon cancer six years after surgery. Two died three years after surgery of noncardiac causes: one from cancer, type unknown, and one from head trauma.

DISCUSSION

The patients in this series are fairly representative of those reported.[8–10] All three malignant tumors were in men aged 18 to 45 years. The youngest male, with primary spindle cell sarcoma (previously reported[11]), illustrated the familial implications of cardiac tumors, both benign and malignant.[12] The long-term outlook was poor for patients with malignancies.

Women between the ages of 40 and 70 comprised most of the myxoma group. Atrial myxomas presented primarily as manifestations of episodic heart failure, obstruction to forward cardiac output, or as embolic events. It is quite common, however, for patients to present with vague and nonspecific symptoms; subsequently, routine echocardiography demonstrated the tumor mass.

Tumors were accurately diagnosed by two-dimensional echocardiography in this series of patients. Since many cardiac myxomas occur in middle-aged adults, cardiac catheterization to assess artery disease and valvular function should be a routine part of the preoperative workup.

Failure to diagnose an atrial tumor was not uncommon with m-mode echocardiography,[13] but diagnostic advances that evolved to present-day techniques accurately localize and characterize the tumor. Modern two-dimensional echocardiography provides excellent imaging detail and diagnostic accuracy.[3] It is probable that widespread use of two-dimensional echocardiography to evaluate the hearts of patients with nonspecific symptoms will result in the diagnosis of myxomas before the tumors enlarge and obstruct flow or embolize.

Cardiac MRI, with standard spin echo acquisition techniques, and, more recently, with gradient echo imaging, contributes important additional information. The anatomic relationship of the tumor mass to the normal intracardiac structures, and extension of tumor to extracardiac vascular and mediastinal structures can be appreciated more easily with MRI than with two-dimensional echocardiography because of the global view of the heart provided by MRI. MRI also provides excellent definition of tumor prolapse, secondary valvular changes, and cardiac chamber size.[14]

MRI imaging may allow better definition of tumor structure.[6,7] Encapsulation of the tumor may be demonstrated, and intratumor calcification may be represented by signal void seen on gradient echo images. In Figure 1, a well encapsulated tumor mass (myxoma) is defined on a standard spin-echo image, in a patient who presented with mitral valve obstructive symptoms. The specimen had a smooth firm capsule. Figure 2 demonstrates a myxoma with a more friable or lobulated appearance. Figure 3 shows dark areas on the gradient echo image, which are secondary to calcification within the tumor mass. The calcium was confirmed by gross and microscopic examination. The signal void is the result of magnetic susceptibility differences between tumor tissue and calcium.

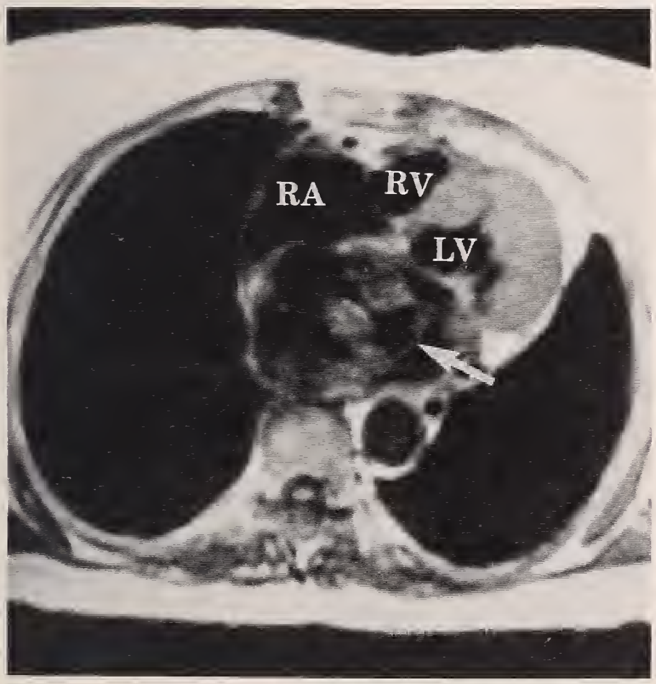

FIGURE 2. Left atrial myxoma. Axial spin-echo image demonstrates a lobulated mass. Arrow, myxoma; LV, left ventricle; RV, right ventricle; RA, right atrium.

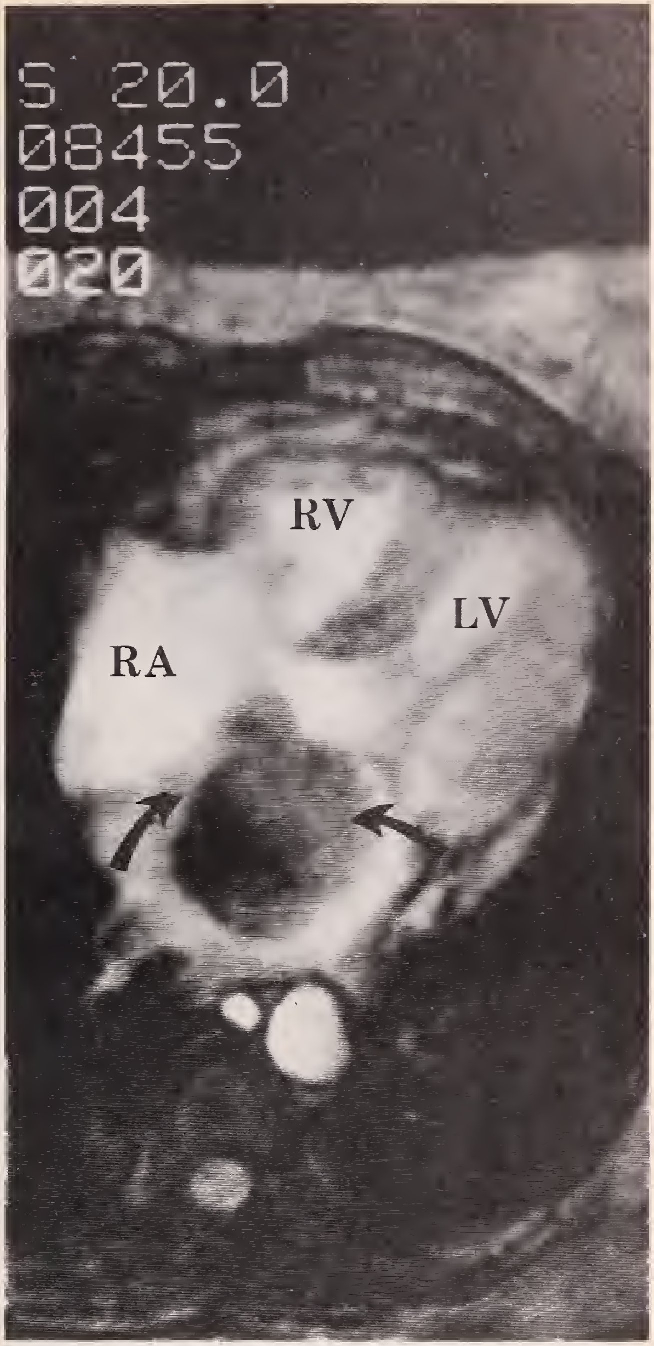

FIGURE 3. Calcified left atrial myxoma. Axial gradient-echo image shows a large area of signal void caused by susceptibility effects. Curved arrow, myxoma; LV, left ventricle; RV, right ventricle; RA, right atrium.

Figure 4 demonstrated one of the cardiac malignancies in the series. The axial spin echo image shows a large anterior mass with compression of the right ventricle and extension into the atrioventricular groove. The T_2 weighted saggital image shows the intrapericardial position of the mass. These images helped determine the incision (sternotomy versus thoracotomy) and the extent of resection in this 45-year-old man.

It should be emphasized that the mere presence of an atrial tumor, symptomatic or not, is an indication for surgery. With time, these tumors all grow and should be removed at the safest brief interval after diagnosis, before symptoms, progression of growth, or complications occur.

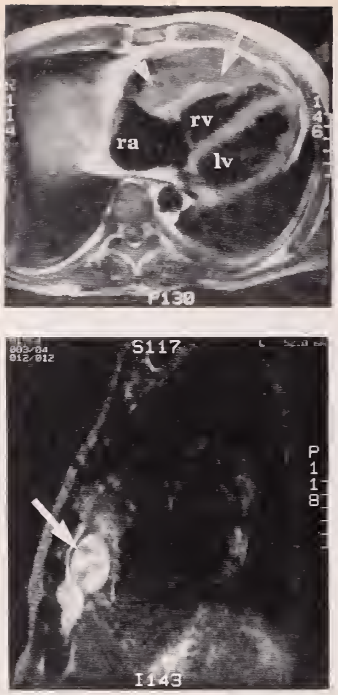

FIGURE 4. Malignant tumor. Upper, Axial image with mass (arrows) anterior to right ventricle. rv, right ventricle; lv, left ventricle; ra, right atrium. Bottom, The T_2 weighted saggital image shows the intrapericardial position of the mass (arrow).

Demonstration of all possible tumor characteristics facilitates timing, planning, and technical details of the surgery.

The long-term prognosis for patients whose myxomas were excised is excellent.[11] Our aggressive intraoperative approach included removal of all tissue with tumor attachment. Many tumors may be removed from the atrial septum; the base, if small, can be cauterized, or a small area removed. Patching the interatrial septum or areas of endocardium is not mandatory, given the rarity of recurrence. The few cases of recurrence reported[15] may have had multicentric origins which became manifest at a later time. The possibility of recurrence, though small, indicated the need for long-term follow-up with noninvasive cardiac imaging.

References

1. Fine G: Neoplasms of the pericardium and heart, in Gould SE (ed): *Pathology of the Heart and Blood Vessels*. Springfield, IL, Chas C Thomas, 1968, pp 851–883.
2. McAllister HA, Fenoglio JJ: Tumors of the heart and cardiovascular system. *Atlas of Tumor Pathology*. Fasc 15 Second Series, Washington DC, Armed Forces Institute of Pathology, 1975, pp 1–3.
3. Lappe DL, Bulkley BH, Weiss JL: Two dimensional echocardiographic diagnosis of left atrial myxoma. *Chest* 1978; 74:55–58.
4. Winkler M, Higgins CB: Suspected intracardiac masses: Evaluation with MR imaging. *Radiology* 1987; 165:117–22.
5. Higgins CB: Overview of MR of the heart-1986. *AJR* 1986; 146:907–18.
6. Lund JT, Ehman RL, Jukural PP, et al: Cardiac masses: Assessment by MR imaging. *AJR* 1989; 152:469–473.
7. Conces DJ Jr, Vix VA, Klatte EC: Gated MR imaging of left atrial myxomas. *Radiology* 1985; 156:445–447.
8. Dein JR, Frist WH, Stinson EB, et al: Primary cardiac neoplasms; early and late results of surgical treatment in 42 patients. *J Thorac Cardiovas Surg* 1987; 93:502–511.
9. Cho BK, Lee DY, Pezzella AT, et al: Surgical treatment of atrial myxomas, a report of 20 cases. *Tex Heart Inst J* 1989; 16:81–86.
10. Hanson EC, Gill CC, Razavi M, Loop FD: The surgical treatment of atrial myxomas. Clinical experience and late results in 33 patients. *J Thorac Cardiovasc Surg* 1985; 89:298–303.
11. Schreiber CS, Becker RH, King BO, et al: Primary cardiac tumor in an 18-year-old man: Manifestations of a familial cancer syndrome. *NY State J Med* 1986; 15:197–198.
12. Powers JC, Folkoff M, Heink RA, et al: Familial cardiac myxoma: Emphasis on unusual cardiac manifestations. *J Thorac Cardiovasc Surg* 1979; 77: 782–789.
13. Salcedo EE, Adams KV, Lever HM, et al: Echocardiographic findings in 25 patients with left atrial myxoma. *J Am Coll Cardiol* 1983; 1:1162–1163.
14. Go RT, O'Donnell JK, Underwood DA, et al: Comparison of gated MRI and 2D echocardiography of intracardiac neoplasms. *AJR* 1985; 145:21–25.
15. Gerbode F, Kerth WJ, Hill JD: Surgical management of tumors of the heart. *Surgery* 1967; 61:94–101.

Part II: Special considerations in the treatment of epilepsy

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Status epilepticus is generally defined as recurrent epileptic seizures without full recovery of consciousness between seizures, or continuous seizures lasting more than thirty minutes, whether or not consciousness is impaired. Status epilepticus can occur with most seizure types. Practically, it can be classified into three main types: generalized convulsive (grand mal) status, nonconvulsive (obscens and complex partials) status, and simple partial status (Table I).[1] Generalized convulsive status is a serious condition with an approximately 5%–50% mortality rate depending on the cause, with 1%–2% mortality rate related to a prolonged seizure per se.[2] Therefore, this situation must be treated quickly with an effective protocol. Persistence of status epilepticus (longer than 30–60 minutes) can result in the beginning of brain damage and can become increasingly difficult to control.[3] Treatment should aim at stopping the seisures as soon as possible. The protocol used at the University of Washington Regional Epilepsy Center is found in Table II and is comparable to that suggested by Delgado-Escueta.[4] After the seisures have been controlled, the etiology should be established and treated accordingly. Abrupt withdrawal of antiepileptic drugs (AEDs) is the most common cause of status epilepticus. Other causes include metabolic disorders (hyperosmolar nonketotic coma, hypoglycemia, hyponatremia, uremia); acute CNS insults (encephalitis, subarachnoid hemorrhage, cerebral infarction, cerebral trauma); and expanding lesions (tumor).

Nonconvulsive (obscens and complex partials) and simple partial status are less common and less threatening than the generalized convulsive status. Absence status can be recognized by a continuous dreamy or twilight state with electroencephalographic (EEG) concomitants of diffuse bilaterally synchronous 3-Hz spike and wave rhythms. Complex partial status shares the manifestations of unresponsiveness, automatism, and confusion, but has a more fluctuating course of repeated attacks.[5] It can be definitely differentiated from the absence status by the presence of temporal ictal EEG activity. Simple partial status presents as unrelenting partial seizures, with continuous focal epileptic discharges on EEG. Treatment of nonconvulsive and simple partial status consists of the administration of intravenous lorazepam or diazepam until the seizures are controlled and subsequent institution of standard AEDs.

PREGNANCY

Approximately 0.5% of all pregnant women have epilepsy.[6] In a recent prospective study of the effect of pregnancy on seizures, there was no change in seizure frequency in 50%, an increase in 37%, and a decrease in 13%.[7] Hormonal and metabolic changes are important factors, but the misperception by pregnant women of the risks that AEDs may have on the fetus can lead to medication noncompliance, which is in part responsible for increase of seizure frequency. Increased elimination of most AEDs (except carbamazepine) occurs during pregnancy[8] and serum levels tend to fall[9]; however, the unbound portion of the drug tends to rise and the overall active drug level may change very little.[10] Therefore, the doses of medication need not be altered unless the seizure frequency increases. It has been recommended that serum levels be followed monthly during pregnancy and then weekly in the postpartum period until drug kinetics return to prepregnancy norms.[11]

Seizures that occur in late pregnancy as a result of eclampsia have commonly been treated with intravenous magnesium sulfate. This treatment is controversial.[12,13] There is evidence from both animal[14] and clinical studies[15] that magnesium sulfate has little effect on central epileptogenic mechanisms. Most epileptologists feel that the use of appropriate standard AEDs is the treatment of choice for this problem.[13]

Although all AEDs have been associated with some increase in congenital anomalies, stopping medications during pregnancy is not recommended because of the risks that uncontrolled seizures may have on both mother and fetus.[16] In addition to those abnormalities, valproic acid is associated with a 1%–2% incidence of neural tube defects.[17,18] *Spina bifida can be diagnosed early in pregnancy by ultrasound and amniocentesis for alpha-fetal protein. Trimethadione is definitely contraindicated during pregnancy as it is associated with birth defects in a highly significant number of the exposed fetuses.[19,20] From several studies, the average incidence of congenital anomalies in fetuses exposed to AEDs is 4%–6%,[11,19,21] which is about twice the rate reported for unexposed fetuses.[21,22] No particular malformation is associated with any specific drug except for valproic acid. Five clinical syndromes have been

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Supported by NIH grant NS17111 and sponsored in part with a grant by New Medico Health Care System.

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*(Note added in proof.) An increase in occurence in spina bifida in fetuses exposed to carbamazepine has recently been noted.[57] This incidence is less than that observed with exposure to valproic acid, but still greater than that in the general population.

TABLE I. Classification of Status Epilepticus

Generalized Convulsive Status (primary or secondarily generalized)
  a) Tonic-clonic status (with or without partial onset)
  b) Myoclonic status
  c) Tonic status
Nonconvulsive Status
  a) Absence status
  b) Complex partial status
Simple Partial Status
  a) Partial motor status (Epilepsia Partialis Continua)
  b) Partial status with sensory, autonomic or affective symptoms

Modified from 1980 Santa Monica International Symposium Classification of Status Epilepticus.

described that are largely characterized by dysmorphic features, especially facial. These dysmorphic features are usually not aesthetically negative; however, the worrisome factor is that there may be an association with impaired intellectual development. In spite of the names given to the syndromes, the patients described are often not on monotherapy. The syndromes include the fetal trimethadione syndrome [23,24] and the fetal hydantoin syndrome,[25] the fetal valproate syndrome,[26] primidone embryopathy,[22] and the fetal carbamazepine syndrome.[28] Since essentially all the AEDs have been associated with some dysmorphic changes, epileptologists now speak of the "fetal AED syndrome." Higher plasma AED levels have been reported in some mothers with malformed infants,[29] and mothers on polytherapy have been reported to have higher rates of infant malformations.[30,31] Therefore, treating mothers during pregnancy with monotherapy at the lowest possible therapeutic level is desirable.

Virtually all AEDs cross the placentas so newborns will have serum levels of AEDs comparable with their mothers' levels.[11] It is the unbound portion of the drug that is found in breast milk. For the drugs that are less bound to protein such as phenobarbital and ethosuximide, more drug will be found in the breast milk. Some newborns exposed to phenobarbital in breast milk may be lethargic or jittery. In this circumstance, breast feeding should be discontinued and the newborn observed for signs of drug withdrawal.[32]

TABLE II. Steps in Treating Generalized Tonic-Clonic (Convulsive) Status Epilepticus in Adults

1. Maintain airway.
2. Place an intravenous line and draw blood for antiepileptic drug levels, glucose, blood urea nitrogen, electrolytes and complete blood count.
3. Administer 50 mL of 50% glucose intravenously.
4. Administer lorazepam, 1 mg/min intravenously (total dose: 4–16 mg).*
   OR
   Administer diazepam, 2 mg/min intravenously, up to 10 mg (unless seizures stop at a lower dose).
5. Administer phenytoin, 50 mg/min intravenously, up to 18 mg per kg.†
6. If seizures persist, administer phenobarbital, 100 mg/min intravenously, up to 10–15 mg/kg. (Respiratory depression is a danger with phenobarbital. This may be circumvented by using 2% paraldehyde instead of phenobarbital. Seizures usually abate with an injection of 0.2–0.4 ml/kg or 5 ml but may recur in a few minutes. A disadvantage of paraldehyde is that extravasation outside the vein is likely to produce local tissue injury. Pulmonary hemorrhage is another rare complication.)
7. If status epilepticus persists, administer general anesthesia under the supervision of an anesthesiologist.
8. When control has been achieved, institute maintenance doses of the preferred antiepileptic drug.

*Lorazepam (Ativan®) is longer acting than diazepam (Valium®) and produces less respiratory depression.

†Phenytoin (Dilantin®) is administered to provide seizure control after the shorter-acting benzodiazepams have been cleared.

Infants exposed to phenobarbital, primidone, phenytoin, carbamazepine, diazepam, methobarbital, amobarbital, and ethosuximide have an approximately 10% risk of having an hemorrhagic syndrome characterized by internal bleeding within the first 24 hours of life.[33] This phenomenon appears to be a result of a deficiency of vitamin-K-dependant clotting factors in the fetus. The Warfarin-like action of AEDs competitively inhibits vitamin K transport across the placenta. Treatment of the mother with oral vitamin K during the last month of gestation may prevent this problem.[34] The routine administration of vitamin K to the infant at birth is recommended but is not considered adequate therapy if any two of factors II, VII, IX, or X fall below 5% of normal levels.[35] Under those circumstances, transfusion of fresh frozen plasma is required.

Since enzyme inducing AEDs such as phenytoin, phenobarbital, carbamazepine, and primidone can increase hormone clearance, they can render contraceptives less effective. Therefore, contraceptive hormone doses may need to be increased in certain patients who are on these AEDs.[36]

PSEUDOSEIZURES

Pseudoseizures or nonepileptic seizures are another challenge to diagnosis and treatment. In many spells, the determination of organicity can be very difficult and prolonged scalp EEG monitoring with video is necessary.[37] If the typical "seizures" do not occur during the period of monitoring, the issue may not be resolved. Determining serum prolactin levels during the postictal period can sometimes be helpful as the level often, but not always, rises two to three times or more above baseline levels after generalized tonic-clonic and complex partial seizures, but not after pseudoseizures.[38] Serum prolactin rises immediately and further at 20 minutes after the generalized tonic-clonic and some complex partial seizures and returns to baseline after one hour.[38] To complicate the picture even more, some patients with pseudoseizures also have organic seizures. There is considerable controversy over the true incidence of these two disorders occurring in the same individuals.[39] When the diagnosis of pseudoseizures is made, it is important to present the diagnosis to the patient in a non-threatening way and help the patient in dealing with the experiences and stresses that have presumably precipitated the disorder.[40]

FEBRILE SEIZURES

Febrile seizures are considered reactive seizures to high body temperature. The children who have this seizure are by definition not epileptic. A predisposition to febrile seizures is inherited, and the incidence is 2%–5% of all children.[41,42] The first febrile seizure in life should be evaluated in a hospital and a lumbar puncture is indicated. However, subsequent febrile convulsions may not need aggressive investigation and may be safely managed at home. There are two aspects of therapy for febrile seizures: the acute treatment of the convulsion, and the prophylactic treatment to prevent recurrence. Since febrile seizures are usually brief, the acute treatment often requires no AEDs. However, diazepam (0.3 mg/kg, rectally) can be administered at home to children with seizures that tend to be prolonged or recur.[43,44] Prophylactic treatment of febrile seizures has been controversial, since approximately one third of the children with a first febrile seizure in life will ever have a second.[44] The rectal administration of diazepam has been shown to be effective in short-term prophylaxis of febrile seizures during febrile episodes; however, the risk of developing subsequent epilepsy is not reduced by this treatment.[45] Long-term prophylactic treatment with phenobarbital has recently been proven to be unsuccessful and significantly affects children's cognitive performance.[46] The use of primidone and valproate for prophylaxis of subsequent epilepsy has also declined due to the presence of adverse effects and the increasing use of intermittent rectal diazepam. Therefore, these AEDs are recommended only in children with high risk of developing subsequent epilepsy. These are patients with abnormal neurologic development, febrile seizures lasting longer than 15 minutes, multiple seizures in a 24-hour period, seizures with post-injury focal onset or followed by transient or persistent neurological abnormalities and a positive family history of epilepsy.[47]

Post-Traumatic and Postoperative Seizures

Seizures that occur after a head injury can be divided into early (within 7-days postinjury) and late onset (after one week). Early seizures are considered as acute brain reaction to the injuries, whereas late seizures are manifestations of post-traumatic epilepsy. The risk of developing post-traumatic epilepsy is increased if there are cortical contusions, intracranial hematomas, depressed skull fractures, penetrating head wounds, and an immediate seizure (within 24-hours post-injury).[48–50] If there is no seizure by three years, post-traumatic epilepsy is unlikely to develop.[50] For a long time, the prophylactic treatment of patients with head trauma with phenytoin has been a common practice and a subject of several clinical trials.[51,52,53] However, these early studies were not controlled. A recent prospective randomized double-blind study by Temkin et al has definitely shown that phenytoin (20 mg/kg, loaded within 24 hours of injury followed by a maintenance dose in high therapeutic range) can reduce the occurrence of early seizures but has no effect in preventing development of late epilepsy.[54] Therefore, phenytoin can be given for a week post-injury to prevent early seizures. After the first week, there is no indication for long-term treatment with phenytoin, unless late epilepsy has developed.

The prophylactic use of AEDs in patients undergoing craniotomy and surgical manipulation of the brain is fairly routine. One randomized double-blind study demonstrates the efficacy of this treatment.[55] This study recommended the use of prophylactic phenytoin seven to ten days before elective brain surgery and an intravenous loading dose of phenytoin for emergency surgery. Since epileptic seizures can complicate the post-operative course, most neurosurgeons attempt to provide their patients with therapeutic levels of an AED at the time of surgery. The known post-operative drop in AED levels can make this goal a challenge.[56] Phenytoin therapy should be maintained two to three months postoperatively, but up to 12 months in those patients with aneurysms, arteriovenous malformations, head injuries, or meningiomas.[55]

Acknowledgments. The authors thank Ms Lee Dulin for her patient secretarial assistance, especially with manuscript revisions, Ms Jill Andrews for obtaining reference articles in a timely fashion, and Jefferey J. Ojeman for helpful comments.

References

1. Gastaut H: Classification of status epilepticus. In: Delgado-Escueta AV, Wasterlain CO, Treiman DM, Porter RJ, (eds.) *Advances in Neurology, Vol 34: Status Epilepticus*, New York, Raven Press; 1983; 15–35 (Based on 1980 Santa Monica International Symposium on Status Epilepticus).
2. Hauser WA: Status epilepticus: epidemiologic considerations. *Neurology* 1990; 40(suppl 5, part 2):9–13.
3. Lothman E: The biochemical basis and pathophysiology of status epilepticus. *Neurology* 1990; 40(suppl 5, part 2):13–23.
4. Delgado-Escueta AV, Wasterlain C, Treiman DM, et al: Current concepts in neurology: Management of Status Epilepticus. *N Engl J Med* 1982; 306:1337–1340.
5. Treiman DM, Delgado-Escueta AV: Complex partial status epilepticus. In: Delgado-Escueta AV, Wasterlain CO, Treiman DM, Porter RJ, eds. *Advances in Neurology, Vol 34: Status Epilepticus*. New York, Raven Press; 1983; 69–81.
6. Svigos JM: Epilepsy and pregnancy. *Aust NZ J Obstet Gynaecol* 1984; 24: 182–185.
7. Schmidt D, Canger R, Avanzini G, et al: Change of seizure frequency in pregnant epileptic women. *J Neurol Neurosur Psychiatry* 1983; 46:751–755.
8. Yerby MS, Friel PN, Miller DQ: Carbamazepine protein binding and disposition in pregnancy. *Ther Drug Monit* 1985; 7:269–273.
9. Bardy AH, Teramo K, Hiilesmaa VK: Apparent plasma clearance of phenytoin, phenobarbitone, primidone, and carbamazepine during pregnancy: results of the prospective Helsinki study. In: Janz D, Bossi L, Dam M, Helge H, Richens A, Schmidt D (eds.) *Epilepsy, Pregnancy, and the Child*. New York, Raven Press; 1982; 141–145.
10. Chen SS, Perucca E, Lee JN, et al: Serum protein binding and free concentrations of phenytoin and phenobarbitone in pregnancy. *Br J Clin Pharmacol* 1982; 13:547–552.
11. Philbert A, Dam M: The epileptic mother and her child. *Epilepsia* 1982; 23:85–99.
12. Dinsdale HB: Does magnesium sulfate treat eclamptic seizures? Yes. *Arch Neurol* 1988; 45:1360–1361.
13. Kaplan PW, Lesser RP, Fisher RS, et al: No, magnesium sulfate should not be used in treating eclamptic seizures. *Arch Neurol* 1988; 45:1361–1364.
14. Koontz WL, Reid KH: Effect of parenteral magnesium sulfate on penicillin-induced seizure foci in anesthetized cats. *Am J Obstet Gynecol* 1985; 153:96–99.
15. Sibai BM, Spinnato JA, Watson DL, et al: Effect of magnesium sulfate on electroencephalographic findings in preeclampsia-eclampsia. *Obstet Gynecol* 1984; 64:261–266.
16. Nelson KB, Ellenberg JH: Maternal seizure disorder, outcome of pregnancy, and neurological abnormalities in children. *Neurology* 1982; 32:1247–1254.
17. Robert E, Guibaud P: Maternal valproic acid and congenital neural tube defects. *Lancet* 1982; 2:937.
18. Bjerkedal T, Czeizel A, Goujard J, et al: Valproic acid and spina bifida. *Lancet* 1982; 2:1096.
19. Nakane Y, Okuma T, Takahashi R, et al: Multi-institutional study on the teratogenicity and fetal toxicity of antiepileptic drugs: a report of a collaborative study group in Japan. *Epilepsia* 1980; 21:663–680.
20. German J, Kowal A, Ehlers KH: Trimethadione and human teratogenesis. *Teratology* 1970; 3:349–362.
21. Kelly TE: Teratogenicity of anticonvulsant drugs. I: Review of the literature. *Am J Med Genet* 1984; 19:413–434.
22. Kalter H, Warkany J: Congenital Malformations (second of two parts). *N Engl J Med* 1983; 308:491–497.
23. Zackai EH, Mellman WJ, Neiderer B, et al: The fetal trimethadione syndrome. *J Pediatr* 1975; 87:280–284.
24. Feldman GL, Weaver DD, Lovrien EW: The fetal trimethadione syndrome: Report of an additional family and further delineation of this syndrome. *Am J Dis Child* 1977; 131:1389–1392.
25. Hanson JW, Smith DW: The fetal hydantoin syndrome. *J Pediatr* 1975; 87:285–290.
26. DiLiberti JH, Farndon PA, Dennis NR, et al: The fetal valproate syndrome. *Am J Med Genet* 1984; 19:473–481.
27. Rudd NL, Freedom RM: A possible primidone embryopathy. *J Pediatr* 1979; 94:835–837.
28. Jones KL, Lacro RV, Johnson KA, et al: Pattern of malformations in the children of women treated with carbamazepine during pregnancy. *N Engl J Med* 1989; 320:1661–1666.
29. Dansky L, Andermann E, Sherwin AL, et al: Maternal epilepsy and birth defects: a prospective study with monitoring of plasma anticonvulsant levels during pregnancy. In: Dam M, Gram L, Penry JK, (eds.) *Advances in Epileptology: XIIth Epilepsy International Symposium*. New York, Raven Press; 1981; 607–612.
30. Nakane Y: Congenital malformations among infants of epileptic mothers treated during pregnancy—The report of a collaborative study group in Japan. *Folia Psyhchiatr Neurol Jpn* 1979; 33:363—369.
31. Lindhout D, Hoppener RJ, Meinardi H: Teratogenicity of antiepileptic drug combinations with special emphasis on epoxidation (of carbamazepine). *Epilepsia* 1984; 25:77–83.
32. Commission on Genetics, Pregnancy, and the Child, International League Against Epilepsy: Guidelines for the care of epileptic women of childbearing age. *Epilepsia* 1989; 30:409–410.
33. Mountain KR, Hirsh J, Gallus AS: Neonatal coagulation defect due to anticonvulsant drug treatment in pregnancy. *Lancet* 1970; 1:265–268.
34. Deblay MF, Vert P, Andre M, et al: Transplacental vitamin K prevents haemorrhagic disease of infant of epileptic mother. *Lancet* 1982; 1:1247.

35. Srinivasan G, Seeler RA, Tiruvury A, et al: Maternal anticonvulsant therapy and hemorrhagic disease of the newborn. *Obstet Gynecol* 1982; 59:250–252.

36. Mattson RH, Cramer JA, Darney PD, et al: Use of oral contraceptives by women with epilepsy. *JAMA* 1986; 256:238–240.

37. Luther JS, McNamara JO, Carwile S, et al: Pseudoepileptic seizures: Methods and video analysis to aid diagnosis. *Ann Neurol* 1982; 12:458–462.

38. Dana-Haeri J, Trimble MR, Oxley J: Prolactin and gonadotropin changes following generalised and partial seizures. *J Neurol Neurosurg Psychiatry* 1983; 46:331–335.

39. Engel J Jr: Differential diagnosis. In: Engel J Jr (eds.) *Seizures and Epilepsy*. Philadelphia, FA Davis; 1989; 340–379.

40. Shen W, Bowman ES, Markand ON: Presenting the diagnosis of pseudoseizure. *Neurology* 1990; 40:756–759.

41. Annegers JF, Hauser WA, Elveback LR, Kurland LT: The risk of epilepsy following febrile convulsions. *Neurology* 1979; 29:297–303.

42. Hauser WA: The natural history of febrile seizures. In: Nelson KB, Ellenberg JH, (eds.) *Febrile Seizures*. New York, Raven Press; 1981; 5–17.

43. Lombroso CT: Intermittent home treatment of status and clusters of seizures. *Epilepsia* 1989; 30(suppl 2):S11–S14.

44. Camfield CS, Camfield PR, Smith E, et al: Home use of rectal diazepam to prevent status epilepticus in children with convulsive disorders. *J Child Neurol* 4: 125–126.

45. Knudsen FU: Effective short-term diazepam prophylaxis in febrile convulsions. *J Pediatr* 1985; 106:487–490.

46. Farwell JR, Lee YJ, Hirtz DG, et al: Phenobarbital for febrile seizures: effects on intelligence and on seizure recurrence. *N Eng J Med* 1990; 322:364–369.

47. National Institute of Health Consensus Development Conference Summary: Consensus statement on febrile seizures. In: Nelson KB, Ellenberg JH, (eds.) *Febrile Seizures*. New York, Raven Press; 1987; 301–306.

48. Annegers JF, Grabow JD, Groover RV, et al: Seizures after head trauma: a population study. *Neurology* 1980; 30:683–689.

49. Weiss GH, Feeney DM, Caveness WF, et al: Prognostic factors for the occurrence of posttraumatic epilepsy. *Arch Neurol* 1983; 40:7–1.

50. Weiss GH, Salazar AM, Vance SC, et al: Predicting posttraumatic epilepsy in penetrating head injury. *Arch Neurol* 1986; 43:771–773.

51. Servit Z, Musil F: Prophylactic treatment of post-traumatic epilepsy: Results of a long-term follow-up in Czechoslovakia. *Epilepsia* 1981; 22:315–320.

52. Young B, Rapp RP, Norton JA, et al: Failure of prophylactically administered phenytoin to prevent early posttraumatic seizures. *J Neurosurg* 1983; 58:231–235.

53. Young B, Rapp RP, Norton JA, et al: Failure of prophylactically administered phenytoin to prevent late post-traumatic seizures. *J Neurosurg* 1983; 58:236–241.

54. Temkin NR, Dikmen SS, Wilensky AJ, et al: A randomized, double-blind study of phenytoin for the prevention of post-traumatic seizures. *N Engl J Med* 1990; 323:497–502.

55. North JB, Penhall RK, Hanieh A, et al: Phenytoin and postoperative epilepsy, a double-blind study. *J Neurosurg* 1983; 58:672–677.

56. Friel P, Clarke H, Ojemann G, et al: Decreased serum anticonvulsant levels after epilepsy surgery. *Epilepsia* 1987; 28:588.

57. Rosa F, Hughes T, et al: Spina bifida in infants of women taking carbamazepine. N Engl J Med 1991; 325:664–667.

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XII. DISCUSSION ON EPILEPSY
4. Mental Epilepsy.

Maniacal disturbance preceding or following convulsions is a comparatively common incident, especially frequent in asylum practice; cases so affected depict the transition from the purely motor levels to the higher volitional centres presiding over organized motion. Rarer are the instances from which convulsion is absent, the explosion limiting itself to the region of voluntary movement. If associated with transient facial pallor, or flushing, momentary fixation of the eyes or limbs, or vertigo, indicating the preliminary attack of *petit mal*, the diagnosis is not difficult.

Whether the initial *petit mal* be theoretically present or not, it is often impossible of detection, and the attacks, characterized solely by ill-directed and causeless fury, simulate genuine recurrent or transitory insanity, for the frequent abuse of which terms epilepsy affords ample excuse.

> M. G., a woman, aged nineteen years, was admitted to the St. Lawrence State Hospital, September 22, 1891. Her sister was an inmate of the institution; no other family history has been ascertained. The medical certificate of insanity stated that the physicians were "unable to have any conversation with her. She met them at the door with a bottle and brick in her hands. They were unable to talk with her, as she threw bricks whenever she saw any one." Newspaper reports of her actions prior to admission gave accounts of outbreaks of sudden and excessive violence, with intermissions in which she "astonished the police by talking rationally." It was also stated that she had assaulted officers, and had bitten them, and had carried and freely used various weapons. Her insanity was said to have existed for two weeks. She was sullen and irritable on admission, and a few moments later began maledictions against policemen and policemen's clubs, and made a sudden and destructive assault upon persons and furniture. After a few hours of continued excitement she became comatose, and awoke quiet, rational, orderly, and well-disposed. Numerous similar attacks have since occurred, and have occasionally been controlled by bromide, ergot, and chloral. She has frequently complained of toothache, sciatic neuralgia, and headache, which have always borne time relation to the maniacal outbreaks. During the attacks her pupils are dilated, expression vacant, and face pale. Coma has been an invariable sequence, and convulsion has never been ascertained. In the intervals between the attacks she is affable, cheerful, courteous, and industrious. She is aware, because of post-paroxysmal discomforts, that she has had an attack, but has never known of its inception or events. She has requested that she be removed from others during the seizures in order that she may inflict no injury, which she afterward always regrets.

Here were genuine epileptic attacks, in which spasm was replaced by co-ordinated, involuntary, motor fury, with complete and instantaneous lapse of memory and without especial dependence upon an excitation in the environment. In the following case the ascent toward cerebral areas of higher than motor function is shown in an "intellectual aura," with ensuing mania, occurring simultaneously with obliteration of memory.

J.M. MOSHER, MD

(*Transactions of the Medical Society of the State of New York for the Year 1893.*
NY, Medical Society of the State of New York; 1893, pp. 169–179)

Island Peer Review Organization educational posters

RAPHAEL P. NENNER, MD

The Education Committee of the Island Peer Review Organization (IPRO), with the cooperation of IPRO's Medical Directors, has developed several educational posters.

IPRO has widely distributed three educational posters covering topics such as "Ten Steps to Effective Medical Record Documentation," "Consultation Guidelines," and "Discharge Planning Guidelines." The topics were selected by IPRO's Office of the Vice President for Medical Affairs because problems in these areas can easily be averted by having access to the correct information. To prevent problems before they start, IPRO has initiated a series of posters to alert physicians and hospitals to specific guidelines in these areas.

The mission of IPRO's poster series is not to disseminate the latest medical news. The posters are guidelines designed to educate and reach out to the physician community. The posters ARE NOT criteria. Rather, they contain information based on problems identified by IPRO's Medical Directors and Physician Consultants. When the posters are strategically placed in a health-care setting, they can serve as simple, everyday reminders of "the basics." We intuitively believe that the quality of care would be measurably improved if more physicians considered the points raised in these guidelines.

POSTER I. Ten Steps to Effective Medical Record Documentation

The following guidelines are designed to assist the physician in preparing a record which accurately and adequately documents the care rendered to a patient in the hospital:

1. A complete history and physical exam including baseline lab values, pap smear, breast examinations and rectal examination are required. Provisional diagnosis must be documented.
2. Daily progress notes must reflect findings, assessment and plan of care. Avoid use of such phrases as "status quo." Progress notes should reflect the *acute* condition of the patient.
3. Physician orders must reflect treatment of the condition for which the patient was admitted or which develops subsequently. If ancillary tests or medical therapies are ordered, which are not consistent with the current diagnosis or condition, they should be justified in the progress notes.
4. Note all abnormal test findings in your progress notes, along with an assessment of the findings' impact on the patient's current condition. A plan for treatment or follow-up must be included.
5. If antibiotics ordered do not conform with sensitivity results, document the reason for your choice.
6. If the patient must undergo unplanned surgery, document indications clearly.
7. Nosocomial infections, transfusion reactions or errors, medication errors, or trauma suffered in the hospital should be completely assessed in the progress notes.
8. Document early efforts to arrange an adequate discharge plan for the patient.
9. Your final note should reflect the medical stability of the patient on discharge. Blood pressure and temperature within normal limits, wound clean and dry, and any abnormal ancillary findings should be addressed with a plan for follow-up post-discharge.
10. Your final summary should be a meaningful recapitulation of the patient's course of illness, hospital management, discharge plan/instructions and include a plan for follow-up care. At discharge, final diagnoses which relate to the current hospitalization should be included.

POSTER II. Consultation Guidelines

A. Except in an emergency, medical consultation is strongly suggested when:
   1. A diagnosis is obscure.
   2. Doubt exists as to the best therapeutic measure(s) to use.
   3. Surgery is planned (excludes emergency surgery) on:
      a. Any patient above age 60 (or any other age as determined by the hospital) who is to receive general, regional, spinal or caudal anesthesia.
      b. Any patient with serious or potentially serious pre-existing medical conditions, regardless of age.
      c. Any patient who is not a good risk for surgery or anesthesia.

B. A satisfactory consultation should include:
   1. A history and review of the patient's medical record.
   2. A physical examination of the patient.
   3. Written opinion signed by the consultant and dated with time.
   4. The consultant's recommendations.

Address correspondence to Dr Nenner, Vice President for Medical Affairs, Island Peer Review Organization, 1979 Marcus Avenue, Lake Success, NY 11042.

POSTER II. *(Continued.)*

C. Consultations are generally to be performed within 24 hours of request; common courtesy suggests adequate notice to consultants, especially when a patient is to be admitted late in the day or on a weekend for scheduled surgery.
D. Pre-operative consults must be written *prior* to procedure.
E. Consultation may be performed only by a physician with privileges to give consultation in the specialty area in which it is sought.
F. The request should include:
   1. Consultation only, or
   2. Consultation and follow-up
G. The consultant is considered to have an *advisory* role; his recommendations may or may not be accepted by the physician of record.
H. The patient's physician of record is the *captain of the ship* and is the physician directing the care; reasons for not following the consultant's recommendations should be carefully documented in the chart.

POSTER III. Discharge Planning Guidelines

1. Hospitals are required to have a coordinated discharge planning program to ensure continuity of care and the most effective utilization of hospital and community-based health and social services.
2. Hospitals are required to have written criteria for an admission high-risk screening system to identify promptly patients who may need post-hospital care and planning.
3. All health care professionals play a role in assessing patients' post-hospital needs and developing individualized, comprehensive, discharge plans consistent with physician orders and patient needs.
4. Physicians have a key role in determining medical readiness for discharge and communicating this information to patients, families, and other staff.
5. Discharge planning shall include patient and family participation in the decision-making process regarding post-hospital care and the selection of specific services.
6. Patients and family representatives shall be provided with information regarding the range of community services available.
7. Post-hospital care planning shall include consideration of the following types of services:
   A. Home care
      a. Certified home health agencies
      b. Long-term home health programs
      c. Home attendance services
      d. Meals-on-wheels programs
      e. Electronic home emergency response systems
   B. Residential health care facilities
      a. Short-term rehabilitation facilities
      b. Long-term care facilities
      c. Specialized facilities for head injury, terminal care, and so forth
   C. Hospice
   D. Day care
   E. Respite care
   F. Community health, mental health, and social agencies
   G. Linkage to entitlements such as Medicaid, food stamps, prescription subsidies, Social Security Disability, public assistance.
8. Hospitals must provide patients with a written discharge notice and a written discharge plan.
9. Patients seen in the emergency room but not admitted shall also be provided with discharge planning services.

These guidelines are based on New York State Department of Health regulations under Article 28, part 4059.

Illicit drug use should not be legalized

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Should heroin and crack use be decriminalized and/or legalized in the United States? In 1929, the Wickersham Commission was appointed by President Herbert Hoover to evaluate the state of law enforcement and especially Prohibition in the United States. The Commission's report helped shape the national debate that preceded the repeal of Prohibition. Nadelmann suggests that a similar commission, composed of North and South Americans, could evaluate the costs and benefits, as well as the potential and limits of the international drug (heroin and cocaine) control approach. The drug problem is an international problem and requires international solutions.[1]

PROPONENTS OF LEGALIZATION

Benoit and Coletti assert that everyone knows that America is losing the war on drugs. What nobody wants to face is the logical alternative: the legalization of drugs. This alternative could be cost effective and could work.[2] In December 1990, Federal Judge Robert Sweet delivered a speech in which he argued that America should legalize drugs. Sweet wants to remove the high profitability of drugs. He would have the government dispense drugs as it does alcohol, thereby driving down black market prices. Money saved on law enforcement would be used to expand treatment programs.[3] Drug czar William Bennett labelled Sweet's position as "morally scandalous."[3]

Nadelmann argues that drug legalization increasingly merits serious consideration as both an analytical model and a policy option. Criminal justice approaches to the drug problem have proved limited in their capacity to curtail drug abuse. They also have proved increasingly costly and counterproductive. He concludes that appropriate drug legalization policies could minimize the risks of legalization, dramatically reduce the costs of current policies, and directly address the problems of drug abuse.[4]

Bayer posits that the prohibitionist social policy applied to the issue of narcotics use has been a failure for over 60 years. Furthermore, the consequences of heroin use have been and continue to be disastrous. For the entire population, the costs of law enforcement, public corruption, and poverty crime are increasingly burdensome. It is in this context, states Bayer, that the increasing calls for decriminalization must be understood.[5] He supports the "pharmacy" model of drug distribution, which involves the prescription of maintenance dosages of heroin by physicians to those already addicted.

A proposal to legalize drugs was presented by Baltimore Mayor Kurt Schmoke and others in September 1988 at hearings held before the US Congress' Select Committee on Narcotics Abuse and Control. Chairman Charles Rangel at first refused to include such proposals saying "I strongly object to even the mention of legalization as an option".[6] Grinspoon and Bakalar from Harvard Medical School point out that we need a policy that recognizes the complexity of drug use as a social phenomenon. They propose that currently illicit drugs be legalized and taxed. The taxes would be used for drug education and research, and for paying the medical and social costs of drug abuse. Under this social arrangement:

> We would no longer suffer the corruption and terror of the drug war. Instead of paying for a bloated enforcement bureaucracy and providing vast profits to criminals, society would extract the costs of drug use from the users in proportion to the amount they contribute to the problem . . .[7]

OPPONENTS OF LEGALIZATION

In 1972, President Nixon appointed James Q. Wilson as chairman of the National Advisory Council for Drug Abuse Prevention. The Commission was charged with providing guidance on how best to coordinate the national war on drugs. That same year, economist Milton Friedman published an essay in *Newsweek* in which he called for legalizing heroin. His argument was twofold: As a matter of ethics, the government has no right to tell people not to use heroin and, as a matter of economics, the prohibition of drug use imposes costs on society that far exceed the benefits. The commission did not take Friedman's advice.

Wilson points out that we now have the same number of heroin addicts that we had in 1972—half a million. This population is relatively stable, composed mainly of an aging pool of heroin addicts plus a relatively small number of new recruits.[8] Many factors appear to explain why heroin lost its appeal to young users, including high prices, reduced supply, and negative health effects such as hepatitis and death. Proponents of legalizing the much more dangerous

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Prepared as an educational and informational report by the Committee on Bioethical Issues of the Medical Society of the State of New York.

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drug cocaine, especially in its potent form, crack, should reflect on the heroin experience. According to Wilson, had the commission recommended legalizing heroin in 1972, "We almost certainly would have had today a permanent population of several million, not several hundred thousand, heroin addicts. If we yield now we will have a far more serious problem with cocaine."[8]

Wilson scoffs at those who say that we have lost the war on drugs. This is exactly what he was told in 1972. The war has not been won, nor has it been lost.

What the nation accomplished then was what most efforts to save people from themselves accomplish: The problem was contained, and the number of victims minimized, all at a considerable cost in law enforcement and increased crime. Was the cost worth it? I think so, but others may disagree . . .[8]

Schuckit agrees with Wilson. He opposes legalization of cocaine primarily because he believes that increased availability will cause increased use, which will lead to large costs for the treatment of physical and psychiatric difficulties.[9]

Other arguments opposing legalization are cited below in the statement of the Committee on Bioethical Issues of the Medical Society of the State of New York.

The Dutch and British Experiences

The Dutch have adopted a compromise between legalization and an intensive war on drugs. The Dutch view the drug abuse problem as a health matter, not as a problem for the police or the justice system. Such an approach fits into Dutch culture and society, which is realistic and practical in its approach to problems, and avoids the overly moralistic or dramatic tone more typical of American reactions. In this common-sense Dutch approach, many kinds of street drugs are available on a quasi-legal basis at cafés in designated sections of towns. This availability attracts addicts from other countries, but the Netherlands has not been swept up in a drug-use epidemic. Engelsman points out that:

> The Dutch acknowledge that the international repressive, prohibitive approach leads to unintentional negative side-effects, both for the individual and for society. They try to avoid a situation in which consumers of cannabis suffer more damage from the criminal proceedings than from the use of the drug itself. They opt rather for a realistic and practical approach to the drug problem rather than for a moralistic and over-dramatized one. The Dutch alternative is the normalization of the drug problem, as a pragmatic compromise between two extreme options: an intensified war on drugs and legalization. The implications of the normalization approach for the prevention and treatment policies (include): AIDS—prevention, harm reduction, instead of detoxification and demystification. It is suggested that the policy of normalization is rather successful and does not produce an increase of drug use.[10]

In Great Britain, until mid-1960s, physicians were allowed to prescribe heroin to certain classes of addicts such as pain-ridden patients who became dependent on opiates. In the late 1960s, new street users of heroin considerably increased the number of addicts. By 1982, London alone was believed to have had 20,000 heroin users.[8] Britain is now wrestling with its own drug-use epidemic.

Physicians' Views Toward Drug Legalization

Linn, Yager, and Leake asked 303 practicing physicians to indicate whether possessing or using marijuana should be considered a felony, a misdemeanor, warrant the issuance of a citation, or be legalized. They found that:

> The position physicians advocated was unrelated to their specialty, experience diagnosing or treating substance abuse problems, their attitudes toward the efficacy of the treatment of drug abuse, or any other work role or habit we measured. Legalization or citation as compared with harsher penalties, however, was more likely favored by physicians who were younger, less religious, politically more liberal, and those less likely to perceive a serious drug problem in society. Legalization was also more likely favored by physicians who themselves had used marijuana, cocaine, and amphetamines but was unrelated to the use of alcohol, cigarettes, or tranquilizers.[11]

These authors conclude that, although physicians' opinion should be sought as society deals with the drug problem, their views are shaped largely by the same factors that shape the view of others.

Most physicians probably oppose legalizing drugs. The American Medical Association's House of Delegates issued a 1988 report on substance abuse that included a statement opposing the legalization of drugs. In 1989, the Medical Society of the State of New York's Council opposed the call for the legalization of drugs used by drug abusers. The Council noted that such an experiment was tried in the past in the United States and that it was a failure.

A retired professor of surgery at Harvard, Vernon Mark, said, "I think it would be catastrophic to legalize drugs."[12] Yale professor of psychiatry Herbert Kleber said, "Crime would not decrease with legalization. It would increase."[12] Other physicians, such as Harvard's Grinspoon, favor legalization.[7]

Philosophical Arguments Regarding Drug Legalization

The debate about whether or not illicit drug use should be legalized does not concern the possibility that such drug use has positive value. Considerable evidence proves that illicit drug use is psychologically, medically, and socially harmful. Even most advocates of legalization agree that drug use is grossly immoral. The debate does not concern overuse of prescriptive drugs by people indulging in self-treatment of real or perceived medical problems. Furthermore, the legalization debate is only about legalization for adults, not for children. Finally, legalization would not lift restrictions on drug use by those in key occupations, such as drivers and heavy machinery operators.

Philosophical arguments in favor of legalization include the "straight utility" thesis that legalization would decrease the number of addicts by removing the aura of illegality and by removing the incentives for pushers to induce young people to use drugs. The "quasi-utilitarian" argument is that although legalization might increase the number of addicts, the long-term benefits of decreased crime, and decreased spread of hepatitis, acquired immunodeficiency syndrome (AIDS), and other sexually transmitted diseases would outweigh the negative aspects of increased drug use. A third argument is that present drug laws have inappropriately increased the size and power of government so that it now infringes on personal liberties and freedoms. This "natural rights" view, derived from Aquinas, Locke, Jefferson, Nozick and others, maintains that people have inalienable rights to live their lives as they wish as long as they do not harm others. Furthermore, innocent people are affected by street crimes perpetrated by drug addicts, and it is argued, such crime might well be decreased by legalization.

Philosophical arguments against legalization include the "straight utilitarian" thesis that non-therapeutic drug use, if legalized, will markedly increase. This thesis is a very strong argument and is much more significant, if true, than the succeeding arguments. Another argument against legalization is that government should be the protector of individuals from themselves.[13] Another philosophical concern relates to the association of drug use with a "counter culture" that espouses group living and communal relationships. Western tradition is profoundly "individualist." Laws relating to marriage are accepted but group or communal marriage is illegal. It is argued that the state should promote basic Western values, and therefore should oppose illicit drug use and its legalization.

COMMITTEE ON BIOETHICAL ISSUES OF THE MEDICAL SOCIETY OF THE STATE OF NEW YORK

The Committee on Bioethical Issues of the Medical Society of the State of New York devoted two meetings to a discussion of the pros and cons of the legalization of illicit drugs. Two experts on the topic presented their views. Robert Newman, MD, President of the Beth Israel Medical Center in New York and former Assistant Commissioner for Addiction Programs of the New York City Department of Health and director of New York City's Narcotic Register, said that the debate over legalization of illicit drugs is a diversion from what should be done, can be done, but is not being done, namely making effective treatment available for those who want it. He advocated massive expansion of the treatment network for those who ask for help, and examination of alternatives for the remaining hard core addicts. For pragmatic, practical, social and political reasons, he concluded that legalization cannot be accomplished and that the priorities of government and law enforcement agencies dealing with this problem should be reviewed.

John Bartels, Esq, former head of the US Drug Enforcement Administration, stated that he is strongly opposed to legalization of illicit drugs and rejects the argument that law enforcement and the criminal justice system have been ineffective in dealing with the problem. He believes that legalization would not only result in greater availability and increased use but would increase first-time use at an early age and thus exacerbate the problem.

Two members of the committee countered the above presentations and offered arguments to support the position that illicit drug use should be legalized. Thus, both sides of the issue were equally considered, giving a fair hearing to both points of view.

The members of the Committee on Bioethical Issues discussed the many aspects of the problem. By a vote of 12 to 2, the committee opposed legalization of illicit drugs. It adopted the following resolution, which supports the official position of the Medical Society of the State of New York.

STATEMENT REGARDING LEGALIZATION OF THE USE OF NON-PRESCRIPTIVE, POTENTIALLY DANGEROUS DRUGS

Physicians should oppose the legalization of the use of non-prescriptive, potentially dangerous drugs such as heroin and cocaine. Use of such drugs poses a serious threat to the health of the individual and society. In countries where such drugs have been legalized, their use has increased. Use of potentially dangerous drugs frequently leads to limited reasoning ability, unproductive and antisocial behavior, an increase in the development of neurologic, malignant, psychiatric, infectious and other medical diseases, and fetal health problems. Maternal narcotism causes damage to the embryo or fetus, which not only increases fetal mortality, but also increases fetal morbidity and mortality later in life. These health considerations outweigh any potential reduction in crime or reduction in the transmission of infection that might be anticipated from the legalization of such drugs.

CONCLUSION

The debate about whether or not to legalize illicit drug usage has generated much heat but little light. Most knowledgeable people agree that drug addicts should be treated as patients rather than criminals, although their criminal behavior should not be tolerated; that is, addicts who engage in criminal conduct should be held accountable by the criminal justice system. The legalization of drugs may be a solution worse than the problem itself.

A massive educational effort is needed in the United States about the terrible consequences of drug use and the lives it ruins. "Just say no" is not enough. To tell a drug addict "just say no" is like saying "have a good day" to a patient with depression. Treatment facilities for those who need and want them are often not available and must be provided. Preventive education should be focused on the young, especially teen-agers. Prevention is better than treatment. Drug addiction is treatable and often curable with multifaceted support services. Drug addicts are a heterogeneous group and may require different and various approaches.

Society does not like addicts. They commit crimes, they acquire AIDS and other diseases at an alarming rate, and they use up enormous social resources. They do not vote and no powerful constituency speaks for them. On the other hand, strong, albeit criminal interests, support the production, distribution and sale of drugs such as heroin and cocaine. Drug addicts are people. As physicians, we recognize them as patients with many medical and psychosocial needs. The first step is to provide treatment programs for all who request them. The high rate of recidivism must also be addressed and solutions proposed. Finally, imaginative ideas are needed to address the problem of the remaining hard core drug addicts who seek no help but desperately need it.

The Committee on Bioethical Issues of the Medical Society of the State of New York opposes the legalization of illicit drug use. As the first part of a comprehensive approach to this enormous public health problem, the Committee strongly urges society to provide public education and treatment for all drug addicts who request it.

REFERENCES

1. Nadelmann EA: US drug policy: A bad export. *Foreign Policy* 1988, No. 70, pp 83–108.
2. Benoit E, Coletti RJ: Drugs: The case for legalization. *Financial World*, Oct. 3, 1989, pp 32–35.
3. Baer D: A judge who took the stand: It's time to legalize drugs. *US News and World Report* April 9, 1990, pp 26–27.
4. Nadelmann EA: Drug prohibition in the United States: Costs, consequences, and alternatives. *Science* 1989 245:939–947.

5. Bayer R: Heroin decriminalization and the ideology of tolerance: A critical view in Smith DE, Anderson SM, Buxton M, et al (eds): *A Multicultural View of Drug Abuse. Proceedings of the National Drug Abuse Conference, 1977.* 1978, Hall/Schenkman, pp 56–70.
6. Marshall E: Drug wars: legalization gets a hearing. *Science* 1988; 241:1157–1159.
7. Grinspoon L, Bakalar JB: The harmfulness to: A new approach to drug control. *Hosp Comm Psychiatry* 1990; 41:483.
8. Wilson JQ: Against the legalization of drugs. *Commentary*, Feb. 1990, pp 21–28.
9. Schuckit MA: Should we legalize cocaine? Vista Hill Foundation (San Diego, Ca) *Drug Abuse and Alcoholism, Newsletter* June 1990, Vol. 19, No. 5, pp 1–4.
10. Engelsman EL: Dutch policy on the management of drug related problems. *Brit J Addiction*, 1989; 84:211–218.
11. Linn LS, Yager J, Leake B: Physician's attitudes toward the legalization of marijuana use. *West J Med* 1989; 150:714–717.
12. Jones L: Legalize or prohibit? MDs join debate spurred by "war on drugs." *American Medical News*, Jan. 26, 1990, pp 2, 32.
13. Bakalar JB, Grinspoon L: Why drug policy is so harsh. *Hastings Cent Rep* 1983; 13:34–39.

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ERGOT IN ALCOHOLISM, MORPHINISMS, AND THE GENERAL CLASS OF DRUG-HABIT CASES

The habitual use of other opiates, alcoholic liquors, absinthe, chloral, cocaine, etc., produces similar trains of nervous symptoms, and I therefore class all these poisons together in considering the therapeutic application of ergot to the conditions produced by any one of them.

Before considering the subject proper I wish to place myself in antagonism to a theory widely promulgated by a few and thoughtlessly accepted by many. This theory regards the habitué as the subject of a specific disease which impels him to the habituation; and specific names are given the so-called diseases, such as inebriety, morphiomania, etc., dependent upon the stimulus used by the habitué. The impulse to the use of, or the craving for, the particular stimulus is classed as a symptom of that specific disease, as cough is a symptom of bronchitis, or pain a symptom of neuralgia. A logical sequence to this theory is the irresponsibility of the subject, as it would be manifestly irrational to chide the bronchitic for coughing or the neuralgic for having a pain. I will not now go further into this matter than to say that while I deny *in toto* the existence of such specific diseases I do not question that diseased states of the circulation, of the nervous system and of special organs result from improper use or abuse of these stimuli. To present my ground in another way, I distinctly prefer the old-fashioned, conventional method of hitching the horse to the cart rather than a method which pleases his head where his tail ought to be.

The most striking characteristic of an habitué who leaves off, or is forcibly deprived of, his stimulant, is a disturbance or agitation of the nervous system proportionate to the per diem quantity of the stimulant that had been used. I have seen this agitation so extreme that while continuously unconscious for twenty-four hours the patient would at a bound throw herself from the dorsal recumbent position to the prone, and even from the middle of a wide bed to the floor, before the attendant with one hand upon her could interfere. Every muscle of the body seemed simultaneously active, and the sudden and combined action inexplicable. The sensory nerves were equally alert and especially along the spine was there extreme sensitiveness.

While the cerebro-spinal system was so intensively agitated the vasomotor system seemed paralyzed, with resultant intense hyperemia and sensation of heat to the hand. It was, of course, not possible to take the temperature with a thermometer. The two things that were soothing in this case and that resulted in twenty-four hours in restoring consciousness and calm were ergot hypodermically and galvanization of the spine.

ALFRED T. LIVINGSTON, MD
(*NY State J Med* 1904; 4:28–33)

The public health crisis in New York City

Imminent Peril: Public Health in a Declining Economy. Edited by Kevin M. Cahill, MD. 148 pp. New York, Twentieth Century Fund Press, 1991. $9.95 (softcover)

On August 22, 1991, the New York City Board of Health convened a symposium to address the city's public health crisis. The 11 speakers included Governor Mario M. Cuomo; Mayor David N. Dinkins; Kevin M. Cahill, MD, Senior Member of the Board of Health; Thomas P. Dowling, Esq, Vice-Chairman of the New York State Public Health Council; John Duffy, an eminent public health historian; Congressman Charles Rangel; David Rogers, MD, the Walsh McDermott Professor of Medicine at Cornell University Medical College, and others. The purpose of this symposium was to focus attention on the growing public health crisis in urban centers in the United States, especially in New York City, and to provide solutions for dealing with it. The various presentations have been edited by Dr Cahill in this volume, which provides powerful documentation about the severity of the current public health crisis.

In essence, the 1980s witnessed a progressive disengagement on the part of the federal government from the funding of a broad range of public health services. This decrease in support occurred at a time when the demands for these very services were dramatically rising because of the acquired immunodeficiency syndrome (AIDS) epidemic, illegal drug use, and a host of social and economic problems. By the end of the 1980s, the economic recession had not merely created more unemployment and under-employment, but had also thrown more people into poverty and homelessness. In addition, the attrition of public health programs resulted in the resurgance of old epidemic diseases such as measles, syphilis, and tuberculosis.

The financial ability of local governments to meet ever-increasing demands for services and to compensate for lost federal funding was eroded as the recession caused significant declines in tax revenues. Thus, in spite of the best intentions of governors and mayors, states and localities are simply unable to provide even some essential public health services.

The true gravity of this crisis is clearly illustrated in the stark statistics presented by Mayor Dinkins and Margaret C. Heagarty, MD, Director of Pediatrics at Harlem Hospital Center. Over 34,000 AIDS cases have thus far been reported in New York City. Reported cases of tuberculosis rose 132% between 1980 and 1990, and 2,400 cases of measles were reported in the city in 1990. Since 1986, a major syphilis epidemic has been in progress in New York State and elsewhere, intimately linked to crack cocaine use and the AIDS epidemic. In the first six months of 1991, there were 5,328 cases of early syphilis in New York City. During 1990, there were 11,735 cases in the city compared with slightly more than 2,000 in 1980. In 1990, there were 1,145 cases of congenital syphilis in all of New York State compared with 27 in 1980. These shocking data describe not only increased morbidity and mortality, but also enormous human suffering.

Growing numbers of working poor and the unemployed without medical insurance must be cared for by public institutions. Emergency rooms in places like New York City have become the source of primary care for many, including those who have nowhere else to turn. Yet, this type of use diverts critical care practitioners from caring for those with truly life-threatening emergencies. The decline in the number of primary care physicians has in part contributed to this problem. In spite of the emphasis placed on primary care in medical schools, fewer graduates are choosing careers in primary care areas such as internal medicine, pediatrics, and family practice. This trend will continue as long as status and financial incentives are on the side of specialty practice.

In his essay entitled, "Public Health: Old Truths, New Realities," Governor Cuomo strongly emphasizes the need for health promotion and disease prevention and calls special attention to the social and economic determinants of disease. Among the latter, poverty, joblessness, substandard housing, and the illegal use of drugs figure in the present public health crisis. When he says that local health challenges are really national ones, he touches on the crux of the problem. The federal government has simply walked away from some of the major health problems facing the country. Successful solutions cannot come from cities and states alone, in spite of their good will and their best efforts. As Congressman Charles Rangel notes, "Federal funding for public-health-related programs has declined," and these declines have had a major negative impact nationwide.

The unique character of AIDS as a major public health problem and its role in placing stress on so many of the city's health care institutions are described in detail by Dr Rogers in his essay, "The Special Case of AIDS in Public Health." He not only provides detailed information about the epidemiology of the disease in New York, but also describes the local efforts that have gone into prevention, control, treatment, and research. These efforts have been remarkable when viewed within the national context. Yet, as Dr Rogers notes, the federal commitment to New York City and New York State with regard to AIDS funding has been disappointing.

With its back to the wall, the New York City administration was forced to propose an executive budget in June 1991 that would have reduced the work force of the Department of Health by almost 25%. The cuts included reducing the Bureau of Laboratories by 50%, sexually transmitted disease programs by 30%, and the Bureau of Lead Poison Control by 50%. Other cost reduction measures included the elimination of mental health and dental services, the entire school health and immunization programs, and the bureau that serves families with special needs.

There was an element of history being repeated in this proposal since similar cuts had been required of the department during the 1974–1976 fiscal crisis. At that time, the department lost 1,100 of its 4,400 full-time and 600 of its 1,600 part-time personnel, representing a staff reduction of 28%. It was forced to close 7 of 20 district health centers, 20 of 75 child health stations, 14 of 21 eye clinics, and 6 of 14 chest clinics, and to eliminate 14 of 19 health educators, 18 of 23 nutritionists, and 10 of 12 public health social workers. The list goes on, and indicates the depth and scope of the programmatic and staff reductions made at that time. Yet, a careful and deliberate retrenchment program enabled the department's leadership to retain essential life-preserving services and some components of lower-priority life-enhancing services. By 1977, with the fiscal health of the city improving, some restitutions were made.[1]

There are a number of important differences between 1974–1976 and 1991. The former budgetary crisis occurred against the backdrop of a relatively healthy economy and a fiscally strong state government that was able to come to the city's aid. Reductions were made gradually over a two-year period, allowing for well-planned retrenchment. In contrast, the current New York City fiscal crisis is taking place at the time of a prolonged and severe recession, when the state is facing its own budgetary deficits and in an environment of reduced federal funding where poverty, new and old disease problems, and a lack of health insurance have all created greatly increased demands for publicly sponsored health services.

In 1991, quick action by members of the New York City Board of Health blunted plans for drastically reducing the department's budget. They and the department's leadership, under Dr Margaret Hamburg, the Acting Commissioner of Health, in effect spoke for the poor and needy, who as Dr Cahill notes in his essay, "Averting Disaster: A New York City Case Study," ". . . are often incapable of arguing for their needs and their rights." They also avoided the state of imminent peril that surely would have followed had all of the proposed budgetary reductions been implemented.

New York City is still in the throes of a severe fiscal crisis. Faced with dwindling resources, increased numbers of poor and disadvantaged, and on the daily firing line with epidemics of new and old diseases—AIDS, drug use, syphilis and tuberculosis, the city's public health officials must deal as best they can with both populations and problems that have been orphaned through federal neglect. The contributors to this volume are vivid proof that there is a wealth of good intentions and a commitment on the part of many New Yorkers to deal with this public health crisis. What is now needed is a change in federal policies and priorities to enable them and others who care to implement solutions. Dr Cahill and his co-authors have produced a volume that is sobering because it bares the harsh facts, but it is also extremely heartening because it shows that many people do care.

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1. Imperato PJ: *The Administration of a Public Health Agency: A Case Study of the New York City Department of Health*, New York, Human Sciences Press, 1983, pp 92–99.

Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome

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Indwelling central venous (CV) catheters are being used increasingly to administer long-term antimicrobial and chemotherapeutic therapies in immunocompromised patients, including patients with acquired immunodeficiency syndrome (AIDS). Both thrombosis and infection are well recognized as potential complications of such indwelling catheters.[1,2] Patients with AIDS may be at particular risk for suppurative complications[3,4] because of diminished granulocyte and B lymphocyte function. Previously published data suggest that patients with AIDS and those testing positive for human immunodeficiency virus (HIV) may be at increased risk for bacteremia and its metastatic complications.[5] In this report, we describe two patients with AIDS with indwelling CV catheters who developed superior vena cava syndrome (SVCS) as a complication of central vein suppurative thrombophlebitis. In both patients treatment with medical therapy alone was successful.

CASE REPORTS

Case 1. A 29-year-old man with a history of *Pneumocystis carinii* pneumonia (PCP) and recently diagnosed cytomegalovirus (CMV) esophagitis, gastritis, and duodenitis had a Hickman catheter inserted for ganciclovir infusion. Seven days after catheter placement, a localized exit site infection was noted which initially responded to oral cephalexin. Four days after cessation of antibiotic therapy fever of 103°F and pleuritic chest pain developed. A chest radiograph showed a new lower lobe infiltrate with bilateral pleural effusions. Nafcillin therapy was instituted. Blood cultures drawn from the catheter and peripheral venous sites grew *Staphylococcus aureus* sensitive to nafcillin. Fevers persisted, and four days later the catheter was removed. Seventy-two hours after catheter removal, facial plethora, neck vein distension, and right upper extremity swelling developed.

A chest film showed widening of the superior mediastinum (Fig 1), and a computed tomography (CT) scan of the thorax with contrast revealed thrombus formation extending into the superior vena cava. Contrast venography revealed complete occlusion of the right subclavian vein with multiple collaterals in the neck and shoulder. The patient was treated with streptokinase and heparin. A repeat venogram showed thrombus extension into the brachial vein. Hemoptysis developed and mandated discontinuation of streptokinase after 72 hours. The SVCS gradually resolved with four weeks of intravenous nafcillin and heparin anticoagulation. The patient remained well until nine months later, when he succumbed to recurrent PCP.

Case 2. A 36-year-old woman with CMV esophagitis and retinitis had a left jugular vein Hickman catheter placed for ganciclovir therapy. Three days after surgery fever to 103.6°F, erythema, and tenderness in the subcutaneous tissues surrounding the catheter developed. No fluctuance or pus was noted. Vancomycin therapy was instituted. Chest radiographs taken over the next two days demonstrated progressive mediastinal widening with bilateral pleural effusions. All four peripheral blood cultures obtained grew *S aureus*.

One day after catheter removal local swelling and tenderness of the left neck developed. The swelling progressed and extended into the face and left upper extremity. Left axillary lymphadenitis was noted. A CT scan with contrast confirmed thrombosis of the left internal jugular and brachiocephalic veins (Fig 2) and the superior vena cava (Fig 3). Para-aortic adenopathy and soft tissue swelling in the anterior mediastinum were apparent. Heparin therapy was initiated eight days after removal of the catheter. Daily fevers

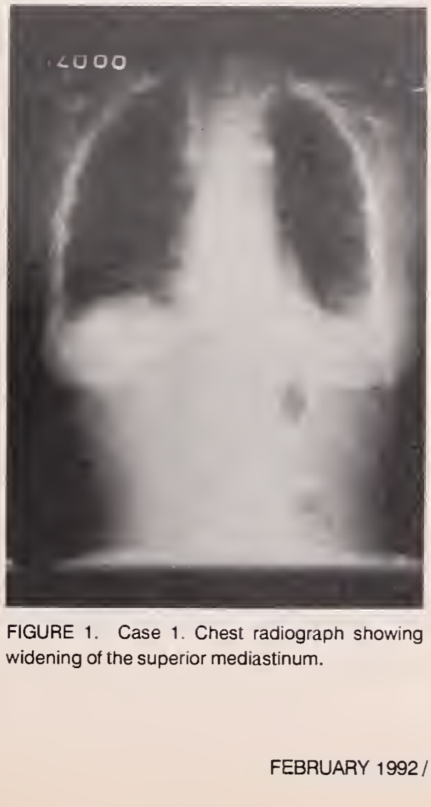

FIGURE 1. Case 1. Chest radiograph showing widening of the superior mediastinum.

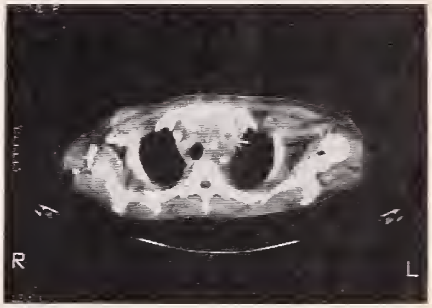

FIGURE 2. Case 2. CT scan of the thorax with contrast confirming thrombosis of the brachiocephalic vein (arrow).

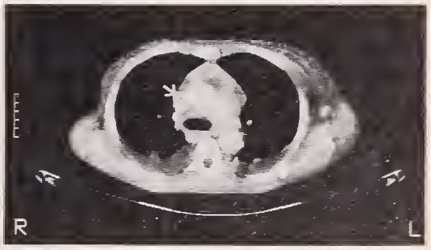

FIGURE 3. Case 2. CT scan of the thorax with contrast confirming thrombosis of the superior vena cava (arrow).

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continued in spite of antibiotic therapy, and blood cultures remained positive 13 days into therapy. Vancomycin was discontinued, and nafcillin and gentamicin were begun. Her fever finally subsided after 18 days of antibiotics. Resolution of facial and arm edema occurred after 17 days. Heparin was continued for 25 days, and nafcillin was continued a total of 42 days (31 days after the last positive blood culture). During this same period, she was successfully treated for PCP. A repeat CT scan performed on day 25 showed residual thrombus in the central veins. She remained without further symptoms of superior vena cava obstruction, but severe wasting developed, and she died six months later from status epilepticus, presumably from central nervous system toxoplasmosis. An autopsy was not performed.

DISCUSSION

The syndrome of SVCS is characteristically associated with the presence of malignancy and portends a poor prognosis.[6] The etiologies of SVCS have changed dramatically since the entity was first reported by Hunter in 1757. Malignancy, including bronchogenic carcinoma, lymphoma, and metastatic carcinoma, is estimated to account for 97% of all cases of SVCS in the adult population.[7] Benign causes of SVCS have been considered rare. The most frequent cause of benign SVCS, granulomatous mediastinitis,[8] constituted only 3%–10% of all cases of SVCS in earlier reviews.[9,10] With the widespread use of indwelling CV catheters, central vein thrombosis is being reported with increasing frequency as a cause of SVCS.[6,11,12] The diagnosis of SVCS in the two patients reported here was suggested by the abrupt onset of facial and upper extremity edema. Chest radiographs supported the diagnosis, showing superior mediastinal widening and pleural effusions in both cases. The lower lobe infiltration observed in the first case was consistent with septic embolization. These clinical findings are in accord with those previously reported in large series.[6,13] In both cases, the CT scan verified thrombosis of the brachiocephalic veins and superior vena cava and was valuable in determining the extent of involvement by thrombosis. A brachial venogram confirmed the diagnosis in the first case. Our observations suggest that CT scan alone is sufficient for diagnosis, since it depicted the site of superior vena cava obstruction and collateral pathways in both cases. Furthermore, the CT scan is able to distinguish between thrombosis and external compression by tumor.

Both patients had resolution of their symptoms after removal of the infected catheter, and institution of antibiotics and anticoagulant therapy. Fibrinolysis with streptokinase was attempted in one patient whose symptoms were present less than 72 hours but was later terminated due to hemoptysis. Lysis of the clot is possible with fibrinolytic agents, but only when the thrombus formation is recent.[11] Heparin may reduce the extent of thrombus and reduce the risk of embolization when the catheter is removed.[11] There is limited published experience supporting the use of surgical debridement of a suppurative central vein,[14] but it may be worthwhile in certain patients. Verghese et al[15] reported two cases of *S aureus* central vein septic thrombophlebitis which did not respond to antibiotic therapy, one involving the subclavian and one the femoral vein. In these two cases, fever and bacteremia resolved after draining of an abscess cavity.

An increased incidence of bacteremia,[5,16] impaired humoral immunity,[16] and depressed neutrophil chemotaxis function[17] in patients with AIDS may contribute to the development of septic thrombophlebitis. Several studies have shown that clinical infection will not occur without thrombus formation; fibrin clot deposits act as a nidus for trapping bacteria protected from host defenses and antibiotics.[2,18] Whether there are additional predisposing factors for thrombus formation in AIDS is unknown. Heightened immune complex deposition, low levels of anti-thrombin III, and AIDS-related tumors associated with hypercoagulable states are theoretical considerations. However, the presence of bacteria, either at the insertion site or in the bloodstream, is probably the critical factor.

Gram-positive organisms, particularly staphylococci, have replaced gram-negative bacteria as the most commonly implicated organisms in CV catheter infections in the immunocompromised host.[2,19] *S aureus* is being increasingly reported as a major cause of catheter sepsis in patients with AIDS.[5,20] Jacobson et al[5] found that intravenous catheters were the most important risk factor in 73% of cases of *S aureus* bacteremia seen among AIDS patients. This study controlled for predisposing factors for staphylococcal infections including intravenous drug use, lymphatic obstruction, and neutropenia. Raviglione et al[3] recently reported a significantly increased incidence of Hickman catheter infections in patients with AIDS when compared to non-AIDS controls. Of the catheter infections in AIDS patients, 87% were due to *S aureau*; septic thrombophlebitis occurred in only 2%. No superior vena cava obstruction was noted. Alterations in B lymphocytes and polymorphonuclear granulocyte function may predispose patients with HIV infection to staphylococcal infection.[5,16,17] The high prevalence of dermatologic disorders, with disruption of normal integumentary barriers, may also contribute to staphylococcal infections in patients with AIDS.

In both patients reported here, septic thrombophlebitis developed from exit site infections within the first week after catheter insertion. The higher infection rate in patients with AIDS reinforces the need for meticulous catheter care and suggests a potential role for prophylactic anti-staphylococcal antibiotics at the time of insertion.

The development of septic thrombophlebitis requires prompt catheter removal and the administration of appropriate antibiotics. Therapy with fibrinolytic agents may be of value if thrombosis is detected early. Surgery was not required in either of these patients but may be a therapeutic option in selected cases. Septic thrombophlebitis must be considered when fever and local infection occur in the setting of an indwelling catheter. Within the context of AIDS, benign as well as malignant etiologies need to be considered in the evaluation of SVCS.

REFERENCES

1. Sitzmann JV, Townsend TR, Siler MC, et al: Septic and technical complications of central venous catheterization. *Ann Surg* 1985; 303:766–770.
2. Press OW, Ramsey PG, Larson EB, et al: Hickman catheter infections in patients with malignancies. *Medicine* 1984; 63:189–200.
3. Raviglione MC, Battan R, Mendez AP, et al: Infections associated with Hickman catheters in patients with AIDS. *Am J Med* 1989; 86:790–786.
4. Saltzman BR, Perlman DC, Levey DI, et al: Infections and complications of indwelling central venous catheters: Comparisons between patients with AIDS and other illnesses. Abstracts of the Fifth International Conference on AIDS, Montreal, June 1989, p 236.
5. Jacobson MA, Gellermann H, Chambers H: Staphylococcus aureus bacteremia and recurrent staph infections in patients with acquired immunodeficiency syndrome and AIDS-related complex. *Am J Med* 1988; 85:172–176.
6. Parish JM, Marschke RF, Dines DE, et al: Etiological considerations in superior vena caval syndrome. *Mayo Clin Proc* 1981; 56:407–413.
7. Lokich JJ, Goodman RL: Superior vena caval syndrome. *JAMA* 1975; 231:58–61.
8. Mahajan V, Strimlan V, Van Ordstrand HS, et al: Benign superior vena cava syndrome. *Chest* 1975; 68:32–35.
9. Lochridge SK, Knibble WP, Dotty DB: Obstruction of the superior vena cava. *Surgery* 1979; 85: 14–24.
10. Kamiya K, Katata Y, Maiki K, et al: Superior vena caval syndrome: Review of the literature and a case report. *Vasc Dis* 1967; 4:59–65.
11. Sculier JP, Feld R: Superior vena cava obstruction syndrome: Recommendations for management. *Cancer Treat Rev* 1985; 12:209–218.
12. Bertrand M, Presant CA, Klein L, et al: Iatrogenic superior vena cava syndrome: A new entity. *Cancer* 1984; 54:376–378.
13. Perez CA, Presant CA, Van Amburg III AL: Management of superior vena cava syndrome. *Semin Oncol* 1978; 5:123–134.
14. Maki DG: Septic thrombophlebitis (part 2). *Hosp Med* 1977; 13:6–29.
15. Verghese A, Widrich WC, Arbeit RD: Central venous septic thrombophlebitis—the role of medical therapy. *Medicine* 1985; 64:394–400.
16. Witt DJ, Craven DE, McCabe WR: Bacterial infections in adult patients with the acquired immunodeficiency syndrome and AIDS-related complex. *Am J Med* 1987; 82:900–906.
17. Nielsen H, Kharazmi A, Faber V: Blood monocyte and neutrophil functions with AIDS. *Scand J Immunol* 1986; 24:291–296.
18. Stillman RM, Soliman F, Garcia L, et al: Etiological catheter-assocaited sepsis: Correlation with thrombogenicity. *Arch Surg* 1977; 112:1497–1499.
19. Jacobs MB, Yeager M: Thrombotic and infections complications of Hickman-Broviac catheters. *Arch Intern Med* 1984; 144:1597–1599.
20. Whimbey E, Gold JWM, Polsky B, et al: Bacteremia and fungemia in patients with the acquired immunodeficiency syndrome. *Ann Intern Med* 1986; 104:511–514.

The strongyloides hyperinfection syndrome

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Chronic infection with *Strongyloides stercoralis* usually causes few or no symptoms. In some individuals, however, *Strongyloides* infection may disseminate and evolve into the hyperinfection syndrome. In this syndrome, any organ of the body can be involved, but the gastrointestinal tract and the lungs are the two most common sites. Involvement of the liver, spleen, lymph nodes, pancreas, gallbladder, ovaries, kidney, prostate, adrenal, thyroid, parathyroid, heart, meninges, and brain have been described.[1-5] The hyperinfection syndrome may be accompanied by gram-negative septicemia, as bacteria may enter the circulation via the migrating larvae or through gastrointestinal ulcerations.

CASE REPORT

A 41-year-old woman with stage IV lymphoma diagnosed four months before admission was admitted to Long Island Jewish Medical Center with fever, abdominal pain, nausea, vomiting, and diarrhea. She had had fever to 39°C for one day; three to four loose stools per day for three days; and abdominal pain, nausea, vomiting, and anorexia for 14 days. During the previous six months she had been treated with two courses of chemotherapy (cyclophosphamide, doxorubicin, vincristine, prednisone, etoposide), the last, one week earlier. She complained of marked generalized weakness and a weight loss of 35 lb. She was born in Jamaica and had emigrated to the United States at the age of seven. There was no recent travel history.

Physical examination revealed a cachectic female with a temperature of 39.4°C; pulse, 104/min; and blood pressure, 100/70 mm Hg. She had small, non-tender lymph nodes in her neck and axillae bilaterally. Cardiac and chest examinations were normal. The abdomen was moderately distended with tenderness in the epigastrium and left lower quadrant. There was no rigidity or rebound tenderness, and bowel sounds were present. There was no perirectal tenderness, and the stool guaiac test was negative. The Hickman catheter site showed no signs of infection.

A complete blood cell count revealed a hemoglobin level of 10.5 g/dL and a white blood cell count of 700/mm^3 with 52% neutrophils, 44% lymphocytes, and 4% monocytes. Serum chemistries showed a sodium of 128 mmol/L; potassium, 3.0 mmol/L; urea nitrogen, 5 mL/dL; creatinine, 0.7 mg/dL; glucose, 141 mg/dL; calcium, 84 mg/dL; phosphorus, 1.9 mL/dL; uric acid, 7.5 mg/dL; total protein, 5.4 g/dL; and albumin, 2.8 g/dL. Serum aspartate aminotransferase (ASAT) was 54 U/L; alanine amino transferase (ALAT), 25 U/L; lactic dehydrogenase (LDH), 825 U/L; amylase, 54 U/L; and alkaline phosphatase, 92 U/L. A roentgenograph of the chest was normal. Stools for culture, sensitivity, ova, and parasites were negative for pathogens.

The patient was given 2 g of parenteral ceftazidime every eight hours, gentamicin 80 mg every eight hours, and vancomycin, 1 g every 12 hours. Her fever decreased within three days. An upright film of the abdomen showed a non-specific gas pattern with multiple air fluid levels. A barium enema was normal. An upper gastrointestinal series with small bowel follow-through showed possible partial obstruction in the lower jejunum or upper ileum. Gastroduodenoscopy revealed no gross abnormalities.

Two weeks after admission intravenous doxorubicin, 50 mg/m^2 intravenous bleomycin, 6 mg/m^2, and prednisone, 60 mg/day, were started. The same day, the patient became febrile, and intravenous ceftazidime, two every eight hours, and tobramycin, 80 mg every eight hours, were begun. The patient's temperature decreased, and she remained afebrile for one week until a temperature of 38.3°C was noted. Vancomycin was added to the above regimen. The patient continued to have fever and abdominal discomfort. Her prednisone dosage was tapered to 0 mg five days later. Ten days after admission a complete blood count revealed a total white cell count of 2,100 with 36% eosinophils. All antibiotics were discontinued with a gradual decrease in temperature to normal. Three days later a large bowel biopsy showed eosinophilic infiltration.

Subsequently, a stool examination showed motile *S stercoralis* larvae with occasional eggs. Twenty-four days later the patient was started on thiabendazole, 25 mg/kg. Three to four hours after thiabendazole was started, the patient became dyspneic and hypotensive. Imipenem, 500 mg every six hours, and amikacin, 500 mg every 12 hours, were begun. Her pulse was 240/min; temperature, 38.5°C; and respiratory rate, 60/min. Arterial blood gas studies on Fio$_2$ of 100% (nasal rebreather) showed a pH of 7.58, with Pco$_2$ of 46, and O$_2$ saturation of 87%. The chest roentgenogram was negative. The patient remained dyspneic. Two blood cultures were negative. A day later, the chest film revealed bilateral diffuse alveolar infiltrates, and a nasotracheal tube was placed. A bronchial wash specimen disclosed motile larvae of *S stercoralis.* Three weeks after admission ivermectin, 10 mg, was obtained from the Centers for Disease Control and given to the patient for two days. Thiabendazole, 25 mg/kg, was continued for two days with the ivermectin, 10 mg per day. Two days later the patient's clinical status improved with clearing of her chest film and a decrease in temperature.

Four days later, the patient developed a leakage in her endotracheal tube. During re-intubation the patient became bradycardic and hypotensive and had a generalized seizure. After this episode, she remained unresponsive. Repeat bronchial alveolar lavage revealed no larvae. The chest film continued to show resolving infiltrates.

The patient's neurologic and general condition gradually deteriorated, and she expired. Autopsy was refused.

DISCUSSION

The hyperinfection syndrome is usually seen in immunosuppressed individuals, but may also occur in the normal host. In one review of 103 cases of hyperinfection syndrome, 89 of the patients were immunocompromised by disease, therapy, or both at the time of diagnosis.[6] Steroid use, as seen in the patient discussed here, is the most frequently associated contributing factor to the hyperinfection syndrome. These agents increased the worm load either by enhancing production of larvae by female worms or by accelerating the conversion of rhabditiform to infective form larvae in the intestinal tract.[7-10] The hyperinfection syndrome is also seen in conditions associated with T-cell dysfunction, such as Hodgkin's disease and non-Hodgkin's lymphoma. Other predisposing factors to the hyperinfection syndrome include anatomic and functional gastrointestinal disorders.[11-14] Decreased gastrointestinal motility, as seen in paralytic ileus, can also increase the worm load by providing more time for rhabditiform larvae to mature into infective filariform larvae in the intestinal tract.[13]

The increased worm load in the gastrointestinal tract may cause abdominal pain, nausea, vomiting, anorexia, constipation, diarrhea, paralytic ileus, or gastrointestinal ulceration. The bowel may perforate with resultant secondary peritonitis.[15]

Pulmonary involvement may manifest as

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cough, dyspnea, wheezing, hemoptysis, pulmonary infiltrate (local or diffuse), and rarely pleural effusion.[3,13,16] Involvement of the central nervous system and heart has also been described.[17]

The results of treatment of *Strongyloides* infection in general and more specifically to the hyperinfection syndrome have been somewhat disappointing. Thiabendazole, which has been the drug of choice for many years, has an efficacy rate of only 75% in chronic strongyloidiasis.[18] This agent has frequent side effects, including nausea, vomiting, diarrhea, and dizziness. Less commonly, it has been associated with pruritis, neurotoxicity, bradycardia, hypotension, and leukopenia.[6,19] Other drugs such as mebendazole, albendazole, levamisole, cambendazole, and pyrental pamoate have been used in the treatment of *Strongyloides* but with varying degrees of efficacy.[20–22]

Treatment of the hyperinfection syndrome is more problematic. If diagnosed early and treated promptly, a cure rate of 60% has been achieved in immunocompetent patients. However, in immunosuppressed individuals, a mortality rate of as high as 86% has been reported.[6] Because of these poor results, new agents are being investigated to treat strongyloides.

The anti-helminthic drug, ivermectin, has been widely used to treat helminthic infections in animals. It is the 22,23-dihydro derivative of ivermectin B1, a macrocyclic lactone produced by an actinomycetes, *Streptomyces avermitilis.* It is thought to act as a gamma aminobutyric acid antagonist (GABA), disrupting GABA–mediated neurosynaptic transmission in the worm, with eventual paralysis and death of the parasite. Because ivermectin does not easily cross the blood brain barrier in humans, it can be used safely. Humans have well tolerated doses of up to 200 mg/kg. The side effects of ivermectin are mild constipation and transient elevations of serum ASAT/ALAT, creatinine, and alkaline phosphatase.

Ivermectin has been used successfully in the treatment of human onchocerciasis and bancroftian filariasis.[23,26] In one recent study in Peru, involving 110 patients with chronic interstitial strongyloidiases, an overall cure rate of 88% was achieved with a single or double dose of ivermectin.[26] Another report describing Guatemalan patients with intestinal strongyloidiasis cited a cure rate of almost 100%, although only three patients were evaluated.[8]

The patient described here continued to have motile strongyloides larvae in her bronchial wash in spite of three days of combined treatment with ivermectin and thiabendazole and a total of seven days of treatment with thiabendazole. Slow clearance of larvae in an immunosuppressed patient with *Strongyloides* infection is not unusual.[27] Several factors may have contributed to the slow eradication of larvae in this patient. Paralytic ileus, which was present in this patient, may have decreased the absorption of drug in the intestinal tract. Additionally, there may have been more time for rhabditiform larvae to mature into infective filariform larvae. The possible presence of blind loops, due to either lymphatic compression of the gut or tumor invasion, may have enhanced development and multiplication of the parasite, by protecting the worm from host defenses and anti-helminthic agents. Recent steroid use may have impaired eosinophil function, the most important arm of defense against *Strongyloides.*

The diagnosis of strongyloidiasis was delayed in this patient because of several negative stool examinations for ova and parasites and because of the absence of a peripheral blood eosinophilic response. Stool samples may be negative, although duodenal aspirates are positive in infected patients. In addition, eosinophils may not be evident, and this is a poor prognostic sign.

In spite of her eventual death, the patient's general condition and respiratory status improved during her hospital course. Her death was due to neurologic damage after a leakage of her endotracheal tube resulted in cardiopulmonary arrest. Since some improvement was seen after adding ivermectin to the drug regimen, it may have helped in the eradication of the larvae. The picture is confounded by the simultaneous administration of thiabendazole.

From the past available data on the human use of ivermectin for *Strongyloides,* it is unclear as to the clinical application of this agent. Preliminary reports seem encouraging, but controlled studies comparing thiabendazole would be helpful in defining the role, if any, of ivermectin in human *Strongyloides* infections.

This patient's course underlines an important clinical problem. Potentially immunocompromised patients with a history of residence or travel to an endemic area of *Strongyloides* should be aggressively evaluated for the presence of helminths before immunosuppressive therapy. In the symptomatic patient, periodic searches for the presence of *Strongyloides* larvae in the stool, duodenal fluid, or sputum should be done. If there is a history of endemic exposure, an empiric course of thiabendazole should be considered before immunosuppressive therapy.

REFERENCES

1. Brown HW, Perna VP: An overwhelming *Strongyloides* infection. *JAMA* 1958; 168:1648–1651.
2. Faust EC, DeGroat A: Internal autoinfection in human strongyloidiasis. *Am J Trop Med* 1940; 20:359.
3. Kyle LH, McKay DG, Sparling HJ: Strongyloidiasis. *Ann Intern Med* 1948; 29:1014–1042.
4. Yim Y, Kikkawa Y, Tanowitz H, Winters M: Fatal strongyloidiasis in Hodgkin's disease after immunosuppressive therapy. *J Trop Med Hyg* 1970; 3: 397–407.
5. Imperato PJ: Strongyloidiasis: A dangerous opportunistic infection. *NY State J Med* 1984; 84:379–380.
6. Igra-Siegman Y, Kapila R, Sen P, et al: Syndrome of hyperinfection with *Strongyloides stercoralis. Rev Infect Dis* 1981;3:397–407.
7. Neva FA: Biology and immunology of human strongyloidiasis. *J Infect Dis* 1986; 153:397–406.
8. Freedman DO, Zierdt WS, Lujan A: The efficacy of ivermectin in the chemotherapy of gastrointestinal helminthiasis in humans. *J Infect Dis* 1989; 159:1151–1153.
9. Kimmelstiel F, Lange M: Fatal systemic strongyloidiasis following corticosteroid therapy. *NY State J Med* 1984; 84:399–401.
10. Purtilo DT, Meyers WM, Connor DH: Fatal strongyloidiasis in immunosuppressed patients. *Am J Med* 1974; 56:488–493.
11. Dwork KG, Jaffe JR, Lieberman HD: Strongyloidiasis with massive hyperinfection. *NY State J Med* 1975; 75:1230–1234.
12. Pollack TW, Perencevich EN: Hyperinfection with *Strongyloides stercoralis* in a patient with Hodgkin's disease. *JAMA* 1976; 76:171–175.
13. Scowden EB, Schaffner W, Stone WJ: Overwhelming strongyloidiasis: An unappreciated opportunistic infection. *Medicine* (Baltimore) 1978; 57:527–544.
14. Cruz T, Reboucas G, Rocha H: Fatal strongyloidiasis in patients receiving corticosteroids. *N Engl J Med* 1966; 275:1093–1096.
15. Lintermans JP: Fatal peritonitis: An unusual complication of *Strongyloides stercoralis* infection. *Clin Pediatr* (Phila) 1974; 14:974–975.
16. Rassiga AL, Lowry JL, Foreman WB: Diffuse pulmonary infection due to *Strongyloides stercoralis. JAMA* 1974; 230:464–427.
17. Meltzer RS, Singer C, Armstrong D, et al: Antemortem diagnosis of central nervous system strongyloidiasis. *Am J Med Sci* 1979; 277:91–98.
18. Franz KH: Clinical trials with thiabendazole against human strongyloidiasis. *Am J Trop Med Hyg* 1963; 12:211–214.
19. Grove DI: Treatment of strongyloidiasis with thiabendazole: An analysis of toxicity and effectiveness. *Trans R Soc Trop Med Hyg* 1982; 76:114–118.
20. Chavarria AP, Swartzwelder JC, Villarejos VM, et al: Mebendazole, an effective based spectrum antihelminthic. *Am J Trop Med Hyg* 1973; 22: 592–595.
21. Rossignol JF, Maisonneuve H: Albendazole: Placebo-controlled study in 870 patients with intestinal helminthiasis. *Trans R Soc Trop Med Hyg* 1983; 77: 707–711.
22. Wang CC, Galli GA: Strongyloidiasis treated with pyrvinium pamoate. *JAMA* 1965; 193:191.
23. Greene BM, Taylor HR, Cupp EW, et al: Comparison of ivermectin and diethylcarbamazine in the treatment of onchocerciasis. *N Engl J Med* 1985; 313:133–138.
24. Kumaraswami V, Ottensen EA, Vijaryasekaran V, et al: Ivermectin for the treatment of onchereria bancrofti filariasis: Efficacy and adverse reactions. *JAMA* 1988; 259:3150–3153.
25. Aziz MA, Diallo S, Diop IM, et al: Efficacy of ivermectin and diethylcarbamazine in the treatment of onchocerciasis. *Lancet* 1982; 2:1171–1173.
26. Naquira C, Jimenez G, Guerra JG, et al: Ivermectin for human strongyloidiasis and other intestinal helminths. *Am J Trop Med Hyg* 1989; 40: 304–309.
27. Smith B, Verghese A, Guiteriez C, et al: Pulmonary strongyloidiasis: Diagnosis by sputum gram stain. *Am J Med* 1985; 79:663–666.

Gastrocolic fistula complicating a benign gastric ulcer

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Gastrocolic fistula complicating a benign gastric ulcer is a rare occurrence. We report such a case, initially diagnosed by gastroscopy and subsequently confirmed by upper gastrointestinal barium study, computed tomography (CT), and surgery.

Case Report

A 71-year-old man sought treatment for weakness and melena. He had been living in Mexico with his wife for the previous six months. Four months before admission he began experiencing crampy abdominal pain without a change in bowel habits. Little pain relief was afforded by 5–10 daily tablets of an over-the-counter product containing aspirin, 325 mg; sodium bicarbonate, 1,916 mg; and citric acid, 1,000 mg. Two months before admission he began having three loose black bowel movements each day and increasing pain unrelated to meals or bowel movements. He had lost 15 pounds in six months.[3]

The patient had no history of peptic ulcer disease and used alcohol sparingly. He had smoked two packs of cigarettes per day for 50 years. He had a history of adenomatous colonic polyps and had undergone colonoscopy at our laboratory seven months prior to admission, the results of which were normal. His surgical history included coronary artery bypass and right femoral-popliteal bypass in 1984. He had been maintained on enteric-coated aspirin, 325 mg; furosemide, 40 mg; and potassium chloride, 20 mEq daily.

Physical examination revealed a well-developed man in no acute distress. His blood pressure was 120/80 mm Hg, and pulse was 84/min without orthostasis. He was afebrile. The abdomen was soft, with tenderness in the mid-epigastrium on deep palpation. Bowel sounds were present. Rectal examination revealed melena, and nasogastric aspiration yielded foul smelling coffee ground material. His hematocrit was 18%, which decreased from 45% at the time of colonoscopy. The white blood cell count, electrolyte levels, liver chemistries, and amylase count were all within normal limits, but albumin and total protein levels were slightly depressed. The chest film was within normal limits, without evidence of free air under the diaphragm.

Endoscopy of the upper gastrointestinal tract was performed. The esophagus, cardia, and fundus of the stomach were normal. A large gastric ulcer was seen along the greater curvature in the body of the stomach. Haustral folds were present in the center of the ulcer base. The endoscope was gently advanced 10 cm into a fistulous tract. Normal colonic mucosa was encountered. The antrum, pylorus, and duodenal bulb and sweep were normal. In the setting of acute upper gastrointestinal bleeding, biopsies of the ulcer were deferred. After discussion with the surgical staff, it was decided not to repeat the endoscopy with biopsies, as surgery appeared to be mandatory whether the process was benign or malignant.

To better define the fistulous region, an upper gastrointestinal series with barium and a CT scan of the abdomen were performed (Figs 1,2). The barium study showed a gastric ulcer communicating with the transverse colon. The CT scan was consistent with a gastrocolic fistula.

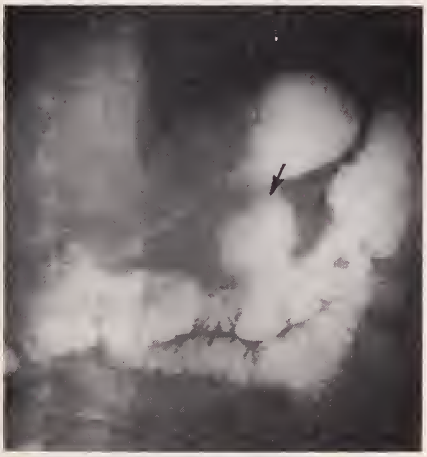

FIGURE 1. Upper gastrointestinal series revealing a gastric ulcer communicating with transverse colon.

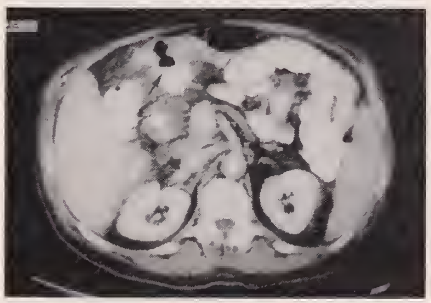

FIGURE 2. CT scan of abdomen with air and contrast material in a gastrocolic fistula.

The patient underwent laparotomy. A frozen section of the ulcer and fistulous tract revealed no evidence of malignancy. Resection of the fistulous tract, partial colectomy, and closure of the defect in the stomach were performed. The surgical team opted not to perform a partial gastrectomy.

The surgical specimen contained a benign gastric ulcer communicating with the transverse colon. The postoperative course was uneventful.

Discussion

This is a case of gastrocolic fistula complicating a benign gastric ulcer. Gastrocolic fistula has been reported to occur secondary to gastric resection and Billroth II anastomosis, or secondary to gastric and colonic carcinoma.[1] Other benign precipitating conditions include trauma, inflammatory bowel disease, irradiation, diverticular disease, tuberculosis, and pancreatic abscess.[1,2]

Gastrocolic fistula is uncommon as a complication of benign gastric ulcer not associated with previous gastric surgery. Approximately 100 cases have been reported in the literature. There have been overlapping reviews of these cases.[3–6] Common presenting symptoms include weight loss, abdominal pain, diarrhea, melena, and feculent vomiting. The use of steroids or nonsteroidal anti-inflammatory agents among these patients is common. In one review, there was ongoing use of these drugs in 18 of 24 patients (75%).[6]

Most of the ulcers associated with gastrocolic fistula are located in the distal two thirds of the stomach, usually on the greater curvature.[3] Imaging techniques such as upper and lower gastrointestinal barium studies and upper gastrointestinal endoscopy are helpful in making the diagnosis. Sensitivity for barium enema has ranged between 90%–100%, compared with 75% for barium meal, and 30%–75% for gastroscopy, although the latter has been employed much less commonly.[3,6]

Although there have been reports of successful outcomes with medical therapy,[7,8] surgery has been the standard approach. Hemigastrectomy, excision of the fistula, and resection of the segment of

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involved colon is the procedure most commonly employed.

This patient is representative of the cases previously described in the literature in terms of symptoms and location of the ulcer. His moderate to heavy use of daily aspirin may have been largely responsible for the ulcer. The gastrocolic fistula was demonstrated by both gastroscopy and upper gastrointestinal radiographic series. Because primary malignancy was a likely cause, the CT scan of the abdomen was helpful in ruling out obvious metastatic disease before surgery. This case is atypical, as the gastrocolic fistula was initially diagnosed by gastroscopy.

REFERENCES

1. Amlicke JA, Ponka JL: Gastrocolic and gastrojejunocolic fistulas. A report of sixteen cases. *Amer J Surg* 1964; 107:744–750.
2. Cody JH, Divincenti FC, Cowick DR, et al: Gastrocolic and gastrojejunocolic fistulae: Report of twelve cases and review of the literature. *Ann Surg* 1975; 181:376–380.
3. Akware OE, Edis AJ, Wollaeger EE: Gastrocolic fistula complicating benign unoperated gastric ulcer. Report of four cases and review of the literature. *Mayo Clin Proc* 1976; 51:233–230.
4. Madsen RE, Chandler KA, Rudd GP: Gastrocolic fistula complicating benign gastric ulcer: Report of a case and review of the literature. *J Am Osteopath Assoc* 1978; 77:684–688.
5. Frikker MJ, Lucas RJ: Gastrocolic fistula caused by benign gastric ulcer in the patient who has not had prior operation: Case reports and review of the literature. *Am Surg* 1986; 52:446–451.
6. Soybel DI, Kestenberg A, Brunt EM, et al: Gastrocolic fistula as a complication of benign gastric ulcer: Report of four cases and update of the literature. *Br J Surg* 1989; 76:1298–1300.
7. Strang GL, Espiner HJ, Mather GH: Benign gastrocolic fistula healing with conservative management. *Arch Surg* 1977; 112:651–654.
8. McCullough KM, Gregson R: Spontaneous healing of a gastrocolic fistula due to a benign gastric ulcer. *Clin Radiol* 1987; 38:431–433.

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PERIDURAL ANESTHESIA FOR THORACIC SURGERY

Peridural anesthesia is by no means a new method of anesthesia.[1] Because of the technical difficulty associated with its administration and the relatively high incidence of unsuccessful blocks resulting from its use, it has not received the attention given other methods of anesthesia. Peridural anesthesia is a method of regional anesthesia in which obtundation of nerve impulses is obtained by the injection of a local anesthetic drug into the space surrounding the spinal dura mater but within the vertebral canal. This space is referred to as the peridural or extradural space and extends from the sacral hiatus to the foramen magnum. It contains venous and arterial plexuses, lymph vessels, adipose and connective tissues, and serves as a dispersal center for the anesthetic solution. The drug must then spread to each paravertebral foramen where the spinal nerve roots have joined to form a mixed spinal nerve and is no longer ensheathed by the dura mater. Conduction is blocked at this area, and the resulting anesthesia is actually a multiple paravertebral block.

The technic involves all cervical and thoracic nerves. Since these particular segments of the nervous system control most of the muscles of respiration, interruption of their motor elements would be hazardous. Considerable attention must, therefore, be given the anesthetic drug. For the anesthesia to be satisfactory the drug must be in sufficient concentration to offer sensory but not motor anesthesia. It must offer a rapid onset and be sufficiently prolonged to outlast the surgical procedure. Finally, the resulting anesthesia must be profound enough in nature to allow extensive surgical manipulation.

Xylocaine has proved decidedly superior to any other local anesthetic drug for peridural anesthesia. Prior to its introduction it was necessary to allow for a latency period of twenty to twenty-five minutes. This has now been reduced to ten minutes. Because of its unusual diffusion ability the incidence of incomplete or partial block is unbelievably small. It offers a very profound anesthesia in dilute solutions which in turn has markedly lowered the incidence of toxic reactions as was previously experienced with stronger solutions of procaine.

ORAL B. CRAWFORD, MD
(*NY State J Med* 1952;52:2637–2641)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Needle localization and biopsy in non-palpable lesions of the breast: An eight-year experience

TO THE EDITOR: Among the most terrifying prospects a woman faces in her life is the possibility of breast cancer. Kelsey and Gammon have stated that one in nine American women will develop breast cancer at some time in her life.[1] It is also projected that breast cancer will account for 175,000 new cases, with 44,500 deaths, in the United States for 1991. Although age-adjusted mortality rates have remained essentially constant since 1930, there has been a continuing and increasing drive to detect and treat these malignancies in their earlier stages, hoping to affect greater cure rates. Along with the campaign for increased public awareness, screening, risk factor identification, and self-examination, it was only natural that radiologic techniques would be employed to facilitate earlier diagnosis. The purpose of this communication is to ascertain the accuracy of the needle localization technique and the influence it has had on the earlier diagnosis of breast cancer.

Materials and Methods. From 1983 to 1990, 777 patients underwent needle localization and biopsy for non-palpable breast lesions at the Cabrini Medical Center. In all patients, the biopsy specimens were X-rayed and were found to contain the suspicious areas noted on mammography.

Results. Of the 777 biopsy specimens, 111 proved to be malignant, producing an overall positive yield of 14.3%. These figures can be compared to those of Meyer et al[2] who found 18.8% malignancies, Stock et al,[3] who found 16% and Denning et al,[4] who discovered 19%.

The diagnostic accuracy of the needle localization technique can be assessed by perusing the follow-up course of these patients. Of the 666 benign cases, only four (0.6%), later presented with malignancy within the ipsilateral breast in the vicinity of the previous biopsy. The new lesions became evident anywhere from four months to three years after the biopsy. Figure 1 illustrates the routine mammogram taken of the left breast of one of these patients, a 45-year-old female who demonstrated no palpable masses. Two areas, one showing a suspicious stellate pattern and the other an area of microcalcifications, were excised using the needle localization technique. Figure 2 shows the specimen radiogram of the biopsy containing the area of microcalcifications. The pathology reports on the two biopsies were as follows: Fibroadenoma of breast with adenosis and cysts and duct cell hyperplasia, microcalcifications, and cysts with adenosis. Four months later, the patient returned to the hospital with masses in the areas of the previous biopsies and therefore underwent reoperation. At this time, the pathology of the area that had contained the microcalcifications revealed multiple areas of intraductal carcinoma. The other area revealed merely subcutaneous and scar tissue.

Discussion. In our opinion, the appearance of a subsequent malignancy indicates one of several possibilities. It might be, as in the case cited above, that a mistaken diagnosis was made in interpreting the original biopsy. It might also be that the biopsy was of insufficient size to include the malignant lesion, or that the needle failed to zone in accurately on the suspicious area. Finally, the initial negative needle localization and biopsy may have been carried out at a time before the development of a malignancy.

Through the cooperation of the Tumor Registry, the stages of all breast cancer cases treated at Cabrini Medical Center for the years 1983 through 1990 were obtained. From these data, an interesting and encouraging trend appears. In 1981, before the introduction of needle localization and biopsy, 18 of 120 (15%) of breast cancers were Stage I at the time of initial diagnosis and treatment. This is in marked contrast to 1990, the final year of this study, when 42 of 94 (44.7%) of breast cancers were found to be Stage I. Thus, in eight-years, the ability to detect and treat this disease at an earlier stage appears to have trebled.

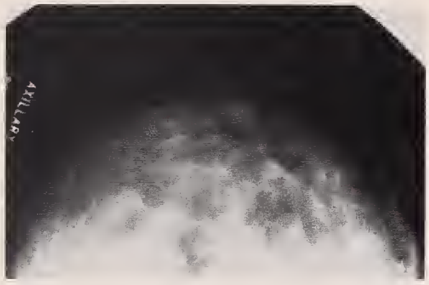

FIGURE 1. Mammogram showing two areas of abnormality, the one circled contained the microcalcifications.

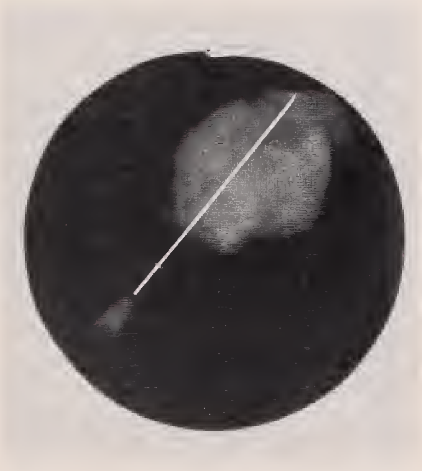

FIGURE 2. Specimen radiogram revealing suspicious area containing microcalcifications, visible in the upper right portion of the film.

Needle localization followed by biopsy of suspicious non-palpable lesions noted on mammography is an accurate method of early detection of breast cancer carrying with it a very low incidence of incorrect diagnoses. The technique has led to a vastly improved number of patients who can receive treatment during Stage I of their disease.

We should like to express our thanks to Allison Attong-Rogers of the Cabrini Tumor Registry for her able assistance in assembling the statistical data in this report.

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1. Kelsey JL, Gammon MD: The epidemiology of breast cancer. *Ca* 1991; 41:146.
2. Meyer JE, Eberlein TJ, Stemper PC, et al: Biopsy of occult lesions. Analysis of 1,261 abnormalities, *JAMA* 1990; 263:2341–2343.
3. Stock RJ, Kunschner A, Mazer J, et al: Mammographic localization and biopsy: The experience of a gynecologic oncology group. *Gynecol Oncol* 1989; 33:172–176.
4. Denning DP, Farha GJ, McBoyle MF: Role of needle localization of nonpalpable breast lesions. *Am J Surg* 1987; 154:593–596.

Phenytoin and smoking

TO THE EDITOR: Phenytoin, used extensively for its anti-convulsant effect, is also known to have a calming effect on violent psychiatric patients. A cross-sectional pilot study was conducted to determine whether this calming effect translates into any reduction in the prevalence and intensity of cigarette (cgs) smoking, which are known to be quite high in the psychiatric hospital population.

At the Manhattan Psychiatric Center, in June 1991, there were 22 patients on neuroleptic agents, who were also receiving phenytoin in oral doses of 100–500 mgm per day for the treatment of seizure disorders. The control group consisted of 22 age- and sex-matched patients on neuroleptics but not on phenytoin. In each group, there were 13 men and 9 women, aged 23 to 74 years. The intensity of smoking habits of patients in both groups was categorized as nominal (1–5 cgs/day), moderate (6–20 cgs/day) and severe (more than 20 cgs/day).

Of the total of 44 patients, 11 (25%) were non-smokers, and 33 (75%) were smokers. This prevalence of smoking is approximately three times the national average. In the phenytoin group, there were 8 of 22 (36.4%) non-smokers, in contrast with only 3 of 22 (13.6%) in the control group.

Nearly two-thirds of the patients in the phenytoin group were either non-smokers, or were nominal smokers. By contrast, two-thirds of the patients in the control group were either moderate or severe smokers. These results were statistically significant at $p < 0.05$ ($\chi^2 = 4.46$, df: 1).

In this group of patients, phenytoin therapy is associated with a lower prevalence and a lower intensity of smoking. These findings are potentially important from the public health and other points of view.

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Hospice care

TO THE EDITOR: I read with great interest in the November 1991 *Journal* Dr Peter Rogatz' paper[1] describing the Visiting Nurse Service hospice care program. It is clearly a resource of great value. I was surprised, however, by the statement that the first hospice in the United States was founded in 1974 in New Haven, Connecticut. I believe that Calvary Hospital in the Bronx was functioning, in effect, as a hospice for many years previous to this. I make this seemingly small point because I believe that the remarkable contributions of Dr James Cimino, Medical Director of Calvary since the 1960s, have had too little recognition.

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[1] Rogatz P, Schwartz F, Dennis J: A community-based hospice program. *NY State J Med* 1991; 91:500–502.

FROM THE LIBRARY

ANOREXIA NERVOSA IN AN INFANT

The term anorexia nervosa is of course self explanatory, but it would seem wise to restrict the use of the term. All gradations of the disease are to be found. Those varying from simple, almost insignificant phases, to those of extreme severity, terminating fatally.

No reference will be made to this condition so commonly found in young children, neither will the common perversions of appetite be considered. These latter have been well worked out and need have no place here.

A negative statement possibly best suggests the application of the term, anorexia nervosa that is, if we are able to find any organic lesion causing the condition we are not dealing with true anorexia nervosa. Forchheimer has expressed it as follows: "There must be absence of organic lesion which would explain the anorexia."

The condition in infants is extremely rare, to judge from the reports, and while nothing done in this case adds to our knowledge of the treatment neither does it throw any light upon the etiology, yet it does seem worthy of report.

The consensus of opinion at present is that it is a neurosis, and while it is extremely interesting to attempt to grasp the underlying psychology, at the same time it must be confessed that there are too many unknown factors in the equation.

Precocity on the part of the infant, or the presence of a very delicately balanced nervous organism in either parent must not be disregarded as factors. To what extent they obtain as a suggesting force must be left undecided.

To sum up the case briefly, we have a healthy infant, unencumbered by parental taint, manifesting a determination not to take food. So far as may be determined the sole cause was the substitution of a new nipple for an old one. A persistent refusal, with a resultant loss of nearly three pounds, to take any food offered in any manner.

Added to this the lack of any organic lesion which we could discover, to explain the disease. Gavage lasting four days, followed by a gradual surrender on the part of the infant.

NORRIS G. ORCHARD, MD
(*NY State J of Med* 1914; 14:36–38)

Reprinted from the July/August 1991 issue of Epidemiology Notes *(Vol 6, No 7), published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

The New York State Dietary Survey: Fat and cholesterol consumption

Introduction

Substantial scientific evidence exists linking serum cholesterol, particularly low-density and very-low-density lipoprotein cholesterol, to the development of coronary heart disease.[1-3] A large body of scientific literature has also established the relationship between diet and serum lipids.[4-7] The interrelationship among diet, serum lipids, and coronary heart disease forms the basis for recommendations to decrease an individual's risk for developing heart disease by lowering the intake of total fat, saturated fat, and cholesterol.

The National Cholesterol Education Program (NCEP) has made dietary treatment the cornerstone of therapy to reduce blood cholesterol levels.[8] NCEP guidelines, as a first step of dietary treatment, call for a daily intake of saturated fat of less than 10% of calories, total fat of less than 30% of calories and dietary cholesterol of less than 300 mg/day. The same recommendations for fat intake are included in *Healthy People 2000: National Health Promotion and Disease Prevention Objectives for the Nation* as an objective to reduce the risk of heart disease. *Healthy People 2000* is a report from the US Department of Health and Human Services, Public Health Service, establishing national health objectives for significant reductions in preventable death and disability, enhanced quality of life, and reduction in the disparity in health status among population groups in the United States.[9]

New York State Dietary Survey

The New York State Dietary Survey is a statewide survey that was conducted in the spring of 1989 to assess the nutrient intake and food consumption patterns of adults 18–64 years of age in New York State. The food frequency questionnaire (FFQ) designed by G. Block and co-workers at the National Cancer Institute was selected for use in this study.[10,11] The choice of this dietary methodology was based on a number of considerations including the following: (1) its validity and reliability, (2) the ability to provide reasonable estimates of a wide range of nutrients, (3) the ability to provide information on food consumption patterns, (4) feasibility and cost-effectiveness, (5) incorporation into an ongoing system of dietary surveillance, and (5) comparability with other surveys.

The Block FFQ includes 60 food items. Respondents are also asked to report the frequency of consuming these foods over the past year. Respondents are asked to report portion sizes for their usual servings of the food items in relative terms (small, medium and large). The interviewer describes what is meant by the different portion sizes. Information on frequency of consumption, together with serving size data, can then be converted to estimate the average daily intake of specific nutrients. Additional information was obtained regarding the frequency of restaurant use and cooking practices in the home, such as trimming the fat from meat, eating the skin on poultry, and the use of fat and oils in cooking. The intake of nutrients in a particular diet was calculated using the nutrient composition of the food, age- and sex-specific portion size and the frequency of consumption. Adjustments in the estimates were made according to reported restaurant usage and cooking practices.

Respondents were contacted via telephone by interviewers trained in the use of the Block questionnaire and the use of computer-assisted telephone interviewing. A random digit-dialing procedure was used to gather data from a representative sample of adults in New York State. The sampling was designed to produce unbiased estimates for all New York State by region, sex, socioeconomic status, age, and race/ethnicity. To obtain a sufficient sample of respondents below poverty, it was necessary to sample disproportionately in poverty areas. This oversampling was restricted to New York City.

Results

A total of 1,527 adults ages 18–64 years of age are included in this analysis. Of these, 671 (44%) are male, and 856 (56%) are female. The ethnic composition includes 1,007 whites (66%), 300 blacks (20%), 163 Hispanics (11%), and 43 of other ethnic origin (3%). Forty-seven percentage of the respondents are less than 35 years of age, 32% are 35–49 years of age, and 21% are between 50 and 64.

Table I shows the average intake of selected nutrients for both males and females. Nutrient values from the New York State (NYS) Dietary Survey are compared to the 1987 National Health Interview Survey (NHIS) and the second National Health and Nutrition Examination Survey (NHANES II) conducted from 1976 through 1980. The surveys use probability sampling methods to provide estimates of nutrients that are representative of the non-institutionalized population of the United States. The dietary methodology used in the New York State survey is modeled closely after the 1987 NHIS while a previous-day recall method was employed in NHANES II.

Comparison of the NYS Dietary Survey to the 1987 NHIS indicates male and female adults in New York State are consuming more calories, protein, carbohydrate, fat, and cholesterol daily than adults across the entire country. These differences may be due in part to the inclusion of adults over 65 years of age in the 1987 NHIS. Older adults are likely to consume less food and fewer nutrients overall. The nutrient values for adults in New York State are generally more similar to estimates from NHANES II.

Apart from these differences, Table I clearly shows that adults in New York State, like adults around the country, are consuming total fat, saturated fat, and cholesterol in excess of current recommendations. Expressed as a percentage of total calories, average daily fat intake across all the surveys ranges from 36 to 38.5; the recommended level is 30. Similarly, saturated fat, as the percentage of total calories, ranges from 12.9 to 14.7; the recommended level is 10. The average cholesterol intake for males ranges from 388 to 444 mg/day, exceeding the recommended level of 300 mg/day. The average cholesterol intake for females falls within the recommended level of cholesterol in all three surveys.

The results of the NYS Dietary Survey further indicate differences in fat intake among groups defined by race/ethnicity and socioeconomic status. Table II shows the age-adjusted average daily intake of nutrients by gender and race/ethnicity. For both males and females, there are significant differences among the ethnic groups for fat expressed as a percentage of total calories and for dietary cholesterol. Whites have significantly higher intakes of fat expressed as a percentage of calories than blacks or Hispanics. Cholesterol intake of Hispanics, particularly females, is significantly greater than whites, with the values for blacks intermediate. All ethnic groups showed higher averages for fat and cholesterol than is currently recommended.

TABLE I. Comparison of the Average Daily Intake of Nutrients: NYS Dietary Survey, 1987 NHIS and NHANES II

Average Daily Intake	NYS Dietary Survey	NHIS	NHANES II
Males			
Calories (kcal)	2,357	2,051	2,489
Protein (g)	95	80	98
Carbohydrate (g)	259	216	261
Fat (g)	96	86	103
Fat (% kcal)	36.7	38.0	36.8
Saturated fat (g)	36.1	33.1	37.5
Saturated fat (% kcal)	14.0	14.5	13.7
Cholesterol (mg)	400	388	444
Females			
Calories (kcal)	1,659	1,346	1,531
Protein (g)	69	54	60
Carbohydrate (g)	189	147	174
Fat (g)	68	58	62
Fat (% kcal)	36.2	38.5	36.0
Saturated fat (g)	25.5	21.7	21.9
Saturated fat (% kcal)	14.1	14.7	12.9
Cholesterol (mg)	289	254	275

TABLE II. Average Daily Intake of Nutrients for Three Racial/Ethnic Groups NYS Dietary Survey

Average Daily Intake	White	Black	Hispanic
Males (n)	(476)	(109)	(67)
Fat (g)	97	98	99
Fat (% kcal)*	37.2	35.4	35.3
Saturated fat (g)	37	36	38
Cholesterol (mg)†	388	445	457
Females (n)	(536)	(191)	(97)
Fat (g)	69	66	73
Fat (% kcal)‡	37.1	34.8	34.8
Saturated fat (g)	26	25	28
Cholesterol (mg)‡	269	309	365

*$p < .05$
†$p < .01$
‡$p < .001$

Figures 1 and 2 show the distribution by gender and race/ethnicity of the percentage of total calories derived from fat, categorized into four groups: ≤25%, 25%–30%, 31%–35%, and >35%. As shown in the graphs, a significant proportion of all ethnic groups have intakes of fat that exceed the recommended 30% of calories. The differences among the ethnic groups are not significant for the males ($\chi^2 = 9.8$, $p < 0.13$) but are significant among the females ($\chi^2 = 14.9$, $p < .021$). For females in New York State, the results show that only 17% of whites, 27% of blacks, and 24% of Hispanics are consuming fat within the recommended range of ≤30% of total calories. Similarly, for males in New York State, 13% of whites, 21% of blacks, and 20% of Hispanics are consuming fat within the range of ≤30% of total calories. Depending on the gender or ethnic group, between 50%–65% of individuals surveyed are consuming fat at or in excess of 35% of total calories daily.

Figure 3 shows the differences in the percentage of calories derived from fat by race/ethnicity and socioeconomic level. For this analysis, socioeconomic level was divided into two categories: those families living at or below 185% of poverty and those families living above 185% of poverty. Since the percentage of calories derived from fat did not differ significantly between males and females within ethnic groups, data by gender were combined into three categories: ≤30%, 31%–35%, and >35%. There is no change in the pattern of fat consumption between the two socioeconomic levels for whites, but there is a substantial difference in fat consumption patterns between the blacks living at > 185% of poverty and those living at levels ≤185% of poverty. There is a shift from a diet where over 65% of the individuals consume more than 35% of their cal ories from fat to a diet where only 45% of the individuals consume more than 35% of their calories from fat. The percentage of black individuals who consume ≤30% of their diet from fat almost doubles with the change from lower or higher socioeconomic level. Among Hispanics, there is also a shift toward decreasing the contribution of fat to the diet, with increasing socioeconomic status. However, over 50% of Hispanic individuals in the survey still derive over 35% of their caloric intake from fat.

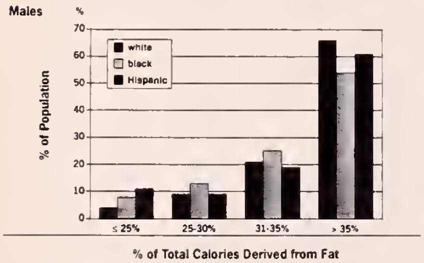

FIGURE 1. Percentage of total calories derived from fat by race/ethnicity and gender.

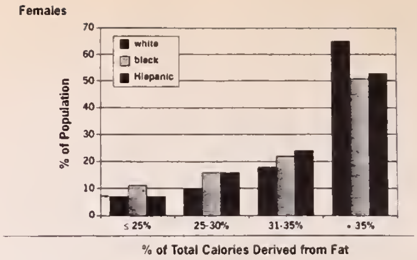

FIGURE 2. Percentage of total calories derived from fat by race/ethnicity and gender.

Conclusions

The results of this analysis show how the consumption of fat and cholesterol among adults in New York State compares with current standards for a healthy diet recommended by the National Cholesterol Education Program and the National Center for Health Statistics. *Healthy People 2000* includes a host of risk–reduction objectives related to dietary patterns and the consumption of nutrients including fat, complex carbohydrates and fiber, calories, calcium, sodium, and iron. Diet-related objectives are included as risk reduction activities in the areas of heart disease and stroke, cancer, and diabetes.

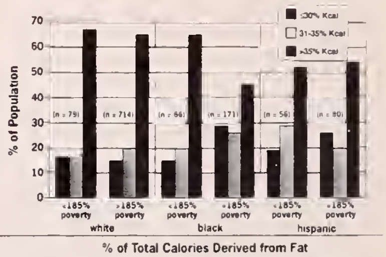

FIGURE 3. Percentage of total calories derived from fat by race/ethnicity and income.

Designing appropriate intervention strategies and monitoring progress toward meeting these national health objectives in New York State requires information on current diet and trends over time. The New York State Dietary Survey provides a baseline assessment of diet among adults and forms the basis for a system of dietary surveillance in the state. Dietary surveillance would monitor statewide trends in food consumption and nutrient intake. Modest revisions in the methodology used in the statewide survey and appropriate sampling strategies will permit examination of diet within small geographic areas and among hard-to-reach population groups defined by racial, ethnic, age, and health characteristics.

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References

1. Lowering blood cholesterol to prevent heart disease. *JAMA* 1985; 253:2080–2086.
2. Shekelle RB, Shryock AM, Paul O, et al: Diet, serum cholesterol, and death from coronary heart disease-the Western Electric Study. *N Engl J Med* 1981; 304:65–70.
3. Lipid Research Clinics Program: The lipid research clinics coronary primary prevention trial results, II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. *JAMA* 1984; 251:365–374.
4. Keys A, Anderson JT, Grande F: Serum Cholesterol response to changes in the diet. IV. Particular saturated fatty acids in the diet. *Metabolism* 1965; 14:776–787.
5. Hegsted DM, McGrandy RB, Myers ML, et al: Quantitative effects of dietary fat on serum cholesterol in man. *Am J Clin Nutr* 1965; 17:281–295.
6. Keys A: Coronary heart disease in seven countries. *Circulation* 1970; 41 (Suppl 1).
7. Department of Health and Human Services, Public Health Service: The Surgeon General's report on nutrition and health. DHHS Pub. No. (PHS) 88–50210. Washington, DC: Government Printing Office, 1988; 83–139.
8. Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. The Expert Panel. *Arch Intern Med* 1988; 148: 36–69.
9. Healthy People 2000: *National Health Promotion and Disease Prevention Objectives.* US Department of Health and Human Services, Public Health Service.
10. Block G, Hartman AM, Dresser CM, et al: A data-based approach to diet questionnaire design and testing. *Am J Epidemiology* 1986; 124:453–469.
11. Block G, Hartman AM, Naughton D: A reduced dietary questionnaire: Development and validation. *Epidemiology* 1990; 1:58–64.

TECHNIQUES IN ARTERIAL SURGERY

Edited by John J. Bergan, MD, and James S.T. Yao, MD. 447 pp, illustrated. Philadelphia, Pa, WB Saunders, 1990. $110.00 (hardcover)

Ten years after the publication of the first edition, Drs Bergan and Yao have collaborated again to co-edit the second edition of a multi-authored text by a distinguished group of American vascular surgeons on techniques in arterial surgery. The stated aim of this edition was "to display standardized surgical procedures accepted as the conventional methods for dealing with occlusions and common aneurysms in the three chief arterial arborizations: the aorta and its branches, the femoral distal circulation and the supra-aortic trunks and their ramifications." The 43 chapters therefore refer not only to conventional surgical procedures but also to less invasive procedures, such as balloon angioplasty, laser angioplasty, and catheter atherectomy.

The style of the book is discursive with varying attempts by different authors to illustrate the details of their operative procedures. This meets with varying degrees of success and in a few instances frank failure, since there is a great variation in the quality and clarity of the illustrative material. The outstandingly successful chapter is that by Dr E. Stanley Crawford whose chapter on "Thoracoabdominal Aortic Aneurysm" is magnificently illustrated in minute detail and with absolute clarity. A close second is the chapter by Dr James C. Stanley and co-authors on "Renal Artery Reconstruction." These two chapters could well serve as models for future editions of similar volumes devoted to surgical techniques and surgical atlases.

As with many texts of this type, there is little room for discussion, debate, or variation in what purports to be a series of "standardized surgical procedures" in a discipline that has defied standardization. The numerous authors and co-authors have by their past activities established credibility in the field of general vascular surgery. There are many other surgeons, however, equally credible, who, although not represented among the authors, can claim that their procedures for similar conditions, although differing substantially from those presented in this work, more closely approach a standard to be achieved.

The book should prove of great help to residents who already have some understanding of the conditions described and the surgical techniques outlined, and can therefore correct for the sometimes inadequate illustrations. The outstanding chapters already mentioned should be of great interest to even accomplished vascular surgeons because of the clarity of detail presented. Medical students would probably be confused rather than enlightened by the texts.

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INFECTIOUS DISEASES OF THE FEMALE GENITAL TRACT

Second Edition. Edited by Richard L. Sweet, MD, and Ronald S. Gibbs, MD. 494 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1990. $68.00 (hardcover)

The purpose of this book is to serve as a resource to clinicians on the subject of infectious diseases to the female genital tract.

Both authors are eminently qualified to present this information. The literature is replete with definitive papers written by them on this topic, and their lectures have further enhanced our knowledge. This book builds on the framework of the previous edition, which was well received. It allows the authors to expand and refine certain areas. They have succinctly presented information that the clinician can take to the bedside.

The book consists of 24 chapters. There is an introduction, which discusses the clinical microbiology, and a chapter on the use of the microbiology laboratory. The authors then proceed to discuss special organisms. Included in this section are new chapters on AIDS, group B streptococcus, hepatitis, and other subjects. There are 12 chapters devoted to gynecologic and obstetric infections. Of particular interest is the new chapter on "Subclinical Infection as a Cause of Premature Labor." A chapter on, "Parasite Disease in Pregnancy" comes in very handy when treating new arrivals from certain parts of the world who suffer from these diseases. The authors conclude with an extensive chapter on antimicrobial agents and antibiotic prophylaxis in obstetrics and gynecology and bacterial endocarditis. In obstetrics and gynecology, they focus on the use of antibiotic prophylaxis on the patient at risk.

The authors present a clear, accurate, generally balanced exposition of the current thinking in infectious diseases of the female genital tract. Their points of view, based on their experience, has not blinded them to other alternatives. This is of particular value to those who wish to pursue the topic in greater depth.

The authors have organized the book in a logical fashion, keeping in mind the physicians for whom it is intended. Their writing style makes for easy reading. They have sought to avoid long, involved dissertations, which tend to lose readers. The text has a profusion of tables. It could, however, have benefited from additional algorithms and pen and ink illustrations. The photographs are adequate. Many of them have been previously seen in color and are less effective in black and white (the cost of color photographs was probably the reason that black and white pictures were used).

The book is a worthy addition to the library of any physician who has to deal with these problems. It compares very favorably with existing books on the subject and is a succinct updated version of an important topic. The book achieves the stated objectives of the authors.

It provides us with a focused review of the relevant infections and a plan as to how they should be identified and treated. Given the speed with which this field is changing one hopes that the authors are busy at work on the third edition. Perhaps, in that edition, they will discuss pelvic tuberculosis in the non-pregnant patient. This infection appears to be on the rise once more.

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THE THYROID GLAND

Edited by Monte A. Greer, MD. 608 pp, illustrated. New York, Raven Press, 1990. $95.00 (hardcover)

Clinicians who purchase this book for casual review and an update on the various clinical syndromes of thyroid disease will find that it exceeds their needs. The editor's objective was to put together a reference that would satisfy the basic scientist and the clinician committed to obtaining detailed information about the thyroid. Although the editor has succeeded in this goal, some would have wished for a second accompanying volume to make the text more encyclopedic.

Chapters begin with a very detailed review of thyroid anatomy and development and a basic science approach to the structure and function of the thyroid cell. However, the biochemistry, synthesis, and secretion of thyroid hormone will only satisfy the researcher. The sections on thyrotropin-releasing hormone, immunity and thyroid function, and the sections on cellular and plasma transport of thyroid hormones are well done. Several chapters on thyroid hormone activity and metabolism follow and will provide only limited interest for the clinician.

The clinician will not find much interest until about halfway through the text when chapters on clinical tests of thyroid function and discussions of thyroid diseases appear. Although the detail in these later chapters is good, the presentation of the material is not conducive to quick review nor is the content as complete as that in previous chapters.

The editor mentions that he assembled a blue-ribbon panel to cover a broad review of the thyroid and allowed some latitude with this focus. The authors are indeed well qualified for their contributions and in some cases bring a unique approach and understanding to their material. However, the style of writing varies from author-to-author, but overall each chapter is well written. The publisher has made some economies in that graphs and tables are often poor and some reproduced photographs and photomicrographs are too dark and definitely substandard.

The editor has assembled a good reference text on the thyroid and certainly succeeds in his objectives. One wishes it could have been expanded to two volumes to make it more encyclopedic and definitive. It is certainly a useful text for medical libraries and for those interested in thyroid research. Those with a more casual interest in the thyroid or clinicians needing a practical reference should seek a different source.

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DIVING MEDICINE

Second Edition. By Alfred A. Bove, MD, and Jefferson C. Davis, MD. 352 pp, illustrated. Philadelphia, WB Saunders, 1990. $50.00 (hardcover)

The popularity of scuba diving has tremendously increased in the past two decades. There now are more than three million certified sport divers in the United States, and about 500,000 more people are newly certified each year. In addition, scuba diving has become an integral part of many occupations. In addition, although the occurrence of serious diving-induced illness or injury is infrequent, the large and growing number of divers has resulted in an increased incidence of diving-related morbidity. Thus, there is a substantial need for medical texts that can assist practitioners to both prevent diving accidents and provide appropriate therapy when they to occur.

Diving Medicine is an updated and significantly expanded edition of the first clinical textbook of diving medicine, which has become a standard reference ever since it was first published in 1976. The book was originally edited by Dr Richard Strauss. This new edition is edited by two of the world's most knowledegable diving medicine clinicians, Drs Alfred Bove and Jefferson Davis. They have continued the practical clinical flavor of the first edition, but regrettably, the premature death of Dr Davis precluded his seeing the volume actually published.

The book's initial chapters provide the physiological background for understanding the effects of the underwater environment on the healthy diver and on the patient with acute or chronic illness, whether they are divers already or wish to become one. The chapter on diving equipment should be especially useful to the non-diving physician, since human underwater physiology is so closely linked to the equipment used underwater.

After the introductory chapters there are a number of chapters dealing specifically with decompression and decompression sickness, barotrauma, gas embolism, inert gas narcosis and other gas toxicities, aseptic necrosis of bone, hypothermia, near drowning, and other diving-related maladies.

New to this second edition are chapters on diving concerns specific to women, the elderly, and the young. These are most welcome additions, since today's diving population is considerably difference from that of the early days of sport diving, when essentially all divers were male, fit, and water-smart. The chapters discussing common pulmonary, neurological, cardiac, and other medical disorders that may interact with the diving equipment are important new additions to this text. These sections, combined with two chapters addressing medical clearance for diving (one each for recreational diving and commercial diving) will be of particular use to clinicians who only occasionally treat patients who are divers.

The chapter on dangerous and toxic marine animals is an admirable new addition, although it is neither quite as current nor as well referenced as the other chapters. It also would benefit from having more photos.

The editors have done a commendable job of recruiting individual authors who are experienced diving physicians or scientists possessing well recognized expertise in diving medicine. The writing is consistent and easy to read, and the text is generally well referenced, and adequately illustrated, although more illustrations might be useful. Although this 25 chapter text concentrates on sport diving, it provides plenty of reliable information that will also make it the standard clinical reference for physicians caring for commercial, scientific, or military divers.

Overall, this new edition of *Diving Medicine* is an excellent clinical text that provides valuable guidance for both the diving medical officer who is involved in the care of sport or commercial divers on a daily basis, and the non-diving physician who sees divers only occasionally as part of a larger patient population.

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TEXTBOOK OF SECRETORY DIARRHEA

Edited by Emanuel Lebenthal, MD, and Michael E. Duffy, PhD. 456 pp, illustrated. New York, Raven Press, 1990. $110.00 (hardcover)

The human gastrointestinal tract is a highly efficient organ capable of transporting large amounts of fluid, electrolytes, and nutrients in order to maintain adequate homeostasis of body fluids, provide for tissue growth and repair and meet bodily energy requirements. Few areas of gastroenterology are as interesting and as rewarding as the study of the derangements of this system that result in diarrhea. This commonplace disorder, defined in part as excessive losses of fluid and electrolytes from the gastrointestinal tract, has a myriad of causes and pathogenic mechanisms. It has been popular in the last decade to attempt to subdivide the pathogenic mechanisms of diarrhea into osmotic, secretory, inflammatory, motility disorders and mixed causes. Secretory diarrhea is that subgroup that results from all conditions in which there is a net increase in intestinal secretion of electrolytes and water.

This multi-authored text attempts to cover the subject of secretory diarrhea comprehensively starting with the physiological basis of intestinal transport and ending with clinical features of various types of secretory diarrhea and therapeutic modalities. It is written to appeal to both the physician and basic scientist.

Any textbook written by more than 50 authors is likely to tax the resourcefulness of editors, and this book is no exception. Chapters range in scope from those dealing with the regulation of electrolyte and water transport to cellular mechanisms of electrolyte transport by enterocytes (including a section on the enterocyte cell membrane) to clinical causes of and therapeutic options for secretory diarrhea. The chapters are generally well written, clearly illustrated, and organized in an orderly fashion. The authors are largely American, university affiliated and active in clinical and basic research.

My main criticism of the book is that the vast amount of material covered is presented in much too diffuse a fashion. In attempting to appeal to a large audience the book consists of 31 individually interesting chapters but the editors have failed to unify them in a "readable" manner. Save for the preface, no overview of the field is given. As a result, this book will serve mainly as a medical library reference work rather than as a true textbook. Clinically inclined pediatricians, gastroenterologists, and endocrinologists will particularly enjoy the chapters on secretory diarrhea in infancy and childhood, bile-acid-induced diarrhea, and neuroendocrine tumors and secretory diarrhea. Surgeons and gastroenterologists will benefit from reading the well-written chapter dealing with ostomy diarrhea.

In spite of the book's shortcomings, I would agree with the editors that it is unique in its thorough and in-depth approach to the problem of secretory diarrhea. Its comprehensive coverage of the topic makes it a mandatory addition to medical libraries, but a textbook it is not.

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THE ICU BOOK

Edited by Paul L. Marino, MD. 713 pp, illustrated. Malvern, PA, Lea & Febiger, 1991. $39.50 (softcover)

The book is a problem-oriented manual for the management of life-threatening organ dysfunction as viewed through the eyes of a practicing critical care physician. It focuses on the principles of care of the critically ill independent of the type of intensive care unit in which the patient is treated.

Arranged according to frequently encountered ICU-based problems, the text offers a basic didactic discussion of care with algorithms for the management of life-threatening abnormalities. It also provides an overview of frequently performed invasive procedures, their indications and contraindications, along with the uses and abuses of routine monitoring devices. There are numerous original tables and charts outlining pragmatic approaches to common pertubations in the critically ill; dosing of ICU medications (eg, vasoactice, inotropic, antiarrhythmic, aerosolized bronchodilators), formulas used in physiologic monitoring, and an overview of mechanical ventilation.

As in any aspect of medical care, there are multiple solutions to similar problems. Although subtleties of care can always be discussed after stabilization of the life-threatening situations, The ICU Book presents a generally reasonable initial approach. As a "generic" text, the author has appropriately excluded such topics as burns, head trauma, and obstetrical emergencies. However, he has, perhaps inappropriately, also excluded such subjects as the technique for pericardiocentesis, the management of drug overdose, and diagnosis and the management gastointestinal hemorrhage.

As a general guide to ICU care the text will be of great usefulness to the house officer and any physician who is inexperienced in the ICU.

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MALES WITH EATING DISORDERS

Edited by Arnold E. Andersen, MD. 288 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $29.95 (hardcover)

Every once in a while the professions need reminding that eating disorders occur in males too. Because eating disorders are so overwhelmingly associated with females, their occurrence in males is often forgotten and overlooked. Andersen's book calls attention to this and does a creditable job of summarizing some of the relevant literature and adding some new contributions.

Nine of the 12 chapters are by authors other than the editor. They are organized into four sections. The first deals with history, sociocultural studies, and psychological functioning; the second with clinical and psychometric studies. The third expounds on treatment and outcome, and the final section is Andersen's integration.

Some of the earliest cases described were males, as noted in the opening chapter by Silverman, which highlights historical clinical vignettes. Anorexia nervosa was always considered rare in males, but even the existence of bulimia nervosa in males has been doubted. An autobiographical case study describes a pattern of bingeing and fasting not altogether unusual in males. Purging, however, the usual hallmark of bulimia nervosa, was absent. The major clinical studies of male anorexia nervosa reviewed are those from St George's Hospital in London and from Toronto General Hospital. They concluded that the illness is similar in the sexes but rare in males. Moreoever, bulimia nervosa in males resembles the illness in females but is exceedingly rare. Studies from Toronto and Johns Hopkins Hospital suggest that a greater proportion of males than females with eating disorders have other psychiatric illnesses. In these studies, males tended either to have a clear-cut eating disorder or to present disordered eating secondary to another psychiatric illness. Many females presented with atypical or mixed eating disorders.

Andersen's chief contributions to the book are the chapters on diagnosis and treatment and the integration. His treatise on treatment presents the multimodal approach he developed at Hopkins. These well-established techniques are as applicable to females as they are to males. The final chapter proposes a new theory of eating disorders, conceptualizing them as motivated behavioral disorders in contradistinction to disorders of mood or thought. It is a thoughtful exposition based on knowledge drawn from long experience. Rather than being an integration of the book, it is a philosophical interpretation of eating disorders in general, and attempts to analyze the evolution of clinical phenomena logically. It could stand alone or be an appropriate contribution to any volume about eating disorders. The theory is not gender specific. Other than for a few asides about males, Andersen does not explicitly address male issues regarding eating disorders.

Andersen's style is lucid and informative. The book serves the purpose of reminding us that eating disorders do occur in men and that the diagnosis should be considered in general medical and psychiatric practice. It does nothing to dispel the notion that eating disorders are chiefly diseases of women, and still leaves largely unanswered the question of why they are so rare in men. Perhaps we know little more than Hippocrates, to whom Andersen attributes the quotation: "It is more difficult for boys to withstand fasting."

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NATURAL MEDICINE FOR CHILDREN

Edited by Julian Scott, PhD. 191 pp, illustrated. New York, William Morrow & Company, Inc, 1990. $24.95 (hardcover)

The 1980s brought a new emphasis to natural medicine. The holistic approach has been incorporated into many medical practices and has generally received positive responses from patients. As we enter the 1990s, natural approaches to medical problems continue to receive attention in both the lay and medical press. *Natural Medicine for Children* is another example of this genre.

This volume claims to be a practical comprehensive guide to herbs, homeopathy, massage and other alternative remedies to common pediatric medical complaints. Dr Scott, an experienced practitioner of natural medicine and acupuncture, extensively studied in China, and this Eastern influence prominently comes across in this book.

An encyclopedia approach has been chosen by the author, who has divided the book into three main sections. Part I concerns itself with developing health. Here the author discusses breastfeeding, immunizations, and childhood disease patterns. In Part II, natural therapies are discussed. This section goes into great detail about herbal medicines, homeopathy, and massage therapy. In addition, this section discusses the Bach remedies, founded and named after Dr Edward Bach. The philosophy underlying the Bach remedies is a positive attitude toward health. The choice remedy is made from the child's mental and emotional state, rather than from the physical symptoms of illness. Although most of us trained in conventional medicine will agree that the well-being of our patients requires a combination of mind and body, the full impact of the Bach remedies will be alien to us.

The book concludes with an overview of treating childhood ailments. Sections on respiratory, digestive, dermatological, and common childhood diseases are included. All sections provide an herbal treatment, as well as brief comments on "orthodox" treatments. There are strong arguments made regarding the treatment of such diseases as measles, mumps, rubella, and varicella. The natural medicine proponents view these illnesses as marking a transition in a child's life. As the child grows up spiritually, emotionally and mentally, there is a corresponding reaction in the physical body which involves the "throwing out" of accumulated poisons and the appearance of a skin rash. This is not a concept based on scientific evidence, and it will not be taken seriously by practitioners of conventional medicine.

Natural Medicine for Children achieves its stated goal of providing a practical guide to the treatment of children with natural and drug free remedies. The primary concepts of good nutrition, exercise, and strong family relationships are good ones that are well accepted and integrated into conventional medicine. The book's greatest appeal will probably be to the lay person, or it will serve perhaps as an interesting encyclopedic guide to those practitioners who may rely on herbal remedies. However, I seriously doubt this book will ever play a significant role in changing the way most pediatricians and family physicians practice medicine.

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PULMONARY MANIFESTATIONS OF SYSTEMIC DISEASE: A CLINICAL APPROACH

Edited by Kevin R. Cooper, MD. 240 pp, illustrated. Mount Kisco, NY, Futura Publishing Company, 1990. $48.00 (hardcover)

The theme of this book is systemic diseases as manifested through their effects on the lung. The book is not intended to be an exhaustive review of the entire topic, but rather a focused coverage of clinical entities more commonly encountered in practice. Nine of the ten contributors are faculty members of the Medical College of Virginia working in internal, pulmonary, and critical care medicine. The book provides a timely and useful update of the topic. The most useful part of it is the summary tables found in most of the chapters. These tables are clear and concise and provide a quick reference guide for the major points in the chapters. The chapters themselves contain many useful points of information that will be of interest to readers at all levels from student to pulmonologist. Particularly useful is the book's coverage of disease entities that do not always initially alter the chest roentgenogram, but may impair pulmonary function and arterial blood gases.

The book starts with an excellent chapter on pulmonary infiltrates in the immunocompromised host. The chronologic focus on the presentation of infiltrates is a useful tool for readers who are accustomed to long lists of differential diagnoses but little direction in prioritization. The chapter on neurologic diseases presents an excellent summation of the effects on pulmonary function of lesions at specific levels in the spinal cord. The interactions of lung with cardiac, gastrointestinal and hepatic disorders, renal diseases, connective tissue diseases, endocrine and metabolic disorders, malignancy, obesity, and drug-induced pulmonary changes are additional major topics covered. Included in these are discussions on fluid and gas movement across the diaphragm, which make for interesting reading. The discussions on the mechanisms of catamenial pneumothorax and the effects of endometriosis on the lung cover topics that remain enigmatic. Pleural effusions are covered under the individual organ systems associated with effusions.

The quality of the material covered is good. It is timely, and in a limited number of pages, packs a large amount of factual material that will be useful to the reader. The facts presented are accurate. Because of the brevity of the book (240 pages), at times, clarity is sacrificed, in the sense that the facts are given but a detailed explanation of the facts is not. This is not necessarily a defect, as the book appears to be designed more for the presentation of factual material rather than for theoretical discussion. The chapters in the book are well organized, and this is due to the effective use of tables. Certain diseases with pulmonary effects (sarcoidosis, neurofibromatosis, thymoma, hematologic diseases) have not been included, and this may limit the utility of the book. The discussion of pleural effusions is dispersed through various chapters and might have been better organized as a single chapter. The book compares well with books previously covering this topic. It includes useful information dealing with cellular and functional changes affecting the lung, whereas earlier work has frequently focused on radiologic changes. The book achieves the author's stated objectives. This makes it a volume that is easily read and from which many important factual points can be derived by the general reader. It does not cover certain topics and is not all encompassing in the topics discussed, and this may limit its usefulness for the reader who is looking for more detailed information.

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MANAGING ASTHMA

Edited by Miles Weinberger, MD. 330 pp, illustrated. Baltimore, Williams & Wilkins, 1990 (hardcover)

A number of studies have shown asthma to be increasing in incidence, with more associated deaths. To reduce morbidity and mortality from asthma, a systematic and integrative treatment plan is needed, including a component of patient self-care.

Managing Asthma, essentially an operations manual, provides just such an approach. The book functions much as a *Washington Manual* (the standard internal medicine resident's handbook). The book chiefly deals with the delivery of care to the asthmatic patient and not with theory. For readers who want an in depth discussion of specialized subjects, the author provides a bibliography.

Dr Weinberger is Professor of Pediatrics and Director of the Pediatric Allergy and Pulmonary Division at the University of Iowa. In his 14 years there, he has also led a large statewide home management program for asthmatics. Believing that the successful elements of that program can be transplanted to other care settings, Dr Weinberger's book prescribes the formula. The author's earlier publications include more than 100 articles and 25 book chapters on asthma.

The first section provides the reader with a summary of the pathophysiology of asthma. The second section deals with the diagnosis of asthma and the classification of asthmatics according to clinical severity over a period of a year. This schema is more useful to a clinician than concepts such as extrinsic vs intrinsic asthma. The third section extensively deals with treatment. Of practical significance are the chapters on pregnancy and asthma, residential treatment of asthmatics, and telephone advice.

The author's philosophy is that education of the patient should be a top priority of all treating physicians. Recent articles have suggested that high rates of morbidity and mortality caused by asthma occur before patients reach the hospital. Successful treatment involves the education of patients and family members in prophylactic therapy, recognition of asthma's severity, and initiation of early curative therapy in acute attacks.

To this end, the book contains approximately 90 pages of patient education materials in the appendices. The appendices also provide the clinician with specifics on how to perform diagnostic testing. For example, allergy testing and its current indications are discussed in detail. In addition, checklists and standard forms are included for physician use and for patient self-monitoring. Author and publisher grant the reader permission to copy all the forms and checklists and to copy and distribute all the patient education materials in the extensive appendices.

Managing Asthma does not discuss any conflicting views about treatment. The author states that he is offering his own views and does not aim to duplicate discussions of controversies present in the literature. Key words and important details are heralded in bold-face type throughout the text. There are many algorithms that are simple to follow for both patient and physician. My only criticism, a minor one, is that the discussion on theophylline kinetics tends to be obscure and should be condensed in keeping with the rest of the book's style.

In addition, the reader should take note that because specific pharmaceutical therapies are recommended (such as the prescription of a specific inhaled steroid), the book may become dated quickly as new and better pharmaceutical products come on the market.

The value of this book is threefold: as a well-organized technical manual to be consulted over and over again, for the unusual feature of the appendices that can be copied for use and for the author's expertise in patient self-management with physician direction. No similar recent manuals on the practical management of asthma are available. I would recommend this book to all physicians treating asthmatics.

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NEONATAL SEIZURES

Edited by Claude G. Wasterlain, MD, and Paul Vert, MD. 336 pp, illustrated. New York, NY, Raven Press, 1990. $98.00 (hardcover)

In this volume, Drs Wasterlain and Vert summarize the proceedings of the International Colloquium on Neonatal Seizures, held in France in 1987. The papers delivered at that conference covered the most important developments in both the basic science and clinical aspects of neonatal seizures.

In a very compact volume the editors bring together many important topics that give the neonatologist, pediatrician, neurologist, and research scientist an excellent overview of this highly specialized field. Major sections include clinical and electroencephalographic (EEG) diagnosis, mechanisms of brain damage, cerebral blood flow and metabolism, ontogenesis of receptors and neurotransmitters, and pharmacology and therapeutics.

The clinical and EEG diagnosis sections carry chapters, which lucidly detail the major aspects of the clinical and EEG findings and the correlation with underlying pathology. A separate chapter reviews benign neonatal seizures. The clearly presented graphs and tables significantly enhance the text.

The middle section of the book contains papers discussing the effects of prematurity, various models of brain insults, and the effects of seizures, hypoxia, and disruption of autoregulation on the brain. Other chapters cover cerebral blood flow, autoregulation, glucose utilization, and receptor development. The volume concludes with a clinical section on the treatment of neonatal seizures, special pharmacological issues in the newborn, and a review of the potential effects of phenobarbital on the developing nervous system.

Some of these may be difficult for the clinician who is not familiar with the underlying research. Yet, even reading these chapters selectively will increase one's understanding of the multiple issues that bearing on neonatal seizures.

Although the book covers many important issues well and can be highly recommended, the recommended price will deter many purchasers who do not have a very specific interest in this area.

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ASSESSING PEDIATRIC PRACTICE: A CRITICAL STUDY

By Raymond S. Duff, MD, edited by Charles D. Cook, MD. 237 pp. Ann Arbor, Michigan, Health Administration Press, 1990. $27.00 (hardcover)

The author of this volume comprehensively documents and assesses the changing pattern of the care of children and the emergence of pediatrics as a primary care specialty. Shortly after ophthalmology became the first specialty to introduce board certification, pediatrics and the other primary care specialties followed. This era was preceded by a time when family practitioners cared for children throughout the country, and most of the time did "routine" surgical procedures, as well.

The preeminent role of the pediatrician began to emerge before World War II, but really came to flourish during the post-war "baby boom" era. With the introduction of various diagnostic and therapeutic advances during these years, family practitioners began to transfer the care of children to pediatricians.

The author presents the contrasting roles of pediatricians in various practice settings, and in so doing, discuss the advantages and problems associated with each. There is also a discussion of the relationship of children and parents toward different physicians in a given partnership or group. Attention is given to the emergence of pediatric sub-specialty areas and the role of technological advances in contributing to their development. The emergence of sub-specialty areas has to some extent changed the role of the general pediatrician. In spite of subspecialty development, general pediatricians and family practice physicians still provide care to the majority of children in the country. In addition to dealing with a spectrum of social and psychological problems, they must also treat substance abuse and acquired immunodeficiency syndrome.

The text does not address the care of children born and cared for in small communities. Pediatricians in these smaller communities have a greater opportunity to understand their patients and their parents in a more congenial and satisfying way. On the other hand, these children and their pediatricians often do not have access to tertiary care centers.

In summary, this book should be read by residents and young pediatricians who are about to or have just entered practice. In so doing, they will be better able to understand the problems that pediatricians encounter.

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SWIMMING UPSTREAM: TEACHING AND LEARNING PSYCHOTHERAPY IN A BIOLOGICAL ERA

Edited by Jerry M. Lewis, MD. 208 pp, illustrated. New York, Brunner/Mazel Publishers, 1990. $25.00 (hardcover)

The intriguing title of *Swimming Upstream: Teaching and Learning Psychotherapy in a Biological Era* foreshadows a fascinating, superbly written and well-researched book, that persuasively demonstrates that even in an age of scientific reductionism, the facilitation of the patient's narrative is, in and of itself, a significant aspect of the therapeutic healing process.

Dr Jerry M. Lewis, the former Psychiatrist-in-Chief of Timberlawn Psychiatric Hospital in Dallas and President of the Southern Psychiatric Association, shares central aspects of his professional and personal life in creating a book that will be beneficial to those who are exploring the profession, as well as veteran practitioners of psychotherapy. Furthermore, all physicians might also benefit from understanding the dynamics of any physician-patient interaction in the terms described by Dr Lewis.

Lewis is clearly concerned about the future of the art of psychotherapy. He notes that the current "reductionism of the neurosciences," with its biological model of psychopathology, dismisses or gives little attention to the role of psychological and social variables. Some believe that they have seen the "promised land . . . and its terrain is biological reductionism." Similarly, Lewis is troubled by the fact that external criteria, such as reimbursement policies, increasingly determine the patterns of psychiatric practice.

Lewis suggests that the beginning of psychiatric training is crucial. "How trainees are introduced to the processes of psychotherapy may influence all of their subsequent professional development." He therefore proposes creating a model training curriculum that would integrate the latest biological findings with an understanding of equally significant psychological and social variables. Furthermore, he recognizes that successful training must initially focus on psychotherapeutic skills, as well as theories.

At the core of Lewis' understanding of psychopathology in his view of causality. Many people think of causality in a linear fashion. In contrast to this approach, Lewis presents a view of complex causality—a biopsychosocial model. In this model, each perspective is complementary, rather than oppositional. The integration of many variables is seen to form patterns of causality. The incorporation of this model means that there are no simple solutions to the issues that continue to challenge patient and therapist alike. One will be left with some areas of uncertainty, doubt, and ambiguity. It is important to recognize and acknowledge this lack of certainty.

Lewis emphasizes that psychotherapy is an art. Training requires not only clinical supervision and personal analysis, but also systematic instruction in the process of psychotherapy. Lewis is consistently open and honest as he presents theory, opinion, and personal experience. In reviewing his own training and maturation as a therapist, he analyzes three changes that occurred in his own work.

First, he became particularly aware of the role of affect within the therapeutic hour—that of the patient, that of the therapist, and that which is created during the session. Second, he moved away from directive approaches to a more collaborative orientation. Third, he began to pay much greater attention to the here-and-now of the therapeutic session.

From Lewis's perspective, the essential role of the psychotherapist is to facilitate the narrative flow of the patient and to hear that patient's particular story. The therapist can view each session as a novel adventure, marked by curiosity and excitement as the uniqueness of each patient's experiences continues to evolve.

An important understanding of the therapeutic session is based on a knowledge of relationship structures, such as those in marital and family systems. Different systems present various patterns of interaction, based on such issues as power and distance. It is particularly instructive and exciting to be able to share with a patient that his or her current conflictual relationships may exhibit structures similar to those encountered in childhood or those exhibited even in the relationship to the therapist.

Lewis stresses repeatedly that the therapist's mandate is to facilitate the patient's narrative. One must enter the patient's story to develop a sense of what life is like for him or her. Furthermore, the therapist must recognize that we all behave in ways that may lead to the fulfillment of our inner hypotheses.

Both understanding and experiencing the patient's narrative are important. Lewis suggests the importance of achieving "affective empathy," by which one develops sensitivity to the patient's affective messages, as well as appropriate responses. Common fears of empathic closeness usually concern issues of self-esteem, such as "If you understand my inner experience that well, will you value me?" Lewis challenges therapists to develop their eyes and ears effectively for dyadic interactions. Ultimately, the therapist must develop heart. The interviewer contributes to the affect of the session as much as the patient. He or she must not collude with the patient to keep the interview free of affect.

Lewis understands the therapist's most fundamental task as "to encourage storytelling, to stay out of the patient's narrative, and not to interrogate the patient. It is an act of faith that the patient will tell you what you need to know, faith in the act of listening without relying on questions." Interviews dominated by the interviewer's questions create a dominant-submissive relationship, in which the asker controls the situation. To facilitate the flow of the patient's narrative, Lewis suggests that the interviewer have a genuine interest in the patient's story and respond affectively, with an emphasis on openness and honesty.

Although the focus of Western consciousness is on the individual, the therapist needs to understand the structures of patient relationships and the distribution of power within the system. In interviewing a couple or family, the interviewer can observe what feelings are expressed and who expresses which feelings. The interviewer's own intentions and assumptions are not necessarily verbalized. ". . .The emotional tone used in asking the question can be the decisive indicator of what type of question is being asked." Marital and family perspectives on psychopathology are complementary to biologic and intrapsychic perspectives.

Lewis demonstrates his own skill in interviewing prospective residents by relating the two questions that he always asks each applicant: "How does it feel to be you?" and "How do you relate what it feels like to be you to the family you grew up in?" Lewis observes that whenever he and his colleagues have selected a resident who does not do well in their program, "it often has involved being dazzled by an applicant's brilliance and intellectualism, somehow overlooking his or her relationship deficits."

The mature psychotherapist must learn the distinction between learning and becoming. As the therapist matures, he or she becomes more respectful of another's subjective realities. In essence, "a psychotherapist comes to be, over time, that which he or she does."

Psychotherapy should be seen as a delicate art of exploration. As previously mentioned, the heart and soul of this method is the facilitation of the patient's narrative. Are these narratives historical truth or narrative truth? Ultimately, this book is about the nature of reality. "Is there a real, 'out there,' discoverable reality existing independently from the observer's perceptions . . .?" To Dr Lewis's emphasis on the patient's narrative, I would add only that I would have liked to see him incorporate a broader discussion of one's psychic reality. For example, Dr James Hillman, a prominent Jungian analyst, proposes that one must be aware of the importance of images in understanding that "the nature of the mind is poetic" (Hillman, *Healing Fiction*, 1983). Furthermore, the art of healing is, as Dr Lewis clearly understands, the ability to transform the patient's narrative into healing images.

Lewis's thoughtful assessment of a biopsychosocial approach is quite convincing, as is his understanding of those qualities and training modalities that will create the most effective therapist. He distinguishes between those who regard the profession as a career and those who view it as a calling. "Although the idea of career involves greater investment of self than does working in order to make a living, a calling both carries more of the individual's identity and incorporates the idea of the common good."

I must include one final, personal note. As a practicing psychotherapist for 13 years and rabbi for 20 years, I wish that I had read this book at the beginning of my career. However, even now I have benefited from reading this work. Lewis's openness in discussing his own personal and professional development endeared him to me. His slim book reviews and explains some of the concepts and methodologies that are central to the therapeutic process.

His sense of adventure, enthusiasm, and excitement pervades this text and reinforces my own feelings about "the pleasure of doing something that feels useful."

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THE DRUNKEN SOCIETY: ALCOHOL ABUSE AND ALCOHOLISM IN THE SOVIET UNION

Edited by Boris M. Segal, MD. 618 pp. New York, Hippocrene Books, 1990. $40.00 (hardcover)

Dr Boris Segal's book is a multidisciplinary study of the problem of alcoholism in the USSR. The book's purpose is to characterize sources and patterns of alcoholism in the Soviet Union, seen not as an isolated medical problem, but as a complex phenomenon embracing various cultural, psychological, economical, and social factors determined by the socio-political system. In his preface, the author appropriately notes that a better title for the book might be *Russian Culture, Society, and Alcohol.*

From 1954 to 1973, Dr Segal led various clinical and research departments in psychiatric and psychotherapeutic institutes in Moscow. He is the author of more than 150 works on psychiatry, psycho-history, psychosocial and cross-cultural issues, and alcoholism. In this book, he proves to be a highly qualified researcher and scientist. In 1967, while still in the Soviet Union, he tried to publish a similar work titled *Alcoholism*; however, because of heavy censorship only strict medical data remained in the printed publication. In 1973, he emigrated to the USA and continued his research at Harvard University.

The new book is a result of long-term scientific research. Apart from his own observations, the author uses data from interviews with various experts, including Soviet government officials, economists, sociologists, lawyers, and physicians; materials published in Soviet press; and data from official American documents and works by his American colleagues related to the problem.

The book will be of interest not only to medical professionals, but also a wide range of researchers in adjoining fields, such as sociologists, historians, economists and politologists. The 12 chapters of the book, each focusing on an important aspect of the problem (eg, Soviet drinking behavior; sex, marriage, and alcohol; alcohol and crime; alcohol-related morbidity and mortality; economic losses due to alcohol abuse) go far beyond the limits of a strictly scientific purpose. The reader is introduced to major events of modern Soviet history, to the life styles of various social groups, and to numerous ethnic minorities. Each chapter includes a comparative study of the problem in the USSR and the USA, and chapter 12 is dedicated exclusively to a comparative analysis of causes and implications of Soviet and American alcoholism. The author's points are in each case supported by rich statistical data.

The thorough character of the research is reflected even in the bibliography, which includes over 1,300 titles by both Soviet and Western workers concerned with different aspects of and approaches to the problem.

The book as a whole is a valuable addition to numerous publications on major problem of substance abuse both in the USSR and the US and is a welcome key to understanding certain aspects of today's situation in the USSR.

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CONTINUING CARE: THE MANAGEMENT OF CHRONIC DISEASE

Second Edition. Edited by John Hasler, OBE, and Theo Schofield, MD. 349 pp, illustrated. New York, Oxford University Press, 1990. $42.50 (hardcover)

The authors are British practitioners who write about the care of patients with chronic disease. The care which these patients require is a challenge to physicians on both sides of the Atlantic, and this book provides insights that would benefit anyone who provides primary care. It is written in the setting of the National Health Service and therefore speaks to many of the organizational hurdles faced by our colleagues in the United Kingdom which do not pertain elsewhere. The bulk of the text, however, is focused on patient care, the essence of which is basically the same no matter where the examination room is located.

The book is organized into two sections. The first addresses the broader issues of caring for chronically ill patients, including the size and nature of the problem, psychologic and sociologic implications, as well as organizational factors. The principles outlined in the first section are applied in the second section to the specific chronic diseases, which are common to daily medical practice (ie, asthma, diabetes, hypertension). Each chapter reviews some basic background information and then makes specific recommendations about the ongoing care of the disease process being discussed.

The success that the authors achieve is variable. The discussion of the scope of the problem is excellent, and anyone who shares the problems, frustrations, and occasional joys of caring for chronically ill patients will quickly identify with the major points. The ensuing discussions, however, can become a bit plodding as one tries to get past theory to the specifics of practice.

The second section also achieves mixed success. Some problems are not addressed in enough detail to achieve their goals. The chapter on cardiovascular disease, for instance, attempts to review the basic pathology, clinical presentation, prognosis, and management of coronary artery disease, congestive heart failure, cor pulmonale, valvular heart disease, and dysrhythmias. This is obviously a Herculean task, which seems impossible to accomplish within the constraints of a single chapter in a work such as this. The result seems superficial.

Other chapters accomplish their goal to greater degree. The discussion of asthma is succinct and to the point. It offers several practical, concrete guidelines for care that can be applied in a variety of practice settings.

Although the authors' success varies from chapter to chapter, the overall work is quite worthwhile reading and is highly recommended to anyone who cares for chronically ill patients. There is a definite void in the literature and in the training of those entering primary care on how to take care of these challenging patients. It is easy to see why, since these long-term problems do not lend themselves to short-term research projects and grants, the authors deserve some applause for taking on the problem.

This volume may also be especially helpful to anyone who is interested in establishing practice guidelines for quality assurance within their own practices. The discussions offer a framework, which could be adapted to many situations and which could be the starting point from which individualized guidelines could be constructed.

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THE AMBULANCE: THE STORY OF EMERGENCY TRANSPORTATION OF SICK AND WOUNDED THROUGH THE CENTURIES

By Katherine Traver Barkley. 207 pp, illustrated. Kiamesha Lake, NY, Load N Go Press, 1990. $13.95 (softcover)

In the spring of 1989, Myron Gittell, of Load N Go Press, was researching a proposal for an ambulance exhibit at a New York museum and came across *The Ambulance: The Story of Emergency Transportation of Sick and Wounded Through the Centuries*, by Katherine Traver Barkley. The book had been out of print for over ten years, but Gittell, himself an emergency medical technician and admitted "ambulance enthusiast," wanted to bring it back, feeling Barkley's work was "the only one ever published in this country exclusively devoted to the history of the ambulance." Fortunately, for those of us interested in medicine and its history, Gittell managed to find Barkley and reached an agreement to republish the book.

Katherine Barkley, a retired medical librarian, has written a well-documented and superbly illustrated history of the ambulance, from centuries past to the present. The first modes of transportation, according to the author, were probably simply two friends, carrying the patient in whatever way was convenient to the transporters. The use of the stretcher soon followed, involving two poles and a hammock, supported also by the strength of the patient's comrades.

Then came the wagon, the true predecessor of today's ambulance. Perhaps the first such conveyance was the Anglo-Saxon hammock-wagon, described by Barkley as "braked by attendants who held snubbing chains around the wheels to keep the wagon from careening downhill . . . the person riding the hammock having a rough ride swinging back and forth."

The author then describes the evolution of this primitive type of wagon to the present. As with much of medical history, medical invention grew out of military necessity, and the evolution of medical transport of the wounded is a prime example. Barkley draws upon *A Treatise on the Transport of Sick and Wounded Troops*, published in 1869 by T. Longmore, Honorary Surgeon to Her Majesty Queen Victoria. Longmore's book was the first critique of the management of wounded soldiers and included descriptions and illustrations of conveyances used that Barkley has reproduced in *The Ambulance*.

For example, General Smith's sick-transport wagon, weighing 2,200 pounds when empty, was difficult to handle on bad roads and harder to pull, even with a team of strong horses. The author also describes the use of animals in transport, including litters attached to pack saddles on a mule, and "the dhooley" (another type of litter) in which patient and pack were strapped to a camel's back.

According to Barkley, either Bellevue Hospital in New York City or the commercial Hospital of Cincinnati, may have had the first hospital ambulance service. Regardless of which hospital really came first, several historical points are interesting. The ambulance medical inventory, for example, included "a quart of brandy, two tourniquets, six bandages, six small sponges, splint material, blankets, and a two ounce vial of persulphate of iron." Drawn by horses over city streets, the vehicles could attain the rather undazzling speed of one mile in five minutes. In contrast to the familiar wailing sirens of today, the public were warned of an oncoming ambulance by "the driver and surgeon shouting at people to get out of the way."

The first automobile ambulances came in 1899, which at full speed could attain 16 miles per hour, and were powered by electricity, not gasoline. Barkley credits the Hess and Eisenhardt Company of Cincinnati, Ohio, as the world's oldest builder of the gasoline-powered ambulance (and hearses as well!). Their 1923 ambulance boasted airsprings over the bumper for smoother riding, a comforting replacement to the typically jarring journey to the hospital.

Barkley also describes the "Medicopter" used in Vietnam and Korea, as well as the "Heartmobile," created to implement current standards of immediate, on-site coronary care. Unfortunately, Gittell chose to reproduce the book unedited, exactly as it was when it first came out over ten years ago, and so the book is not at all up-to-date. Indeed, there are pictures labeled as the "Ambulance of the Future" which look just like the ambulance in use today. *The Ambulance* also cites a 1970s report of the National Academy of Sciences, recommending the establishment of 911 as an emergency telephone number nationwide. Obviously, 911 has gone a long way since then and is deserving of current evaluation, but because of Gittell's decision, such documentation is completely absent from the book.

Even with the shortcomings, *The Ambulance* stands as an interesting book about the history of the emergency transport of the sick and wounded. It should be appealing to a wide audience, emergency care providers, and students of medical history alike.

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BOOKS RECEIVED

AIDS

AIDS Prevention and Treatment: Hope, Humor, and Healing. Edited by M. Ross Seligson, PhD, and Karen E. Peterson, PhD. 276 pp. Hemisphere Publishing Corp, 1992. $29.95 (softcover)

CARDIOLOGY

Progress in Cardiology. Edited by Douglas P. Zipes, MD, and Derek J. Rowlands, MD. 223 pp, illustrated. Malvern, Pa, Lea & Febiger, 1991. $45.00 (softcover)

DERMATOLOGY

Handbook of Skin Clues of Systemic Diseases. Paul H. Jacobs, MD, and Todd S Anhalt, MD. Second Edition. 150 pp. Malvern, Pa, Lea & Febiger, 1992. $15.95 (softcover)

GERIATRICS

Granny Flats as Housing for the Elderly. Edited by N. Michael Lazarowich. 74 pp. Binghamton, NY, Haworth Press, 1991. $19.95 (hardcover)

Drug-Related Problems in Geriatric Nursing Home Patients. Edited by James W. Cooper, PHARMPhD, FASCP. 148 pp. Binghamton, NY, Haworth Press, Inc, 1991. $10.95 (softcover)

Evaluation and Treatment of the Psychogeriatric Patient. Edited by Diane Gibson, MS, OTRL. 111 pp. Binghamton, NY, Haworth Press, 1990. $22.95 (hardcover)

Activities with Developmentally Disabled Elderly and Older Adults. Edited by M. Jean Keller. 156 pp. Binghamton, NY, Haworth Press, 1991. $12.95 (softcover)

HEALTH CARE ADMINISTRATION

Hospitals in Transition. The Resource Management Experiment. By T. Packwood, J. Keen, and M. Buxton. 193 pp. Bristol, Pa, Open University Press, 1991. $24.95 (softcover)

Paying the Doctor. Health Policy and Physician Reimbursement. Edited by Jonathan D. Moreno, PhD. 194 pp. Westport, CT, Greenwood Press, Inc, 1991. $39.95 (hardcover)

HISTORY OF MEDICINE

The History of Yellow Fever. By Francois Delaporte. 181 pp. Cambridge, Mass, The MIT Press, 1991. $22.50 (hardcover)

Medical Malpractice in Nineteenth-Century America. Origins and Legacy. By Kenneth Allen DeVille. 319 pp, illustrated. New York University Press, 1990. $34.95 (hardcover)

INTERNAL MEDICINE

Harrison's Principles of Internal Medicine. Edited by Jean D. Wilson, MD; Eugene Braunwald, AB, MD; Kurt J. Isselbacher, AB, MD; Robert G. Petersdorf, AB, MD; Joseph B. Martin, MD, PhD; Anthony S. Fauci, MD; Richard K. Root, MD. 2,397 pp, illustrated. Summit, Pa, McGraw Hill Inc, 1991. $95.00 (hardcover)

Contemporary Internal Medicine. Clinical Case Studies, Volume 3. Edited by Juan M. Bowen, MD, and Ernest L. Mazzaferri, MD. 286 pp, illustrated. New York, Plenum Publishing Corp, 1991. $75.00 (hardcover)

MISCELLANEOUS

The Best of Medical Humor. A Collection of Articles, Essays, Poetry, and Letters Published in the Medical Literature. Edited by Howard J. Bennett, MD. 228 pp, illustrated. Philadelphia, Pa, Hanley & Belfus 1991. $25.00 (hardcover)

Resources for People with Disabilities and Chronic Conditions. 215 pp. Lexington, Mass, Resources for Rehabilitation, 1991. $44.95 plus $5.00 for shipping and handling. (softcover)

The Chaplain-Physician Relationship. Edited by Larry VandeCreek, DMIN, and Laurel Arthur Burton, TPHD. 75 pp, Binghamton, NY, Haworth Press, 1991. $12.95 (hardcover)

Activities in Action. Proceedings of the National Association of Activity Professionals. 1990 Conference. 98 pp. Binghamton, NY, Haworth Press, 1991. $19.95 (hardcover)

Alternatives to Infertility: Is Surrogacy the Answer? By Lita Linzer Schwartz, PhD. 207 pp. New York, Brunner/Mazel Publishers, 1991. $25.95 (hardcover)

Defeating Pain. The War Against a Silent Epidemic. By Patrick D. Wall, MD, and Mervyn Jones. 285 pp. New York, Plenum Publishing Corp, 1991. $24.95 (hardcover)

Hippocrates' Handmaidens: Women Married to Physicians. By Esther M. Nitzberg. 401 pp. Binghamton, NY, Haworth Press, Inc, 1991. $24.95 (softcover)

The Case of Preconception Care of Men and Women. By Margaret Wynn and Arthur Wynn. 169 pp. Bicester, Oxen, England, AB Academic Publishers, 1991. $59.00 (softcover)

Muscular Dystrophy and Other Neuromuscular Diseases. Edited by Leon I. Charash, Robert E. Lovelace, Claire F. Leach, Austin H. Kutscher, Rabbi Jacob Goldberg, David Price Royce, Jr. 249 pp. Binghamton, NY, Haworth Press, 1991. $34.95 (hardcover)

Families, Intergenerational and Generational Connections. Edited by Susan P. Pfeifer, PhD, and B. Sussman, PhD. 389 pp. Binghamton, NY, Haworth Press, 1991. $45.00 (hardcover)

Final Exit. The Practicalities of Self-Deliverance and Assisted Suicide for the Dying. By Derek Humphry. 192 pp. New York, Carol Publishing Group, $16.95 (hardcover)

NEUROSURGERY

Microsurgery of the Cervical Spine. By Paul H. Young, MD. 196 pp, illustrated. New York, Raven Press, 1991. $110.00 (hardcover)

OBSTETRICS AND GYNECOLOGY

Obstetrics and Gynecology Review 1991. By Harrison H. Sheld. 435 pp. Philadelphia, Pergamon Press, 1991. $34.95 (softcover)

ONCOLOGY

Malignant Mesothelioma. Edited by Douglas W. Henderson, Keith B. Shilkin, Suzanne Le P. Langlois, and Darrel Whitaker. 383 pp, illustrated. Bristol, Pa, Hemisphere Publishing Corp, 1992. $99.50 (hardcover)

PSYCHIATRY

Psychotherapy Training. Contextual and Developmental Influences in Settings, Stages, and Mind Set. Edited by Thomas H. Peake, PhD, and J.D. Ball, PhD. 204 pp. Binghamton, NY, Haworth Press, Inc, 1991. $24.95 (hardcover)

Pathways to Reality. Erickson-Inspired Treatment Approaches to Chemical Dependency. By John D. Lovern, PhD. 228. New York, Brunner/Mazel Publishers, 1991. $26.95 (hardcover)

Families as Nurturing Systems: Support Across the Life Span. Edited by Donald G. Unger and Douglas R. Powell. 251 pp. Binghamton, NY, Haworth Press, 1991. $29.95 (hardcover)

Commonalities in Psychosomatic Medicine. By Harold Geist, PhD. 119 pp. St Louis, Mo, Warren H. Green, 1991. $17.95 (softcover)

The Psychoanalytic Vocation: Rank, Winnicott, and the Legacy of Freud. By Peter L. Rudnytsky. 220 pp. New Haven, CT, Yale University Press, 1991. $28.50 (hardcover)

Marriage and Family Therapy. A Sociocognitive Approach. By Nathan Hurvitz, PhD, MSW, and Roger A. Straus, PhD. 238 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

PUBLIC HEALTH

Imminent Peril: Public Health in a Declining Economy. Edited by Kevin M. Cahill, MD. 148 pp. New York, Twentieth Century Fund Press, 1991. $9.95 (softcover)

RADIOLOGY

Geriatric Radiology. Edited by Manuel Viamonte, Jr. MD. 228 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1991. $65.00 (hardcover)

Rheumatology a Practical Handbook of Joint Fluid Analysis. Second Edition. By Robert A. Gatter, MD, and H. Ralph Schumacher, MD. 122 pp, illustrated. Lea & Febiger, Malvern, Pa, 1991. $42.50 (hardcover)

SUBSTANCE ABUSE

Behavioral and Biochemical Issues in Substance Abuse. Edited by Frank R. George, PhD, and Doris Clouet, PhD. 249 pp. Binghamton, NY, Haworth Press, 1991. $29.95 (hardcover)

John Peter Burghardt, MD, Douglaston. Died November 6, 1991; age 85. Dr Burghardt was a 1931 graduate of the State University of New York Health Science Center at Brooklyn, College of Medicine. He was a Fellow of the American Academy of Family Physicians, the Medical Society of the County of Queens, and the Medical Society of the State of New York.

William F.X. Campion, MD, Mount Vernon. Died October 30, 1991; age 72. Dr Campion was a 1943 graduate of Georgetown University School of Medicine, Washington, DC. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Hilliard Dubrow, MD, New York. Died June 3, 1991; age 79. Dr Dubrow was a 1936 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Obstetrics and Gynecology, a Fellow of the American College of Surgeons, and a Fellow of the American College of Obstetricians and Gynecologist. He was a member of the New York State Surgical Society, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Louis Kava, MD, Hollywood, Florida. Died October 25, 1991; age 93. Dr Kava was a 1921 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Obstetrics and Gynecology, a Fellow of the American College of Obstetricians and Gynecologist, and a Qualified Fellow of the International College of Surgeons. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Ping P. Liu, MD, Elmira. Died November 10, 1991; age 87. Dr Liu was a 1942 graduate of the University of Minnesota Medical School, Minneapolis. He was a member of the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Chemung, and the Medical Society of the State of New York.

Lucile Elise Loseke, MD, New York. Died September 25, 1991; age 81. Dr Loseke was a 1940 graduate of the University of Nebraska College of Medicine, Omaha. She was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. Her memberships included the American Medical Women's Association, the Society of Surgical Oncology, the Pan American Medical Association, the New York County Medical Society, and the Medical Society of the State of New York.

Alfred Francis Luhr, MD, Kenmore. Died November 9, 1991; age 73. Dr Luhr was a 1943 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the Society of Surgical Oncology, the American Society of Head & Neck Surgery, the Society of Head and Neck Surgeons, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Robert C. McCormick, MD, Islip. Died November 12, 1991; age 74. Dr McCormick was a 1943 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a Diplomate of the American Board of Thoracic Surgery, a Diplomate of the American Board of Surgery, and a Fellow of the American College of Surgeons. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

John Paul Ruppe, Sr, MD, Bay Shore. Died November 16, 1991; age 94. Dr Ruppe was a 1921 graduate of Georgetown University School of Medicine, Washington, DC. He was a member of the American Thoracic Society, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Felix Schwartz, MD, Clearwater, Florida. Died July 7, 1907. Dr Schwartz was a 1934 graduate of the Faculty of Medicine, University of Edinburgh, Edinburgh, Scotland. He was a member of the Sullivan County Medical Society and the Medical Society of the State of New York.

Marvin S. Tuchman, MD, Marietta, Georgia. Died October 30, 1991; age 66. Dr Tuchman was a 1947 graduate of the University of Health Science Chicago Medical School, North Chicago. His memberships included the New York State Society of Internal Medicine, the American Geriatric Society, the American Society of Internal Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

William Vilardo, MD, Ticonderoga. Died April 1, 1991; age 77. Dr Vilardo was a 1940 graduate of the University of Vermont College of Medicine, Burlington. He was a member of the Medical Society of the County of Essex and the Medical Society of the State of New York.

Solomon Wechsler, MD, Fort Lauderdale, Florida. Died November 3, 1991; age 83. Dr Wechsler was a 1933 graduate of the University of Iowa College of Medicine, Iowa City. He was a member of the American Academy of Family Physicians, the Bronx County Medical Society, and the Medical Society of the State of New York.

Paul N. Yu, MD, Rochester. Died October 8, 1991; age 76. Dr Yu was a 1939 graduate of the National Shanghai Medical College, Shanghai, Kiangsu, China. He was a Diplomate of the American Board of Internal Medicine, a Fellow of the American College of Physicians, and a Fellow of the American College of Cardiology. He was a member of the New York Academy of Medicine, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

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MISCELLANEOUS—CONT'D

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[1] Data on file, Lilly Research Laboratories.

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Laboratory Tests–False-positive tests for urobilinogen with Multistix™ may occur during therapy.

Drug Interactions–No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility–A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups. The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy–Teratogenic Effects–Pregnancy Category C–Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers–Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use–Safety and effectiveness in children have not been established.

Use in Elderly Patients–Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic–Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular–In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS–Rare cases of reversible mental confusion have been reported.

Endocrine–Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic–Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental–Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity–As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other–Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

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Index to Advertisers

AMA 7A
Classified Advertising 8A, 9A, 12A
Glaxo Pharmaceuticals . . . . . 2nd Cover, 1A
Health Research, Inc. 1A
Eli Lilly and Company 10A, 11A
McGraw Hill Publications 12A
Medix Management Services 11A
Med Pro 5A
Palisades Pharmaceuticals 3A
Sharp Disposal 11A
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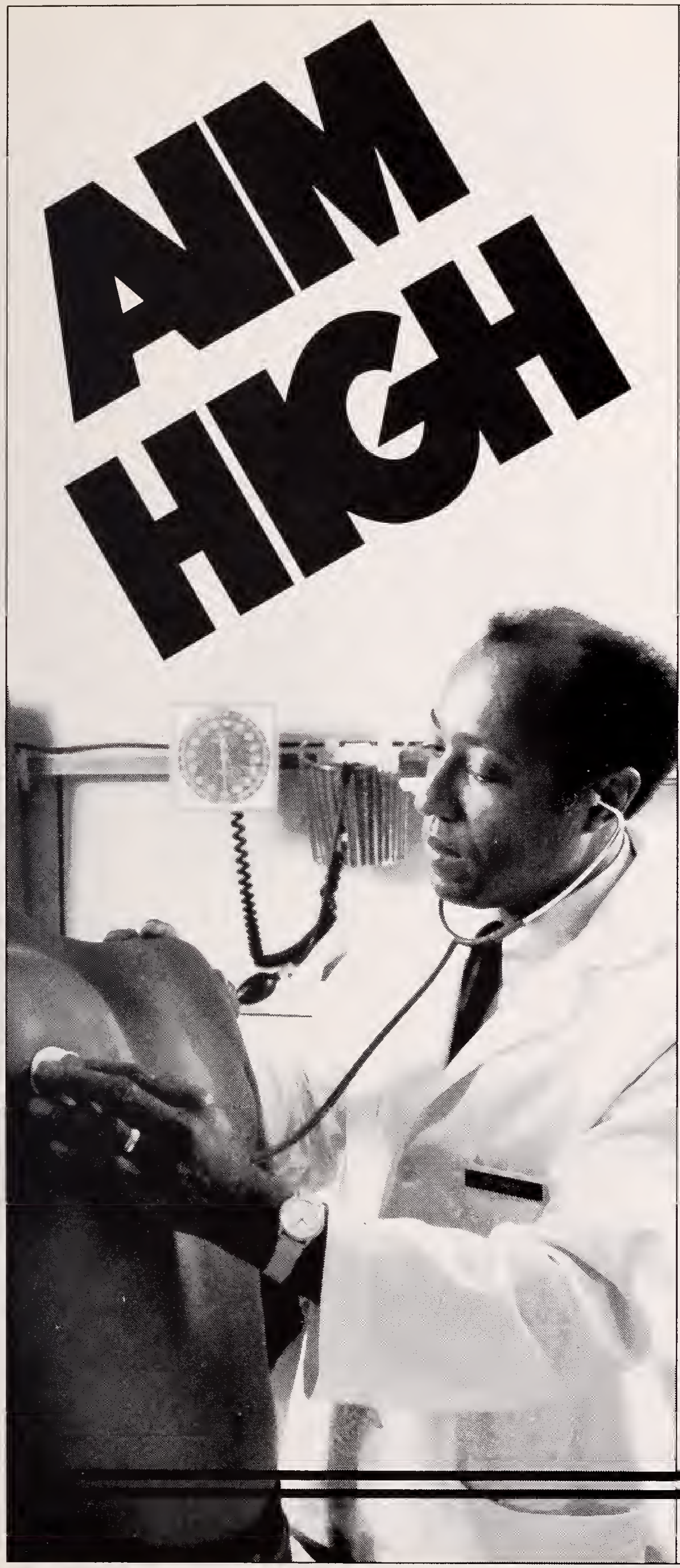
U.S. Army Reserve 8A, 9A



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BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticária, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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THE NON-PSYCHIATRIC PHYSICIAN'S RESPONSIBILITIES FOR THE SUICIDAL ADOLESCENT

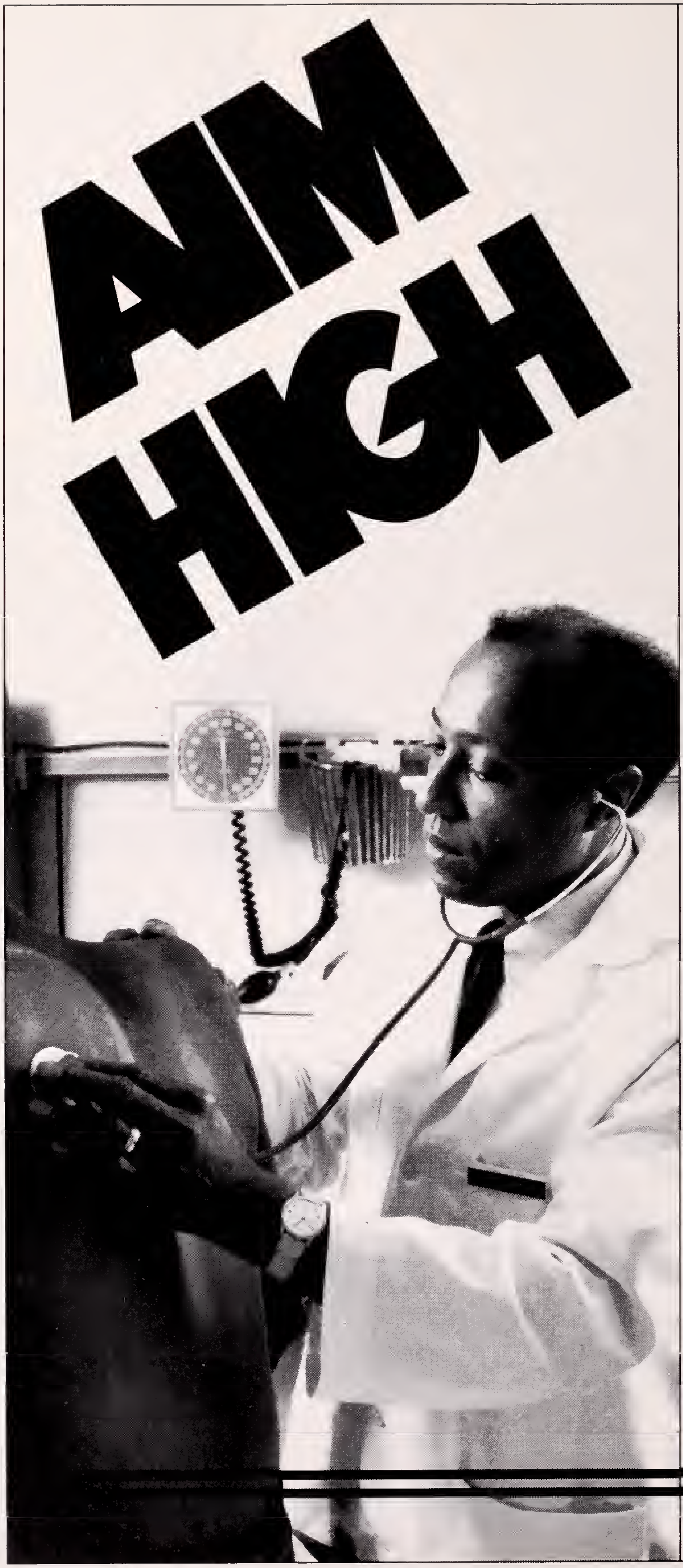
DRUG DEVELOPMENT AND PATENT LAW

GOUTY ARTHRITIS AFTER A GUNSHOT WOUND

ACUTE BUDD-CHIARI SYNDROME SECONDARY TO PAROXYSMAL NOCTURNAL HEMOGLOBINURIA

THROMBOSIS IN PAROXYSMAL NOCTURNAL HEMOGLOBINURIA

MORPHOLOGY OF VENTRICULAR PREMATURE BEATS AND LEFT VENTRICULAR DYSFUNCTION



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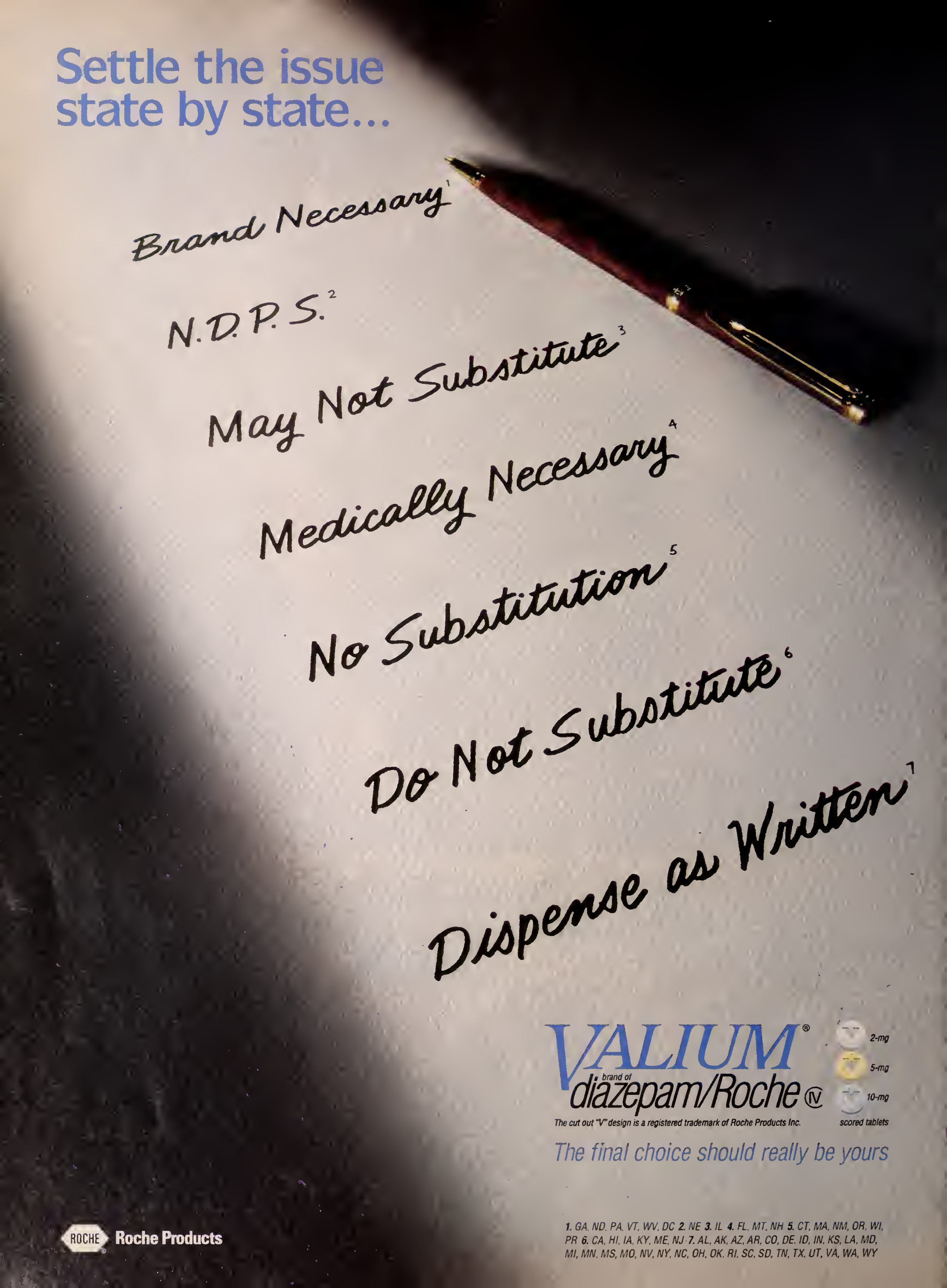

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New York State
Journal of Medicine

MARCH 1992 / Volume 92, Number 3

Contents

COMMENTARIES

85 **Prevention of youth suicide**
CYNTHIA R. PFEFFER

86 **Drug development and patent law**
NEIL J. NUSBAUM, JR, MD

88 **The continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria**
PAM WESTMORELAND SHOLAR, MD

RESEARCH PAPER

89 **Is the morphology of ventricular premature beats a marker for left ventricular dysfunction?**
KATHERINE ANGELES, MD; ROBERT BETZU, MD; LAWRENCE A. GOULD, MD

REVIEW ARTICLE

92 **Update in epilepsy—Part III: Surgical therapy of intractable epilepsy**
WEBSTER H. PILCHER, MD, PhD; CHAICHON LOCHARERNKUL, MD; DAVID PRIMROSE, MD; LINDA MORETTI OJEMANN, MD; GEORGE A. OJEMANN, MD

ESSAYS IN PEDIATRICS

97 **The non-psychiatric physician's responsibilities for the suicidal adolescent**
JEAN R. ASCHKENASY, PhD; DAVID C. CLARK, PhD; L. DAVID ZINN, MD; ANTHONY J. RICHTSMEIER, MD

SPECIAL ARTICLE

105 **The challenge to psychiatry by psychologists: Economic versus professional issues**
MICHAEL S. ARONOFF, MD, ZIRA De FRIES, MD

(continued on p 6A)

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

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AROUND THE STATE

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Apr 16. **Frontiers in Hepatobiliary Disease.** Edith Macy Conference Center. Contact: Office of Continuing Medical Education, 3301 Bainbridge Ave, Bronx, NY 110467. Tel: (212) 920-6674.

MANHATTAN

Apr 23–24. **Computers and Nursing Practice.** NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295. *Apr 26.* **Fourth Annual Symposium on Treatment of Headaches and Facial Pain.** New York Marriott. Contact: Alexander Mauskop, MD, Director, The Downstate Headache Center, 132 Atlantic Ave, Brooklyn Heights, NY 11201. Tel: (718) 935-9666.

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Apr 1–4. **17th Annual Primary Care Update.** Westward Look Resort, Tucson. Contact: Continuing Medical Education, Arizona Health Sciences Center, Tucson, Arizona 85724. Tel: (602) 626-7832.

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Apr 2–4. **Postgraduate Course in General Surgery.** Ritz-Carlton Hotel, San Francisco. *Apr 24–26.* **West Coast Regional Conference on Pediatric Psychology.** Union Square Holiday Inn, San Francisco. Contact: Extended Programs in Medical Education, Room LS-105, University of California School of Medicine, San Francisco, CA 94143. Tel: (415) 476-4251.

Apr 9–11. **Current Regulatory Issues in Medical Physics.** *Apr 19–24.* **12th Annual Resident's Radiology Review Course.** *Apr 25.* **Interventional Radiology.** Hotel Del Coronado, San Diego. Contact: Dawne Ryals, Ryals & Associ-

(continued on p 13A)

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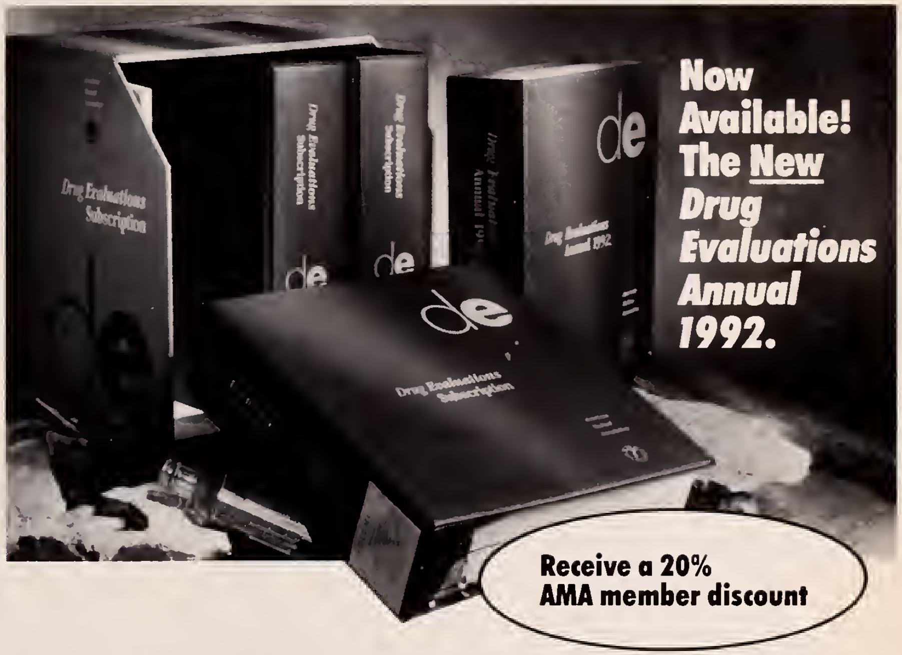

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CASE REPORTS

109 **Recombinant tissue-plasminogen activator for acute Budd-Chiari syndrome secondary to paroxysmal nocturnal hemoglobinuria**
TAK KWAN, MD, PAUL HANSARD, MD

110 **Gouty arthritis following gunshot wound: A case report**
EDDYS DISLA, MD; HARLEEN BRAR, MD; ANGELO TARANTA, MD

111 **Facial necrotizing fasciitis due to *Streptococcus pyogenes***
RAPHAEL BARRERA, MD; PHILIP V. FELICE, MD; PAUL RAGNAUTH, MD; JANUSZ SAWICKI, MD; IRA L. SALOM, MD

113 **Pneumococcal cellulitis and dysgammaglobulinemia**
ALI HAMMAD, MD; MOLLY ZITTEL, DO; EUGENE KALMUK, MD; JOSEPH MYLOTTE, MD

LETTERS TO THE EDITOR

115 **Impaction of an ingested table fork in a patient with a surgically restricted stomach**
SEBASTIANO CASSARO, MD, MAURIZIO DALIANA, MD

115 **Joseph Isaac Bluestone**
MALVIN E. RING, DDS

115 **Project diabetes 2000**
JAMES McGROARTY, MD

116 **The human tail**
ALFRED L. STANDFAST, MD

116 **The ultimate care givers**
RANDALL D. BLOOMFIELD, MD

117 LEADS FROM EPIDEMIOLOGY NEWS

118 BOOK REVIEWS

122 UNIFORM REQUIREMENTS FOR MANUSCRIPTS SUBMITTED TO BIOMEDICAL JOURNALS

126 GUIDELINES TO AUTHORS

5A MEDICAL MEETINGS AND LECTURES

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COMPARATIVE PHARMACOLOGY OF TWO ANALGESICS

	Constipation	Respiratory Depression	Sedation	Emesis	Physical Dependence
HYDROCODONE		X			X
OXYCODONE	XX	XX	XX	XX	XX

Blank space indicates that no such activity has been reported. Table adapted from Facts and Comparisons 1991 and Catalano RB. The medical approach to management of pain caused by cancer. *Semin. Oncol.* 1975; 2; 379-92 and Reuler JB. et. al. The chronic pain syndrome: misconceptions and management. *Ann. Intern. Med.* 1980 588-96.

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2. Standard industry new prescription audit

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CONTRAINDICATIONS: Hypersensitivity to acetaminophen or hydrocodone.

WARNINGS:

Allergic-Type Reactions: VICODIN/VICODIN ES Tablets contain sodium metabisulfite, a sulfite that may cause allergic-type reactions including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in certain susceptible people.

Respiratory Depression: At high doses or in sensitive patients, hydrocodone may produce dose-related respiratory depression.

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Acute Abdominal Conditions: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions.

PRECAUTIONS:

Special Risk Patients: VICODIN/VICODIN ES Tablets should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture.

Cough Reflex: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN/VICODIN ES Tablets are used postoperatively and in patients with pulmonary disease.

Drug Interactions: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN/VICODIN ES Tablets may exhibit an additive CNS depression. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus.

Usage in Pregnancy:

Teratogenic Effects: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN/ VICODIN ES Tablets should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nonteratogenic effects: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever.

Labor and Delivery: Administration of VICODIN/VICODIN ES Tablets to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used

Nursing Mothers: It is not known whether this drug is excreted in human milk Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN/VICODIN ES Tablets, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: Safety and effectiveness in children have not been established

ADVERSE REACTIONS:

The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting. These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include.

Central Nervous System: Drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, psychic dependence and mood changes.

Gastrointestinal System: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia. Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation.

Genitourinary System: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported.

Respiratory Depression: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated.

DRUG ABUSE AND DEPENDENCE:

VICODIN/VICODIN ES Tablets are subject to the Federal Controlled Substance Act (Schedule III). Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN/ VICODIN ES Tablets should be prescribed and administered with caution.

OVERDOSAGE:

Acetaminophen Signs and Symptoms: In acute acetaminophen overdosage, dose-dependent, potentially fatal hepatic necrosis is the most serious adverse effect. Renal tubular necrosis, hypoglycemic coma, and thrombocytopenia may also occur. Early symptoms following a potentially hepatotoxic overdose may include: nausea, vomiting, diaphoresis and general malaise. Clinical and laboratory evidence of hepatic toxicity may not be apparent until 48 to 72 hours post-ingestion.

Hydrocodone Signs and Symptoms: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur.

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Please see brief summary of prescribing information on adjacent page.

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From Depression...Into the Mainstream

ONCE-A-DAY

Zoloft™

(sertraline HCl) 50 mg, 100 mg scored tablets

BRIEF SUMMARY

ZOLOFT™ (sertraline HCl)

INDICATIONS AND USAGE: ZOLOFT (sertraline hydrochloride) is indicated for the treatment of depression.

CONTRAINDICATIONS: None known. **WARNINGS: In patients receiving another serotonin reuptake inhibitor drug in combination with a monoamine oxidase inhibitor (MAOI), there have been reports of serious, sometimes fatal, reactions including hyperthermia, rigidity, myoclonus, autonomic instability with possible rapid fluctuations of vital signs, and mental status changes that include extreme agitation progressing to delirium and coma. These reactions have also been reported in patients who have recently discontinued that drug and have been started on an MAOI. Some cases presented with features resembling neuroleptic malignant syndrome. Therefore, it is recommended that ZOLOFT (sertraline hydrochloride) not be used in combination with an MAOI, or within 14 days of discontinuing treatment with an MAOI. Similarly, at least 14 days should be allowed after stopping ZOLOFT before starting an MAOI.**

PRECAUTIONS ***General:*** **Activation of Mania/Hypomania** - During premarketing testing, hypomania or mania occurred in approximately 0.4% of ZOLOFT (sertraline hydrochloride) treated patients. Activation of mania/hypomania has also been reported in a small proportion of patients with Major Affective Disorder treated with other marketed antidepressants. **Weight Loss** - Significant weight loss may be an undesirable result of treatment with sertraline for some patients, but on average, patients in controlled trials had minimal, 1 to 2 pound weight loss, versus smaller changes on placebo. Only rarely have sertraline patients been discontinued for weight loss. **Seizure** - ZOLOFT has not been evaluated in patients with a seizure disorder. These patients were excluded from clinical studies during the product's premarket testing. Accordingly, like other antidepressants, ZOLOFT should be introduced with care in epileptic patients. **Suicide** - The possibility of a suicide attempt is inherent in depression and may persist until significant remission occurs. Close supervision of high risk patients should accompany initial drug therapy. Prescriptions for ZOLOFT should be written for the smallest quantity of tablets consistent with good patient management, in order to reduce the risk of overdose. **Weak Uricosuric Effect** - ZOLOFT is associated with a mean decrease in serum uric acid of approximately 7%. The clinical significance of this weak uricosuric effect is unknown, and there have been no reports of acute renal failure with ZOLOFT. **Use in Patients with Concomitant Illness** - Clinical experience with ZOLOFT in patients with certain concomitant systemic illness is limited. Caution is advisable in using ZOLOFT in patients with diseases or conditions that could affect metabolism or hemodynamic responses. ZOLOFT has not been evaluated or used to any appreciable extent in patients with a recent history of myocardial infarction or unstable heart disease. Patients with these diagnoses were excluded from clinical studies during the product's premarket testing. However, the electrocardiograms of 774 patients who received ZOLOFT in double-blind trials were evaluated and the data indicate that ZOLOFT is not associated with the development of significant ECG abnormalities. ZOLOFT is extensively metabolized by the liver. The pharmacokinetics of ZOLOFT have not been studied in patients with significant hepatic dysfunction nor have patients with significant hepatic dysfunction been evaluated during treatment with ZOLOFT. Accordingly, ZOLOFT should be used with caution in such patients. Since ZOLOFT is extensively metabolized, excretion of unchanged drug in urine is a minor route of elimination. However, until the pharmacokinetics of ZOLOFT have been studied in patients with renal impairment and until adequate numbers of patients with severe renal impairment have been evaluated during chronic treatment with ZOLOFT, it should be used with caution in such patients. **Interference with Cognitive and Motor Performance** - In controlled studies, ZOLOFT did not cause sedation and did not interfere with psychomotor performance. ***Information for Patients:*** Physicians are advised to discuss the following issues with patients for whom they prescribe ZOLOFT: Patients should be told that although ZOLOFT has not been shown to impair the ability of normal subjects to perform tasks requiring complex motor and mental skills in laboratory experiments, drugs that act upon the central nervous system may affect some individuals adversely. Patients should be told that although ZOLOFT has not been shown in experiments with normal subjects to increase the mental and motor skill impairments caused by alcohol, the concomitant use of ZOLOFT and alcohol in depressed patients is not advised. Patients should be told that while no adverse interaction of ZOLOFT with over-the-counter (OTC) drug products is known to occur, the potential for interaction exists. Thus, the use of any OTC product should be initiated cautiously according to the directions of use given for the OTC product. Patients should be advised to notify their physician if they become pregnant or intend to become pregnant during therapy. Patients should be advised to notify their physician if they are breast-feeding an infant. ***Laboratory Tests:*** None. ***Drug Interactions:* Potential Effects of Coadministration of Drugs Highly Bound to Plasma Proteins** - Because sertraline is tightly bound to plasma protein, the administration of ZOLOFT (sertraline hydrochloride) to a patient taking another drug which is tightly bound to protein (e.g., warfarin, digitoxin) may cause a shift in plasma concentrations potentially resulting in an adverse effect. Conversely, adverse effects may result from displacement of protein-bound ZOLOFT by other tightly bound drugs. In a study comparing prothrombin time AUC (0-120 hr) following dosing with warfarin (0.75 mg/kg) before and after 21 days of dosing with either ZOLOFT (50-200 mg/day) or placebo, there was a mean increase in prothrombin time of 8% relative to baseline for ZOLOFT compared to a 1% decrease for placebo ($p<0.02$). The normalization of prothrombin time for the ZOLOFT group was delayed compared to the placebo group. The clinical significance of this change is unknown. Accordingly, prothrombin time should be carefully monitored when ZOLOFT therapy is initiated or stopped. **CNS Active Drugs** - In a study comparing the disposition of intravenously administered diazepam before and after 21 days of dosing with either ZOLOFT (50 to 200 mg/day escalating dose) or placebo, there was a 32% decrease relative to baseline in diazepam clearance for the ZOLOFT group compared to a 19% decrease relative to baseline for the placebo group ($p<0.03$). There was a 23% increase in T_{max} for desmethyldiazepam in the ZOLOFT group compared to a 20% decrease in the placebo group ($p<0.03$). The clinical significance of these changes is unknown. In a placebo-controlled trial in normal volunteers, the administration of two doses of ZOLOFT did not significantly alter steady-state lithium levels or the renal clearance of lithium. Nonetheless, at this time, it is recommended that plasma lithium levels be monitored following initiation of ZOLOFT therapy with appropriate adjustments to the lithium dose. The risk of using ZOLOFT in combination with other CNS active drugs has not been systematically evaluated. Consequently, caution is advised if the concomitant administration of ZOLOFT and such drugs is required. **Hypoglycemic Drugs** - In a placebo-controlled trial in normal volunteers, administration of ZOLOFT for 22 days (including 200 mg/day for the final 13 days) caused a statistically significant 16% decrease from baseline in the clearance of tolbutamide following an intravenous 1000 mg dose. ZOLOFT administration did not noticeably change either the plasma protein binding or the apparent volume of distribution of tolbutamide, suggesting that the decreased clearance was due to a change in the metabolism of the drug. The clinical significance of this decrease in tolbutamide clearance is unknown. **Atenolol** - ZOLOFT (100 mg) when administered to 10 healthy male subjects had no effect on the beta-adrenergic blocking ability of atenolol. **Microsomal Enzyme Induction** - Preclinical studies have shown ZOLOFT to induce hepatic microsomal enzymes. In clinical studies ZOLOFT was shown to induce hepatic enzymes minimally as determined by a small (5%) but statistically significant decrease in antipyrine half-life following administration of 200 mg/day for 21 days. This small change in antipyrine half-life reflects a clinically insignificant change in hepatic metabolism. **Electroconvulsive Therapy** - There are no clinical studies establishing the risks or benefits of the combined use of electroconvulsive therapy (ECT) and ZOLOFT. **Alcohol** - Although ZOLOFT did not potentiate the cognitive and psychomotor effects of alcohol in experiments with normal subjects, the concomitant use of ZOLOFT and alcohol in depressed patients is not recommended. ***Carcinogenesis, Mutagenesis, Impairment of Fertility:*** Lifetime carcinogenicity studies were carried out in CD-1 mice and Long-Evans rats at doses up to 40 mg/kg in mice (10 times on a mg/kg basis and the same on a mg/m^2 basis as the maximum recommended human dose) and at doses up to 40 mg/kg in rats (10 times on a mg/kg basis and 2 times on a mg/m^2 basis, the maximum recommended human dose). There was a dose-related increase in the incidence of liver adenomas in male mice receiving sertraline at 10-40 mg/kg. No increase was seen in female mice or in rats of either sex receiving the same treatments, nor was there an increase in hepatocellular carcinomas. Liver adenomas have a variable rate of spontaneous occurrence in the CD-1 mouse and are of unknown significance to humans. There was an increase in follicular adenomas of the thyroid in female rats receiving sertraline at 40 mg/kg; this was not accompanied by thyroid hyperplasia. While there was an increase in uterine adenocarcinomas in rats receiving sertraline at 10-40 mg/kg compared to placebo controls, this effect was not clearly drug related. Sertraline had no genotoxic effects, with or without metabolic activation, based on the following assays: bacterial mutation assay; mouse lymphoma mutation assay; and tests for cytogenetic aberrations *in vivo* in mouse bone marrow and *in vitro* in human lymphocytes. A decrease in fertility was seen in one of two rat studies at a dose of 80 mg/kg (20 times the maximum human dose on a mg/kg basis and 4 times on a mg/m^2 basis). ***Pregnancy—Pregnancy Category B:* Teratogenic Effects** - Reproduction studies have been performed in rats and rabbits at doses up to approximately 20 times and 10 times the maximum daily human mg/kg dose (4 to 4.5 times the mg/m^2 dose), respectively. There was no evidence of teratogenicity at any dose level. At doses approximately 2.5-10 times the maximum daily human mg/kg dose, sertraline was associated with delayed ossification in fetuses, probably secondary to effects on the dams. There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. **Non-teratogenic Effects** - There was also decreased neonatal survival following maternal administration of sertraline at doses as low as approximately 5 times the maximum human mg/kg dose. The decrease in pup survival was shown to be most probably due to *in utero* exposure to sertraline. The clinical significance of these effects is unknown. **Labor and Delivery** - The effect of ZOLOFT on labor and delivery in humans is unknown. **Nursing Mothers** - It is not known whether, and if so in what amount, sertraline or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when ZOLOFT is administered to a nursing woman. **Pediatric Use** - Safety and effectiveness in children have not been established. **Geriatric Use** - Several hundred elderly patients have participated in clinical studies with ZOLOFT. The pattern of adverse reactions in the elderly was similar to that in younger patients. **ADVERSE REACTIONS** ***Commonly Observed:*** The most commonly observed adverse events associated with the use of ZOLOFT (sertraline hydrochloride) and not seen at an equivalent incidence among placebo-treated patients were: gastrointestinal complaints, including nausea, diarrhea/loose stools and dyspepsia; tremor; dizziness; insomnia; somnolence; increased sweating; dry mouth; and male sexual dysfunction (primarily ejaculatory delay). ***Associated with Discontinuation of Treatment:*** Fifteen percent of 2710 subjects who received ZOLOFT in premarketing multiple dose clinical trials discontinued treatment due to an adverse event. The more common events (reported by at least 1% of subjects) associated with discontinuation included agitation, insomnia, male sexual dysfunction (primarily ejaculatory delay), somnolence, dizziness, headache, tremor, anorexia, diarrhea/loose stools, nausea, and fatigue. ***Incidence in Controlled Clinical Trials:*** The table that follows enumerates adverse events that occurred at a frequency of 1% or more among ZOLOFT patients who participated in controlled trials comparing titrated ZOLOFT with placebo. Most patients received doses of 50 to 200 mg per day.

Treatment-Emergent Adverse Experience Incidence in Placebo-Controlled Clinical Trials*

(Percent of Patients Reporting) **(Percent of Patients Reporting)**

Adverse Experience	**ZOLOFT** (N=861)	**Placebo** (N=853)	**Adverse Experience**	**ZOLOFT** (N=861)	**Placebo** (N=853)
Autonomic Nervous System Disorders			Hot Flushes	2.2	0.5
Mouth Dry	16.3	9.3	Fever	1.6	0.6
Sweating Increased	8.4	2.9	Back Pain	1.5	0.9
Cardiovascular			**Metabolic and Nutritional Disorders**		
Palpitations	3.5	1.6	Thirst	1.4	0.9
Chest Pain	1.0	1.6	**Musculoskeletal System Disorders**		
Central and Peripheral Nervous System Disorders			Myalgia	1.7	1.5
			Psychiatric Disorders		
Headache	20.3	19.0	Insomnia	16.4	8.8
Dizziness	11.7	6.7	Sexual Dysfunction-Male (1)	15.5	2.2
Tremor	10.7	2.7	Somnolence	13.4	5.9
Paresthesia	2.0	1.8	Agitation	5.6	4.0
Hypaesthesia	1.7	0.6	Nervousness	3.4	1.9
Twitching	1.4	0.1	Anxiety	2.6	1.3
Hypertonia	1.3	0.4	Yawning	1.9	0.2
Disorders of Skin and Appendages			Sexual Dysfunction-Female (2)	1.7	0.2
Rash	2.1	1.5	Concentration Impaired	1.3	0.5
Gastrointestinal Disorders			**Reproductive**		
Nausea	26.1	11.8	Menstrual Disorder (2)	1.0	0.5
Diarrhea/Loose Stools	17.7	9.3	**Respiratory System Disorders**		
Constipation	8.4	6.3	Rhinitis	2.0	1.5
Dyspepsia	6.0	2.8	Pharyngitis	1.2	0.9
Vomiting	3.8	1.8	**Special Senses**		
Flatulence	3.3	2.5	Vision Abnormal	4.2	2.1
Anorexia	2.8	1.6	Tinnitus	1.4	1.1
Abdominal Pain	2.4	2.2	Taste Perversion	1.2	0.7
Appetite Increased	1.3	0.9	**Urinary System Disorders**		
General			Micturition Frequency	2.0	1.2
Fatigue	10.6	8.1	Micturition Disorder	1.4	0.5

*Events reported by at least 1% of patients treated with ZOLOFT are included.

(1) - % based on male patients only: 271 ZOLOFT (primarily ejaculatory delay) and 271 placebo patients.

(2) - % based on female patients only: 590 ZOLOFT and 582 placebo patients.

Other Events Observed During the Premarketing Evaluation of ZOLOFT (sertraline hydrochloride): During its premarketing assessment, multiple doses of ZOLOFT were administered to approximately 2700 subjects. Events are further categorized by body system and listed in order of decreasing frequency according to the following definitions: frequent adverse events are those occurring on one or more occasions in at least 1/100 patients (only those not already listed in the tabulated results from placebo-controlled trials appear in this listing); infrequent adverse events are those occurring in 1/100 to 1/1000 patients; rare events are those occurring in fewer than 1/1000 patients. Events of major clinical importance are also described in the PRECAUTIONS section. **Autonomic Nervous System Disorders**—*Infrequent:* flushing, mydriasis, increased saliva, cold clammy skin; *Rare:* pallor. **Cardiovascular**—*Infrequent:* postural dizziness, hypertension, hypotension, postural hypotension, edema, dependent edema, periorbital edema, peripheral edema, peripheral ischemia, syncope, tachycardia; *Rare:* precordial chest pain, substernal chest pain, aggravated hypertension, myocardial infarction, varicose veins. **Central and Peripheral Nervous System Disorders**—*Frequent:* confusion; *Infrequent:* ataxia, abnormal coordination, abnormal gait, hyperesthesia, hyperkinesia, hypokinesia, migraine, nystagmus, vertigo; *Rare:* local anesthesia, coma, convulsions, dyskinesia, dysphonia, hyporeflexia, hypotonia, ptosis. **Disorders of Skin and Appendages**—*Infrequent:* acne, alopecia, pruritus, erythematous rash, maculopapular rash, dry skin; *Rare:* bullous eruption, dermatitis, erythema multiforme, abnormal hair texture, hypertrichosis, photosensitivity reaction, follicular rash, skin discoloration, abnormal skin odor, urticaria. **Endocrine Disorders**—*Rare:* exophthalmos, gynecomastia. **Gastrointestinal Disorders**—*Infrequent:* dysphagia, eructation; *Rare:* diverticulitis, fecal incontinence, gastritis, gastroenteritis, glossitis, gum hyperplasia, hemorrhoids, hiccup, melena, hemorrhagic peptic ulcer, proctitis, stomatitis, ulcerative stomatitis, tenesmus, tongue edema, tongue ulceration. **General**—*Frequent:* asthenia; *Infrequent:* malaise, generalized edema, rigors, weight decrease, weight increase; *Rare:* enlarged abdomen, halitosis, otitis media, aphthous stomatitis. **Hematopoietic and Lymphatic**—*Infrequent:* lymphadenopathy, purpura; *Rare:* anemia, anterior chamber eye hemorrhage. **Metabolic and Nutritional Disorders**—*Rare:* dehydration, hypercholesterolemia, hypoglycemia. **Musculoskeletal System Disorders**—*Infrequent:* arthralgia, arthrosis, dystonia, muscle cramps, muscle weakness; *Rare:* hernia. **Psychiatric Disorders**—*Infrequent:* abnormal dreams, aggressive reaction, amnesia, apathy, delusion, depersonalization, depression, aggravated depression, emotional lability, euphoria, hallucination, neurosis, paranoid reaction, suicide ideation and attempt, teeth-grinding, abnormal thinking; *Rare:* hysteria, somnambulism, withdrawal syndrome. **Reproductive**—*Infrequent:* dysmenorrhea (2), intermenstrual bleeding (2); *Rare:* amenorrhea (2), balanoposthitis (1), breast enlargement (2), female breast pain (2), leukorrhea (2), menorrhagia (2), atrophic vaginitis (2).

(1) - % based on male subjects only: 1005; (2) - % based on female subjects only: 1705.

Respiratory System Disorders—*Infrequent:* bronchospasm, coughing, dyspnea, epistaxis; *Rare:* bradypnea, hyperventilation, sinusitis, stridor. **Special Senses**—*Infrequent:* abnormal accommodation, conjunctivitis, diplopia, earache, eye pain, xerophthalmia; *Rare:* abnormal lacrimation, photophobia, visual field defect. **Urinary System Disorders**—*Infrequent:* dysuria, face edema, nocturia, polyuria, urinary incontinence; *Rare:* oliguria, renal pain, urinary retention. **Laboratory Tests**: In man, asymptomatic elevations in serum transaminases (SGOT [or AST] and SGPT [or ALT]) have been reported infrequently (approximately 0.8%) in association with ZOLOFT administration. These hepatic enzyme elevations usually occurred within the first 1 to 9 weeks of drug treatment and promptly diminished upon drug discontinuation. ZOLOFT therapy was associated with small mean increases in total cholesterol (approximately 3%) and triglycerides (approximately 5%), and a small mean decrease in serum uric acid (approximately 7%) of no apparent clinical importance. **DRUG ABUSE AND DEPENDENCE** ***Controlled Substance Class*** - ZOLOFT (sertraline hydrochloride) is not a controlled substance. ***Physical and Psychological Dependence*** - ZOLOFT has not been systematically studied, in animals or humans, for its potential for abuse, tolerance, or physical dependence. However, the premarketing clinical experience with ZOLOFT did not reveal any tendency for a withdrawal syndrome or any drug-seeking behavior. As with any new CNS active drug, physicians should carefully evaluate patients for history of drug abuse and follow such patients closely, observing them for signs of ZOLOFT misuse or abuse (e.g., development of tolerance, incrementation of dose, drug-seeking behavior). **OVERDOSAGE** ***Human Experience*** - There have been 3 cases of ZOLOFT (sertraline hydrochloride) overdosage (approximately 750-2,100 mg). No specific therapy was required for any of the 3 patients, all of whom recovered completely. ***Management of Overdoses*** - Establish and maintain an airway, insure adequate oxygenation and ventilation. Activated charcoal, which may be used with sorbitol, may be as or more effective than emesis or lavage, and should be considered in treating overdose. Cardiac and vital signs monitoring is recommended along with general symptomatic and supportive measures. There are no specific antidotes for ZOLOFT. Due to the large volume of distribution of ZOLOFT, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. In managing overdosage, consider the possibility of multiple drug involvement. The physician should consider contacting a poison control center on the treatment of any overdose.

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COMMENTARIES

Prevention of youth suicide

Health care in the United States has undergone tremendous changes with advances in diagnostic and treatment technologies that have, in some cases eradicated or diminished certain pathologies. In consonance with this, dramatic changes have occurred that have been highlighted by earlier and increased prevalence of depression, violence, substance abuse, and eating disorders especially among youth.[1] For example, in recent years, intentional death has been among the three most common causes of death among young people. In 1988, approximately 4,929 individuals between 15- and 24-years old committed suicide.[2] This is equivalent to a rate of 13.2 per 100,000 population. Physicians are therefore more likely to work with youthful patients who have a variety of physical and psychosocial concerns; an emphasis of health care that requires clinicians to develop additional evaluation and intervention skills.

The realization that youth suicidal behavior is a national mental health problem with high rates of morbidity and mortality and the need for intensive intervention for youngsters at risk occurred in the early 1980s after several clusters of youth suicide were noted in communities such as Westchester County, New York, Plano, Texas, Chicago, Illinois. Public alarm stimulated efforts to prevent this tragic phenomenon. The earliest efforts at prevention included media discussions of youth suicide and factors indicative of suicidal risk. These media reports, although useful to sensitize others about the incidence and prevalence of youth suicidal behavior, also were observed to increase risk for youth suicide, especially within the first two weeks of media reports of an adolescent suicidal death.[3] Caution is now needed in media presentations about youth suicidal behavior.

Other important prevention efforts for youth suicidal behavior were developed within local communities to decrease risk for imitative suicide among teenagers. School-based suicide prevention programs to educate all students about signs indicative of suicidal risk were instituted. However, most of these programs were not evaluated for their effectiveness. Recent research suggests that there may not be benefits of such programs, which are geared to educating youth about suicidal behavior.[4]

National efforts to prevent youth suicidal behavior advocated public health techniques to decrease rates of youth suicide. In 1987, the Secretary's Task Force on Youth Suicide Prevention was sponsored by the US Department of Health and Human Services with an aim of setting national priorities to develop approaches for prevention of youth suicide. A four volume report of this Task Force was published in 1989.[5] The main recommendations were the following: develop data on youth suicide and attempted suicide, conduct research on risk factors for youth suicide, evaluate the effectiveness and cost of interventions to prevent youth suicide, support delivery of youth suicide prevention services, inform and educate public and health service providers about youth suicide, and involve public and private sectors in prevention of youth suicide.

The paper in this issue of the *Journal* by Aschkenasy et al[6] is written in the spirit proposed by the National Task Force on Youth Suicide. It aims to educate non-psychiatric physicians about the epidemiology, risk factors, assessment techniques, intervention and prevention methods for youth suicidal behavior. Its important messages for practicing clinicians are to know their patients, to monitor emotional response and behavior, to intervene in a timely fashion, and to refer patients to mental health specialists when suicidal risk is appreciable. Comprehensive care of vulnerable youngsters who have a history of suicidal behavior are at high risk for repeated suicidal episodes.[7,8]

Is it feasible to prevent youth suicide? It may not be possible to answer this question definitively because suicide occurs with too low a frequency to evaluate it in most studies as an outcome. For example, it may not be feasible to conduct prospective studies to evaluate the effects of assessment or intervention on suicide rates especially because populations needed to identify cases of youth suicide would have to be exceptionally large. However, a public health model of intervention is based on the administration of treatments or the implementation of prevention techniques among large populations. These approaches can be evaluated with respect to reduction of youth suicide rates. Several public health approaches may be suggested to reduce youth suicide in the United States.

First, research suggests that the most common method used by youngsters to commit suicide is firearms.[8] Therefore, prevention efforts that successfully reduced accessibility to firearms may effectively decrease youth suicide rates.

Second, vulnerability factors, such as substance abuse, violence, depression, and environmental stress are important correlates of youth suicide. A variety of programs to decrease the prevalence of such factors need to be developed. For example, vulnerable youngsters are often seen by physicians. Therefore, it is the medical responsibility of all physicians to identify such youngsters and intervene or refer them to appropriate treatment settings.

Third, high standards of effective medical care must be maintained and enhanced by instituting programs and disseminating information to educate physicians about youth suicidal behavior. By maintaining uniformly high standards of medical care, youth who are vulnerable to suicidal behavior may be adequately identified and treated. This type of public health approach will increase the network in which help for youngsters at risk can be provided.

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1. Klerman GL: Suicide, depression and related problems among the Baby Boom cohort. In: *Suicide Among Youth: Perspectives on Risk and Prevention,* Edited by C.R. Pfeffer, American Psychiatric Press Inc, Washington, DC, 1989.

2. National Center for Health Statistics: Monthly vital statistics report. 1988; 39.

3. Philips DP, Carstensen LL, Paight DJ: Effects of mass media news stories on suicide, with new evidence on the role of story content. In: *Suicide Among Youth: Perspectives on Risk and Prevention,* Edited by C.R. Pfeffer, American Psychiatric Press Inc, Washington, DC, 1989.

4. Shaffer D, Vielaud V, Garland A, Rojas M, Underwood M, Busner C: Adolescent suicide attempters: Response to suicide-prevention programs. *JAMA* 1990; 264:3151-3155.

5. Alcohol, Drug Abuse, and Mental Health Administration. Report of the Secretary's Task Force on Youth Suicide. DHHS. Publication No. (ADM) 89-1621. Washington, DC, Superintendent of Documents, US Government Printing Office, 1989.

6. Aschkenasy JR, Clark DC, Zinn LD, et al: The non-psychiatric physician's responsibilities for the suicidal adolescent. *NY State J Med* 1991; 91:97-104.

7. Pfeffer CR, Klerman GL, Hurt SW, et al: Suicidal children grow up: Demographic and clinical risk factors for adolescent suicide attempts. *J Amer Acad Child Adolesc Psychiatry* 1991; 30:609-616.

8. Fingerhut LA, Kleinman JC, Godfrey E, et al: Firearm mortality among children, youth, and young adults 1-34 years of age, trends and current status: United States, 1979-1988. Monthly vital statistics report: Volume 39, number 11, supp. Hyattsville, Maryland, National Center for Health Statistics.

Drug development and patent law

The process of drug development and marketing in the United States is influenced by many factors, including the economic incentives for pharmaceutical companies. Patent protection for drugs is a major factor in this regard. As such, the patent law involves important and competing public policy considerations.

The costs of developing and marketing a new drug in the United States have recently been estimated to average $231 million.[1] This estimate is based on a number of assumptions and includes not only the cost of developing the minority of successful drugs but the products that are never brought to market. Critics have noted the "softness" of this $231 million estimate, but it seems clear that the investment in development of a successful new pharmaceutical agent is substantial.[2] Drug development costs can be expected to be considerably less, and the chances of success are presumably greater, when one develops a relatively minor modification of an existing pharmaceutical rather than a radically different product.

This economic environment creates a strong incentive to continually develop new products and to obtain the widest possible market for each of them. This is particularly important for those pharmaceutical concerns whose fortunes are tied to one or a few drugs.[3] A drug marketing program therefore is likely to consider not only marketing in a single country, but also methods of obtaining sales on an international basis. Both drug development[4] and drug marketing[5] today should be understood as efforts on a global scale.

The complexity of inventions and the cost of their development have certainly increased both in the pharmaceutical industry and elsewhere. The basic principles underlying the availability of patent protection for inventions are long standing. They are expressed in the United States Constitution as "To promote the Progress of Science and useful Arts by securing for limited Times to Authors and Inventors the exclusive Right to their respective Writings and Discoveries . . . ."[6] In exchange for obtaining patent protection, the inventor is obliged to place information regarding his invention in the public record.[7]

In at least one respect, the United States has been especially generous in the scope of patent protection afforded to pharmaceutical inventions. The United States, and recently also some European countries, allow a patent to be obtained not only for a totally new drug, but also for a new pharmaceutical application of a known compound.[8] For example, a patent was issued in 1989 for the use of an androgenic steroid, already widely used in hematologic practice, as a claimed method of treatment for sickle cell anemia.[9] It should be noted that the incremental costs of developing a new application for a pharmaceutical that has already been Food and Drug Administration (FDA) approved are likely to be much smaller than the figures for developing a new drug. In particular, there is likely to exist extensive pre-clinical data on the drug and clinical experience with the drug that can identify important drug interactions and important contraindications for drug use.

Furthermore, FDA regulations make it easier for individual physicians to explore unapproved uses of drugs than to do clinical investigations of unapproved drugs.[10] All this makes it considerably easier to obtain regulatory approval for FDA labelling for a new indication, once efficacy for the new indication has been established.

The process of FDA regulatory approval has an important economic impact on the return on drug development. In addition to the cost of preparing documentation to secure approval from the FDA for marketing, any attempt of a manufacturer to recoup its investment by marketing the drug will be delayed by the amount of time it takes to obtain approval for marketing. In the case where the FDA is much slower in granting marketing approval for the patented product, much of the 17-year term of patent protection will run out without economic benefit to the patentee. To deal with this problem, 1984 federal legislation provided limited circumstances under which the patent terms on a new drug could be extended for up to five years beyond the usual patent term.

Congress also noted the fact that the patent law could pose an impediment to competing companies wishing to pursue FDA applications for competing products to be marketed after the original patent expires. The legislation accordingly gives permission for competitors, even during the time when the patent is still in force, to use or even sell the patented invention in "uses reasonably related" to the process of obtaining information for federal regulatory approval of the competing product. In this way, the competitor could be ready in theory to bring his competing product to market as soon as the initial drug patent had expired. In a 1990 case heard by the United States Supreme Court,[11] the court applied much of this regulatory scheme not only to pharmaceuticals, but also to a medical device, a ventricular defibrillator.

Many intellectual discoveries, of course, build to some degree on earlier work, and the important question of public policy is what degree of protection to afford these incremental advances. This issue frequently arises in the case of patents, but also at times with regard to other forms of intellectual property. In another case heard in 1990 by the Supreme Court, for example, the justices considered how to define the relative rights of the author of a story and the producers of a motion picture based on the story.[12] The Supreme Court in both these recent decisions engaged in lengthy attempts to interpret statutory language that some of the justices themselves found murky. However, the important underlying policy issue in both cases was how to balance the rights of the creator of an initial piece of intellectual property with the rights of others who sought to exploit further some of the same material. The underlying paradox here reflects the fact that to encourage innovation in general, the innovator is rewarded with a potentially lucrative monopoly for a period of time, even though that protection may remove some incentives for competitors to make additional innovations.

This whole scheme of encouragement of innovation is based on the balance of potential costs of product development and the potential gains to be obtained. In the area of copyright law, the financial investment is often relatively small, as in the case of a writer whose main investment in a creative work may be his time. However, it may be considerably larger in the case of an expensive production, such as a motion picture. The costs of commercial development of a patent are often considerable, and as noted above, in the case of pharmaceutical development the costs may be immense. As a result, patent production is likely to be more of an inducement to drug development and marketing where the potential economic gain is foreseen in a large potential market for a new drug product.

To encourage the development of drugs for which the potential market is more limited, the Orphan Drug Act of 1983 offers a company a seven-year monopoly on the marketing of a drug that would serve less than 200,000 patients. Rights granted under the Orphan Drug Act potentially may conflict with those provided under the patent law, as is illustrated by recent litigation between two companies trying to market erythropoietin.[13]

It seems that both patent protection and Orphan Drug Act protection may be available not only for a totally new pharmaceutical, but also for a new use by a new route of an older drug. This is illustrated by the diuretic agent amiloride, which has found recent application in the nebulized form for treatment of cystic fibrosis, under the apparent protection of both the patent law[14] and the orphan drug statute.[15]

Congress revisited the question of orphan drug protection in its consideration of the orphan drug amendments of 1990. These amendments were designed to limit the scope of the exclusivity in the case of drug products that were later shown to have a broader patient market than was at first apparent. It also contained provisions for shared exclusivity in the case of two drug developers in certain circumstances. The bill was passed by Congress but failed to become law because of the disapproval (by pocket veto) of President Bush. The President noted in his disapproval message his concern that a change in the rules might diminish the strength of the incentives provided by the Orphan Drug Act for orphan drug development.[16]

In conclusion, Federal legislation has created incentives for drug development in the form of a protected market for new products. The scope of pharmaceutical innovations that are protected under American patent law is quite broad. Finally, even beyond the protection afforded to drug development under the patent laws, the Orphan Drug Act of 1983 has created an additional set of rules to encourage the development and marketing of drugs for which the anticipated market is relatively limited. There is a real tension between the public policy goals of encouraging pharmaceutical innovation on the one hand and using competition on the other to keep drug costs down, reflected most recently in the abortive 1990 amendments to the Orphan Drug Act.

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1. Merz B: Drug R&D costs doubled in decade. *American Medical News,* May 18, 1990, p 3.
2. Freudenheim M: The drug makers' defense on costs. *NY Times,* May 14, 1991, p 2.
3. Markoff J: More growth for Lilly as a new drug stars. *NY Times,* July 17, 1990, p d-1.
4. Rosenthal E: For more drugs, first test is abroad. *NY Times,* August 7, 1990, p c-1.
5. Smith T: Policies on drugs in the new Europe. *BMJ* 1990; 290:1476–1477.
6. United States Constitution, Article 1, Section 8.
7. Nusbaum NJ: Cooperation, not compulsion, in distribution of research materials. *Cell* 1989; 56:729–730.
8. Grubb PW: *Patents in chemistry and biotechnology.* Clarendon Press, Oxford 1986, pp 146–49.
9. Harrington WJ, Ahn YS, Mylvaganam R: Method of treating sickle cell anemia with danazol. US patent number 4,835,146, patent date May 30, 1989.
10. Kessler DA: The regulation of investigational drugs. *N Engl J Med* 1989; 320:281–288.
11. *Eli Lilly & Company v Medtronic Co. Inc.* 110 S Ct. 2683 (1990).
12. Stewart V. Abend, 110 S. Ct. 1750 (1990).
13. Andrews EL: Kidney drug setback for Genetics Institute. *NY Times,* January 15, 1991, p d-4.
14. Knowles MR, Church NL, Waltner WE, Yankaskas JR, et al: A pilot study of aerosolized amiloride for the treatment of lung disease in cystic fibrosis. *N Engl J Med* 1990; 332:1189–94.
15. Senator Hatch: *Congressional Record–Senate,* 1990, (Oct 12): S15213.
16. Memorandum of Disapproval for the Orphan Drug Amendments of 1990, November 8, 1990, *Weekly Comp Pres Doc* 1990: 26:1796–1797.

The continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria

Paroxysmal nocturnal hemoglobinuria (PNH) was first reported by Strubing in 1882.[1,2] The prognosis is usually poor with a short survival.[3] The commonest cause of mortality in reported series is intra-abdominal venous thrombosis,[4–6] and death usually results from complications of the thrombotic obstruction.[7–9]

To date, many attempts at treating the life-threatening thrombosis have been reported, including heparinization,[10,11] long-term coumadinization,[5,6] surgery,[12] streptokinase,[13] urokinase,[14] angioplasty,[15] and stent placement.[16] Although these treatments have met with some success, none have been entirely satisfactory in preventing progression of the Budd-Chiari syndrome, which often results in death.

Streptokinase and urokinase have been shown to be superior to heparin in resolving, by way of dissolution, deep venous thromboses.[17] During the 1970s, thrombolytic therapy with these agents became widespread.[18] Certainly, thrombolytic therapy has offered improvement in the treatment of Budd-Chiari syndrome;[13] however, the treatment of Budd-Chiari syndrome even with these agents is not always successful.[14] Heparin continues to be of value in the treatment and prevention of thrombus formation in patients with PNH.

Recently, there have been many advances in the field of thrombolytic therapy. Recombinant tissue-plasminogen activator (rt-PA) has risen to the forefront in the treatment of myocardial infarction. Thrombolytic therapy has contributed to increased survival with less morbidity in myocardial infarction and has now become the "standard of care" for the early treatment of anterior myocardial infarction. rt-PA has the advantage of not sensitizing the patient to subsequent exposure, which is an improvement over streptokinase.

There are theoretical advantages to the use of rt-PA, as pointed out by Kwan and Hansard in this issue of the *Journal*;[19] rt-PA has a short half-life of about five minutes, allowing rapid reversal in case of bleeding.[20] The use of rt-PA has not been previously reported in the treatment of Budd-Chiari syndrome. In this issue of the *Journal*, Kwan and Hansard describe transient improvement following treatment with rt-PA.[19] Although the patient died, illustrating the refractory nature of Budd-Chiari syndrome, further investigation of the use of rt-PA in this situation is clearly warranted.

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REFERENCES

1. Strubing P: Paroxysmalehaemoglobinurie. *Dtsch Med Wochenschr* 1882; 8:1–17.
2. Crosby WH: Paroxysmal nocturnal hemoglobinuria: a classic description by Paul Strubing in 1882, and bibliography of the disease. *Blood* 1951; 6:270–284.
3. Dacie JV: Paroxysmal noctural hemoglobinuria. *Proc R Soc Med* 1963; 56:587–596.
4. Hartmann RC, Luther AS, Jenkins DE Jr, et al: Fulminant hepatic venous thrombosis (Budd-Chiari syndrome) in paroxysmal nocturnal hemoglobinuria: definition of a medical emergency. *Johns Hopkins Med J* 1980; 146:247–254.
5. Leibowitz AI, Hartmann RC: Annotation: The Budd-Chiari syndrome and paroxysmal nocturnal hemoglobinuria. *Br J Haematol* 1981; 48:1–6.
6. Peytremann R, Rhodes RS, Hartmann RC: Thrombosis in paroxysmal nocturnal hemoglobinuria (PNH) with particular reference to progressive diffuse hepatic venous thrombosis. *Ser Haematol* 1972; 5:115–136.
7. Bell WR, Spitz R: Paroxysmal nocturnal hemoglobinuria. *Johns Hopkins Med J* 1978; 142:218–223.
8. Zimmerman D, Bell WR: Venous thrombosis and splenic rupture in paroxysmal nocturnal hemoglobinuria. *Am J Med* 1980; 68:275–279.
9. Blum SF, Gardner FH: Intestinal infarction in paroxysmal nocturnal hemoglobinuria. *N Engl J Med* 1966; 274:1137–1138.
10. Rosse WF: Paroxysmal nocturnal hemoglobinuria, in Brain MC, McCulloch PB (eds): *Current Therapy in Hematology-Oncology* 1983–1984. St Louis, Mo, CV Mosby, 1983, pp 41–44.
11. Hartmann RC, Kolhouse JF: Viewpoints on the management of paroxysmal nocturnal hemoglobinuria (PNH). *Ser Haematol* 1972; 5:42–60.
12. Orloff MJ, Johansen KH: Treatment of Budd-chiari syndrome by side-to-side portacaval shunt: experimental and clinical results. *Ann Surg* 1978; 188:494–512.
13. Sholar PW, Bell WR: Thrombolytic therapy for inferior vena cava thrombosis in paroxysmal nocturnal hemoglobinuria. *Ann Intern Med* 1982; 60: 20–23.
14. Greenwood LH, Yrizarry JM, Hallett JW Jr, et al: Urokinase treatment of Budd-Chiari syndrome. *Am J Rad* 1983; 141:1057–1059.
15. Ilan Y, Oren R, Shouval D: Postpartum Budd-Chiari syndrome with prolonged hypercoagulability state. *Am J Obstet Gynecol* 1990; 162:1164–1165.
16. Weernink EEM, Huisman AB, Ten Napel CHH: Treatment of Budd-Chiari syndrome by insertion of wall-stent in hepatic vein. *Lancet* 1991; 338:644.
17. Elliot MS, Immelman EJ, Jeffrey P, et al: A comparative randomized trial of heparin versus streptokinase in the treatment of acute proximal venous thrombosis: an interim report of a prospective trial. *Br J Surg* 1979; 66:838–843.
18. Bell WR, Meek AG: Guidelines for the use of thrombolytic agents. *N Engl J Med* 1979; 301:1266–1270.
19. Kwan T, Hansard P: Recombinant tissue-plasminogen activator for acute Budd-Chiari syndrome secondary to paroxysmal nocturnal hemoglobinuria. *NY State J Med* 1992; 92:102–104.
20. Tissue-type plasminogen activator for acute coronary thrombosis. *The Medical Letter* 1987; 29:107–109.

Is the morphology of ventricular premature beats a marker for left ventricular dysfunction?

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ABSTRACT. The morphology of ventricular premature beats has been described as a marker for the presence or absence of myocardial disease. Furthermore, the premature beat has been reported to be a potential marker for a dilated and hypokinetic left ventricle.

To verify this previously tested hypothesis, 37 healthy patients with ventricular premature beats on an electrocardiogram (ECG), Holter monitor ECG, or a stress test ECG were classified according to the ventricular premature beat morphology. Group 1 had ventricular premature beat QRS complexes with a smooth contour or with narrow (<40 msec) notching. Group 2 had ventricular premature beats with broad (>40 msec) notching or shelves. All of these patients had normal or borderline normal ECGs and normal multiple-gated acquisition (MUGA) scans. Nine patients had type I ventricular premature beats, 20 patients had type II ventricular premature beats, and eight patients had both type I and type II ventricular premature beats. We conclude that the presence of group 2 ventricular premature beats is so frequent in patients with normal left ventricular ejection fractions that the use of this marker in identifying abnormal left ventricular function is suspect.

(NY State J Med 1992; 92:89–91)

The morphologic appearance of ventricular premature beats have been described as a marker for the presence or absence of myocardial disease. Soloff[1] found that ventricular premature beats with a distorted appearance were highly suggestive of cardiac disease, whereas those beats with a smooth and undistorted appearance indicated a healthy heart. Similarly, Scherf and Schott[2] noted that ventricular premature beats with very wide QRS complexes on the EGG were usually seen in cardiac disease.

Recently, Moulton et al[3] reported that broadly notched ventricular premature beats of greater than 160 msec duration are reliable markers for a dilated and globally hypokinetic left ventricle, whereas a ventricular premature beat with a smooth contour or narrow notching and with a duration of less than 160 msec reflects a normal size heart with normal or near normal systolic function.

The purpose of this study was to ascertain the morphologic appearance of ventricular premature beats in a healthy population. If broadly notched ventricular premature beats of greater than 160 msec duration were frequently seen in this population, their presence in a population with cardiac disease would probably not serve as a marker for normal or abnormal systolic function.

METHODS

Normal MUGA scans, obtained from April 1989 to June 1990, were reviewed in 550 consecutive patients. The Siemans ZLC 7500S gamma camera was used. The 45° left anterior oblique and anterior views were obtained in all patients. In selective cases, a lateral view was also obtained. The corresponding 12-lead ECGs were also reviewed for each patient. If the ECGs were normal or showed nonspecific ST-T wave changes, they were also analyzed for ventricular premature beats. In addition, Holter monitor ECGs and stress tests were reviewed in these patients for the presence of ventricular premature beats. The Holter monitors were ordered because either the clinicians had detected premature beats on physical examination or the patient had complained of palpitations. The ECGs, Holter monitor ECGs, and stress tests were obtained within three months of the performance of the MUGA scans. Thus, this was a retrospective study.

The ventricular premature beats were classified into two groups on the basis of the morphology described by Moulton et al.[3] Group 1 consisted of patients having classic ventricular premature beat QRS complexes with either a narrow and uninterrupted contour or with narrow (<40 msec) notches. Group 2 ventricular premature beat QRS complexes contained wide (>40 msec) notches or shelves. A shelf was defined as a horizontal interruption in the inscription of the ventricular premature beat QRS complex (Fig 1).

RESULTS

Evaluation of 550 normal MUGA scans revealed 129 patients with normal ECGs and 95 patients with nonspecific ST-T wave abnormalities. Review of the ECGs of these 224 patients, as well as Holter monitor ECGs and stress test ECGs, produced 37 patients with one or more ventricular premature beats. Of these patients, 21 had normal ECGs, and 16 had nonspecific ST-T wave abnormalities.

These 37 patients ranged in age from 32–93 years and had ejection fractions from 47%–81%. All of these patients had normal contractility on the MUGA scan and normal cardiac size (Table I). Ventricular premature beats were found by Holter monitor in 24 patients, by ECGs in 12 patients, and by stress tests in five patients. In three patients, ventricular premature beats were found on the Holter monitor and the ECG, and in one patient they were noted on both the ECG and the stress tests.

Nine patients had type I ventricular premature beats, 20 patients had type II ventricular premature beats, and eight patients had both type I and type II ventricular premature beats (Table II). All of the type I ventricular premature beats were less than 160 msec, and all of the type II ventricular premature beats were greater than 160 msec. Twelve patients had ventricular premature beats on their ECGs; four patients had type I ventricular premature beats, five had type II ventricular premature beats, and three patients had both type I and type II ventricular premature beats.

A statistical analysis was performed comparing the nine patients who only had type I ventricular premature beats and the 28 patients who had type II ventricular premature beats. A two population variance F test was used. The F value was 1.99, which was significant ($p < 0.05$).

It appears unlikely that a sampling error explains these results. The type II class of ventricular premature beat was the most frequently observed in this healthy population.

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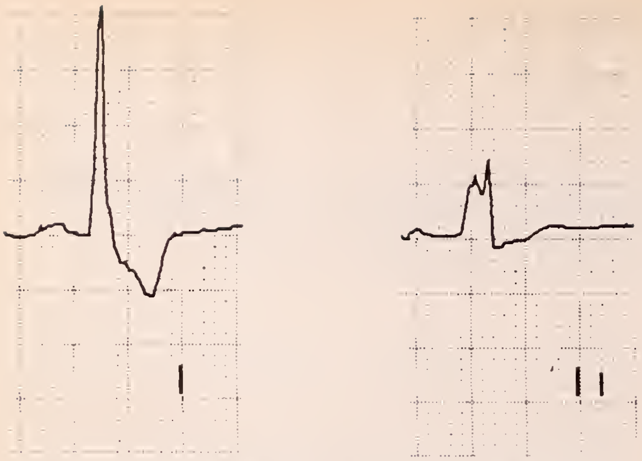

FIGURE 1. Representative type I and type II ventricular premature beats.

TABLE II. Types of Ventricular Premature Beats in the 37 Patients

Ejection Fraction (%)	Ventricular Premature Beats Type I	Type II	Both
47–50	—	3	—
51–60	2	3	4
61–70	6	4	2
71–81	1	10	2
Total	9	20	8

DISCUSSION

All of these patients had normal MUGA scans and normal or borderline normal ECGs. Both the smooth contour of ventricular premature beats and the notched or shelved type of ventricular premature beats were found in this population. Furthermore, in some patients both types of ventricular premature beats were found. Thus, the morphology of the ventricular premature beats cannot distinguish between the presence or absence of underlying cardiac disease.

All of the patients studied in Moulton et al[3] recent study had cardiac disease. The investigators also found both type I and type II ventricular premature beats in their study population. They also concluded that the morphology of the ventricular premature beats cannot discriminate between the presence or absence of heart disease. However, they did state that a broadly notched ventricular premature beat of long duration is a reliable marker for a dilated, globally hypokinetic, left ventricle.

In the healthy population of patients described here, the type II or broadly notched ventricular premature beat was

TABLE I. Ejection Fractions and Ventricular Premature Beats in 37 Patients

Age (yr)	Sex	Ejection Fraction (%)	Holter Monitor*	ECG	Stress Test	Ventricular Premature Beats Type I	Type II
76	F	47	+	−	−	−	+
64	M	49	−	+	−	−	+
53	F	50	+	−	−	−	+
74	F	52	+	+	−	+	+
47	M	52	−	+	−	+	−
63	M	52	−	+	+	+	+
63	M	52	+	+	−	+	+
36	F	53	+	−	−	−	+
54	M	54	+	−	−	−	+
56	F	55	−	−	+	+	−
62	M	56	+	−	−	+	+
63	M	60	−	−	+	−	+
55	M	61	−	−	+	+	+
78	F	61	−	+	−	+	−
82	F	62	−	+	−	−	+
93	M	63	+	−	−	+	−
80	F	64	+	−	−	−	+
49	F	65	+	−	−	+	−
51	M	65	−	−	+	+	−
74	F	67	+	−	−	+	−
68	F	68	−	+	−	+	−
69	F	69	+	−	−	−	+
89	M	70	+	−	−	+	+
57	M	70	+	−	−	−	+
67	F	71	+	−	−	−	+
35	M	72	+	−	−	−	+
72	F	72	−	+	−	−	+
74	F	72	+	−	−	−	+
84	F	73	+	−	−	+	+
55	M	73	+	−	−	−	+
89	F	73	−	+	−	+	−
73	M	75	+	−	−	−	+
88	F	76	−	+	−	−	+
32	M	79	+	−	−	−	+
82	F	79	+	−	−	+	+
81	F	80	+	−	−	−	+
82	F	81	+	+	−	−	+

*+ indicates positive for ventricular premature contractions; − indicates negative for ventricular premature contractions.

found more frequently than the smooth or type I ventricular premature beat. Thus, the type II ventricular premature beat clearly is not a reliable marker for a dilated, hypokinetic, left ventricle. However, there may be possible explanation for the different results in our study and Moulton et al's. Perhaps in the healthy population, the ventricular premature beats originated from the right ventricular outflow tract, had a left bundle branch block (LBBB) pattern, and required a longer conduction time to activate the left ventricle. Among the diseased hearts studied by Moulton et al, the ventricular premature beats presumably originated more frequently in the left ventricle. They could have been of either a right bundle branch block (RBBB) pattern, originating from the free wall, or a LBBB pattern, originating from the septum.

In addition, only 12-lead ECGs were analyzed in Moulton et al's study. In this investigation, both Holter monitor ECGs and 12-lead ECGs were used. The Holter monitor may have technical difficulties in reproducing high frequency events. Thus, high-frequency notching (<40 msec) could be slowed into the (>40 msec) broad notched group.

In our study, however, 12 patients had ventricular premature beats on their ECGs, and the type II ventricular premature beats were found in eight of these patients. Thus, this smaller group again showed the frequent occurrence of type II ventricular premature beats.

In conclusion, the presence of Group 2 ventricular premature beats is so frequent in patients with normal left ventricular ejection fractions that the use of this marker in identifying abnormal left ventricular function is suspect.

References

1. Soloff LA: Ventricular premature beats diagnostic of myocardial disease. *Am J Med Sci* 1961; 242:315–319.
2. Scherf D, Schott A: *Extrasystoles and Allied Arrhythmias.* New York, Grune and Stratton, Inc, 1953, p 30.
3. Moulton KP, Medcalf T, Lazzara R: Premature ventricular complex morphology. a marker for left ventricular structure and function. *Circulation* 1990; 81:1245–1251.

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THE TREATMENT OF FAILING COMPENSATION IN CHRONIC VALVULAR DISEASE OF THE HEART

Although largely a question of mechanics the study of the deranged vascular mechanism cannot be accurately prosecuted without full consideration of the factors of nutrition, resistance, heredity and the personal equation which embraces more than all of these elements, the actual vital possession which has been developed through both heredity and environment. Let us remember in considering the treatment of chronic valvular disease of the heart that the first act performed by the circulatory wave is the provision made for the nourishment of the heart muscle and that as long as the heart is enabled to care for its own nutrition just so long is it possible for it to support the extra muscular development which constitutes compensatory cardiac hypertrophy. The slightest degree of coronary insufficiency is followed by corresponding failure in the heart's power to overcome the effects of an obstructing or leaking valvular fold. It is the presence of this bar to the circulation, or the abnormal aperture caused by the distortion of the valvular folds, that gives rise to the adventitious sounds we designate murmurs, the essential diagnostic elements. The mere detection of a murmur however gives us little or no insight into the effects of the lesion on either heart wall or general circulation. It is the reaction of the heart's muscle to the strain thrown on it by the lesion that forecasts the history of the patient, weighed, of course, by the aggregate of clinical experience; and, too, it is this reaction which should guide the physician in his care and general management of the case.

W. M. GIBSON, MD
(*NY State J Med* 1910; 10:69–73)

REVIEW ARTICLE

Update in epilepsy

Part III: Surgical therapy of intractable epilepsy

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Advances in our understanding of the pathophysiology and response to therapy of the epilepsies, along with the development of regional comprehensive epilepsy programs (CEP), have stimulated increased use of surgical therapies for these disorders.[1] The future role of that therapy was the theme of the recent National Institutes of Health Consensus Development Conference on Epilepsy Surgery[2] in which it was suggested that not only was the outcome of surgery in selected patients superior to that of medical therapy, but that the capacity to perform these operations would be expanded nationwide. The need for accurate, early identification and operative treatment of pediatric patients was particularly emphasized, since this age group appears to benefit most from relief from the seizures which interfere with normal cognitive, psychologic, and adaptational development.[3–5]

EPIDEMIOLOGY

The prevalence of chronic epilepsy is estimated to be 0.5%–1%.[6,7] Although medical therapy renders 60%–70% of all epileptics seizure-free,[7] subsets of patients with complex partial and generalized tonic-clonic seizures respond less well to medical intervention.[1,7–9] Approximately 40% of all epileptics are afflicted specifically with complex partial seizures,[10] of which 60%[8] are medically intractable.[11,12] As many as 200,000 of this group alone may be candidates for surgical therapy.[4,13] At the current national rate of 800–1,000 surgical cases per year, these patients are vastly underserved by existing resources. In the past, many of these patients were managed unsuccessfully for decades, and surgical referral was not contemplated. It is now accepted that the success rate of optimal medical therapy declines exponentially after one year.[14] Patients with persistent seizures after 1–2 years of aggressive antiepileptic drug (AED) therapy are "medically intractable" and merit evaluation as candidates for surgical therapy.

MORBIDITY OF INTRACTABLE EPILEPSY

The morbid sequelae of intractable epilepsy encompass the effects of both persistent seizures and AED administration. Unfavorable manifestations associated with significant, persistent seizures include reduced life expectancy, attributable to sudden death,[15,16] accidents,[17] and suicide,[18] diminished cognitive,[19–22] psychosocial, and vocational measures,[19,23–26] and the risk of spreading of the epileptic abnormality[27] or kindling.[28] Untoward effects of AED administration, especially polytherapy, include sedation, diminished cognitive and behavioral function, and myriad adverse drug reactions, including allergies and idiosyncratic effects.[9,29,30] It is believed that many of these deleterious effects may be arrested or even reversed in patients who are seizure-free after surgery.[31]

LIMITATIONS OF MEDICAL MANAGEMENT

Although over all epilepsy is a relatively benign condition, with the majority of patients responding to AED therapy, there is a subset of patients with a very intractable disease. This intractable nature is very evident. In a recent study conducted at the University of Washington Regional Epilepsy Center,[32] where the outcome of five or ten years of medical therapy was determined in 26 patients with complex partial seizures who satisfied the criteria for surgical therapy but elected, instead, aggressive medical therapy with primary and experimental AEDs, these patients had an average pretreatment seizure history of 19.3 years. After five or ten more years of therapy, none of these patients were seizure-free, and only one had significantly improved regarding the seizure control interval. In a comparable surgically treated population, 68% were seizure-free, and 7% were significantly improved five or ten years postoperatively. Thus, the most aggressive medical therapy was unsuccessful in a cohort of patients in whom we would otherwise anticipate a favorable outcome after epilepsy surgery.

PREOPERATIVE EVALUATION

With the goal of therapy a seizure-free patient with no new functional deficits, initial evaluation focuses on establishing each patient's candidacy for the surgical procedure most likely to achieve that goal, a resective operation. The *sine qua non* of resective surgery is the identification and surgical removal of the epileptogenic zone, defined as the brain region responsible for the generation and propagation of seizures.[13] If a discrete epileptogenic zone cannot be identified, or if resection is contraindicated due to involve-

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Supported by NIH grant NS17111 and sponsored in part with a grant by New Medico Health Care System.

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ment of essential cortex, corpus callosotomy may be considered.[1,33]

Patient Selection

Patients entered into preoperative evaluation must demonstrate medical intractability;[14] reduced quality of life because of recurrent seizures and/or AED effects; identification of a single zone of seizure onset, based on convergence of evidence including electroencephalic (EEG) abnormalities; evidence that excision of this epileptogenic zone is unlikely to result in a new neurologic deficit; an understanding of the risk and benefits of surgery; and adequate support systems to facilitate postoperative recovery and community reentry.[34,35] In the realm of relative exclusionary criteria, patients harboring significant psychiatric or progressive degenerative or metabolic central nervous system disease or who are severely retarded are less optional candidates for surgical therapy.[35] About 80% of patients meeting these widely accepted criteria will have an epileptogenic zone in one temporal lobe. A wide variety of approaches to the identification of the epileptogenic zone and the subsequent resection have been developed for those patients. These different approaches to temporal lobe resection have been discussed in detail previously.[36,37] Here we present the authors' present preoperative and intraoperative approach, as one suitable for all patients considered for resective surgery. However, the intraoperative technique used has an impact on how extensive the preoperative evaluation needs to be, so that a different preoperative evaluation might be necessary for a different intraoperative approach.

Identification of the Epileptogenic Zone

The key to a successful resective operation is the accurate localization of the epileptogenic zone because its complete surgical removal correlates with a favorable postoperative seizure outcome.[38] The most important localizing information comes from EEG studies. Other investigations provide confirmatory data. These include imaging studies, neuropsychological tests, and video analysis of seizure semiology. Ideally, the data from all studies are "convergent" and point to a single, restricted region of abnormal cortex. Complex patterns of epileptogenicity or "non-convergence" of data in initial studies requires that more extensive studies be undertaken.

Phase I of our recommended evaluation consists of serial, outpatient EEG studies (interictal EEG) conducted over a minimum period of 6–12 months. If EEG abnormalities are simple, focal, and consistent, and if gadolinium-enhanced magnetic resonance imaging (MRI) scans are either normal or reveal a structural abnormality in the same location as the EEG changes (convergent data), the patient may be referred for surgery without inpatient video-EEG analysis. If EEG abnormalities are widespread or bilateral or if imaging abnormalities do not coincide with focal EEG changes (non-convergent data), Phase II evaluation is recommended. In this evaluation, patients are admitted to the CEP for 1–2 weeks and the EEG and behavioral manifestations of their seizures are examined, using scalp electrodes and continuous video-EEG recordings. AEDs may be withdrawn to increase the yield of seizures. Those patients in whom a localized site of ictal onset preceding clinical seizure onset is identified that is consistent with other findings are referred for surgery. If not, patients are referred for Phase III evaluation that involves readmission to the CEP for 1–2 weeks, and the surgical placement of intracranial recording electrodes in the area of suspected ictal onset.[36,37]

Imaging Modalities

Computed tomography and MRI scans have been valuable in identifying structural lesions, including cortical atrophy, malformations, and tumors, that are usually, but not always, within or adjacent to the epileptogenic zone.[1] More recently, computer-assisted MRI hippocampal volumetric analysis has been used to identify hippocampal atrophy,[39,40] frequently correlating with the side of seizure onset. In temporal lobe epilepsy, positron-emission tomography[41,42] scans identify areas of widespread interictal hypometabolism and single-photon-emission computed tomography scans[13,42,43] recorded during seizures have revealed local hyperperfusion, in either case aiding in predicting the side of seizure onset.

Neuropsychological Assessment

Our neuropsychological evaluation addresses cognitive and psychosocial indexes that are particularly sensitive to problems encountered by epileptics.[44,45] This battery has been useful in the evaluation of the cognitive and psychosocial effects of epilepsy surgery and also in the identification of factors that predict a favorable outcome after surgery.[46] This assessment is also valuable in identifying patients in whom surgery may be contraindicated, including those with psychiatric disorders, psychogenic seizures, and severe mental retardation. Abnormalities that can be localized to one brain area are also used by some centers to provide confirmatory evidence with respect to the location of the epileptogenic zone.

The intracarotid sodium amytal procedure[47,48] or Wada test[49] is performed preoperatively on all patients with intractable temporal lobe epilepsy. This test identifies the language dominant hemisphere and evaulates the ability of each temporal lobe to sustain short-term memory independently.[50] Because the neurodegenerative process of mesial temporal sclerosis is often bilateral, the temporal lobe in which seizures arise may sometimes be the only temporal lobe capable of independently sustaining memory. In such patients, a standard temporal lobectomy[51] or an amygdalohippocampectomy[52] could produce a permanent and disabling global memory deficit. Therefore, if the Wada test suggests a risk for memory impairment, the extent of medial, as well as lateral temporal resection, must be modified.[53,54]

Invasive Monitoring Techniques

Surgically implanted electrodes are occasionally required to localize the epileptogenic zone and define its relationship to essential cortical areas. The proportion of cases evaluated in this way varies greatly between centers; in these investigators' approach to resective surgery, about 25% of cases require intracranial recordings. Under general anesthesia, subdural strip electrodes are placed over the surface of the brain[36] or, alternatively, depth electrodes are inserted directly into the brain to acquire recordings from deeper structures.[37,55] The placement of depth electrodes is associated with a 1%–2% risk of permanent neurologic complications.[56–58] We prefer to use subdural electrodes, whenever possible, associated with a lower risk and a lower cost to the patient. Subdural grid electrodes cover extensive regions of the brain surface and facilitate identification of the site of seizure onset,[37,55] in extratemporal cases. These may also be used to map cortical functions using electrical stimulation techniques where this cannot be done intraoperatively because of lack of patient cooperation or where the relationship of the epileptogenic zone to essential cortex must be determined. Use of grids, however, approximately doubles the risk and cost of resective surgery.

SURGICAL APPROACHES

The goal of the resection is the removal of as much of the epileptogenic zone as possible without encroaching on "eloquent" areas related to motor and sensory functions and language and occasionally memory. The authors tailor the resection to the extent of epileptogenic zone in the individual patient as indicated by presence of interictal epileptiform abnormalities, and the eloquent areas as indicated by electrical stimulation mapping in that individual patient at time of surgery. Usually both the extent of interictal activity and location of eloquent areas are based on information acquired intraoperatively.

TEMPORAL LOBECTOMY

Technique of Tailored Resection. Most resections are performed with the patient awake, under local anesthesia and intravenous sedation.[37] This minimizes anesthetic effects on the electrocorticogram (ECOG) used to identify this epileptogenic zone and allows for mapping of language, if needed. After craniotomy and cortical exposure, an ECOG is performed with surface and subdural strip electrodes, to determine the full extent of the epileptogenic abnormalities as a guide to the extent of cortical resection. Motor and sensory cortex are identified by the application of small currents to the cortical surface, and in dominant hemisphere operations, language cortex[54,59] is identified through the interruption of object-naming by focal cortical stimulation. If the Wada Test suggests a risk to memory, memory is also mapped intraoperatively.[54] The regions of EEG abnormalities and essential cortex are marked on the brain surface with tickets and a "tailored resection" is designed that preserves essential brain and maximizes removal of the epileptogenic zone. After resection of lateral temporal cortex, ECOG recordings of mesial structures (eg, amygdala, hippocampus, parahippocampal gyrus) are performed and used to guide the extent of resection of mesial structures as well. The extent of the medial resection is reduced if preoperative evaluation has raised concerns about memory function.[50]

Perioperative Course. Patients are hospitalized for approximately one week after most resective operations. Postoperative recovery is usually uneventful except for transitory anomia in dominant hemisphere operations, modest wound tenderness, and mild fatigue. Rarely, complaints of word finding difficulty may persist for several months, with eventual resolution. Occasionally, "neighborhood seizures" from post-surgical cortical irritation may occur during the immediate postoperative period, but these do not necessarily portend a diminished long-term seizure outcome.[60] After discharge, patients may return immediately to most non-stressful activities at home and return to full-time work within one or two months. Our patients are maintained on their preoperative AEDs for at least one year postoperatively, and if they remain seizure-free, medications are tapered over several months and discontinued.

Seizure Outcome. Seizure outcome is affected by patient selection criteria, which may vary significantly between centers, and this must be considered when discussing the results of different approaches. In a 1987 survey[60] of 2,336 patients undergoing temporal lobectomy at 40 centers, 55% were "seizure-free," 28% were "improved," and 17% experienced minimal or no improvement. In our experience based on the situation in the fourth and fifth years after a temporal resection in 70 patients, 57% were "seizure-free," and 10% experienced more than a 75% reduction of seizures and less than five seizures yearly. Thus, 67% of our patients benefitted significantly from the operation with respect to the control of seizures.

Cognitive and Psychosocial Outcome. Postoperative global memory deficits[51,61] are extremely rare occurrences, since the advent of Wada testing, and most cognitive indexes, including summary IQ scores, are unchanged postoperatively.[62] After dominant temporal lobectomy, some patients may experience a modest, and usually transitory, decline in the performance of language-dependent cognitive tasks, particularly those related to verbal memory.[63] In patients who are rendered seizure-free, a measurable improvement in visual-spatial memory and performance IQ has been noted after left dominant temporal lobectomy.[31] Even greater improvements in verbal IQ and verbal memory occur after non-dominant lobectomy that is also associated with an improvement in overall IQ scores. Surgical therapy resulting in seizure relief is similarly associated with improvement in psychosocial status and in measures of social adjustment[64] one year after surgery.[31]

Surgical Complications. Infection (1%), infarction (1%), and postoperative hematoma (0.5%), occasionally resulting in permanent neurological deficits, constitute the major surgical complications of temporal lobe operations.[58,65] Most infections resolve with antibiotic therapy, but postoperative hematomas may require reoperation. Infarctions in the middle cerebral or anterior choroidal artery territories produce motor and sensory deficits and, in the dominant hemisphere, language deficits, which gradually resolve to a variable extent. Minor, subquadrantic visual field defects in the contralateral upper quadrant are not uncommon, and result from the interruption of optic radiation fibers during the course of anterior temporal lobe resection. These minor visual defects are seldom recognized either by the physician or by the patient without formal perimetry testing. Functionally significant visual field defects occur rarely (1%–2%) and may initially be troublesome for affected patients, who usually achieve adequate functional adaptation over several months.

EXTRATEMPORAL RESECTIONS

Over 20% of all resective operations involve removal of "extratemporal" (eg, frontal, parietal, occipital) cortex. Resection of an epileptogenic zone in frontal or parietal cortex when it impinges on the rolandic (motor/sensory) cortex presents special challenges relating to the precise localization of both the site of seizure onset and the regions of essential brain.[33] This often requires ictal monitoring with an implanted grid electrode to facilitate video-EEG seizure detection and functional brain mapping.[66] These patients do less well postoperatively than those undergoing temporal lobectomy, with 43% being "seizure-free" and 28% being "improved" in Engel's survey.[60]

MODIFIED (FUNCTIONAL) HEMISPHERECTOMY

Patients with hemiparesis and intractable seizures, as occur in the infantile hemiplegia syndrome, are candidates for a modified "functional" hemispherectomy, provided that fine finger movements and foot tapping are not present on the affected side.[33,67] In this operation the peri-rolandic and temporal lobe cortex are resected and the frontal and occipital lobes are disconnected via an incision in the white matter. Seventy-seven percent of this group experienced complete seizure relief postoperatively, in Engel's survey,[60] and many could be eventually withdrawn from AEDs completely. In those with complete seizure relief, an improvement of ten IQ points may be anticipated postoperatively. A diligent, ongoing, postoperative follow-up is mandatory in this group since delayed complications, including hydrocephalus and superficial hemosiderosis, may present as long as 5–10 years after surgery.

CORPUS CALLOSOTOMY

This procedure is an alternative for selected patients who are not candidates for resective surgery. As originally developed by van Wagenen at the University of Rochester,[68] this operation consisted of the complete surgical interruption of the corpus callosum. A more restricted anterior two-thirds callosotomy is now generally favored, which may diminish cognitive and psycho-behavioral complications associated with the original procedure. Patients who are considered for this operation include[69] those with frequent atonic (akinetic) seizures or drop attacks and those with secondarily generalized seizures arising from frontal areas or from within an unresectable (ie, essential brain region) epileptogenic zone. A preoperative Wada test to assure that language, dominant handedness, and verbal memory are ipsilateral is essential.[70] With proper patient selection, relief of atonic and generalized seizures can be quite dramatic; focal seizures are occasionally exacerbated, however, and the postoperative recovery may be more protracted than that associated with resective operations. In Engel's survey,[60] only 5% were "seizure-free," 71% were "improved," and 24% experienced minimal or no improvement.

PEDIATRIC EPILEPSY SURGERY

All physicians caring for patients with pediatric onset intractable epilepsy have witnessed the disheartening, progressive deterioration in cognitive and behavioral function that accompanies frequent intractable seizures and AED polytherapy.[24,71,72] The adult temporal lobectomy patients in our center report an average preoperative history of 19.3 years, usually beginning in childhood. These adults, if selected earlier, could have been spared the insult of recurrent seizures and AED effects throughout their important developmental years.[3,5] Postoperative seizure relief in adults appears to provide less benefit in terms of psychosocial, cognitive, and vocational improvement than that observed in children.[5] Children experience fewer problems related to adjustment, depression, or withdrawal, with 80% demonstrating educational or vocational progress postoperatively.[5] A major emphasis in the field of epilepsy surgery in the 1990s is to identify surgical candidates early in their course before the cognitive and psycho-behavioral deterioration has become irreversible.

BRAIN TUMORS AND EPILEPSY

Brain tumors such as gangliogliomas and low-grade glial neoplasms are often associated with seizures that are difficult to control. Removal of tumor alone may be associated with a 20%–30% rate of persistent seizures postoperatively. In selected patients, we perform tumor resections in combination with an intraoperative ECOG to identify the epileptogenic zone resected along with the tumor. In our recent series of 14 patients with ganglioglioma, we achieved a nearly 100% seizure-free rate at three-year follow-up. We suggest that patients with intractable seizures in association with brain tumors, particularly temporal lobe tumors, should be considered for a combined operation, including intraoperative ECOG evaluation and appropriate cortical resection.

Acknowledgments. The authors thank Ms Lee Dulin for her patient secretarial assistance, especially with manuscript revisions, and Ms Jill Andrews for obtaining reference articles in a timely fashion.

REFERENCES

1. Engel J Jr. Alternative therapy, in: Engel J Jr (ed). *Seizures and Epilepsy*. Philadelphia, FA Davis, 1989, pp 443–474.
2. NIH Consensus Conference: Surgery for epilepsy. *JAMA* 1990; 264:729–733.
3. Goldring S: Pediatric epilepsy surgery. *Epilepsia* 1987; 28(suppl1):S82–S102.
4. Henriksen O: Clinical aspects in children. *Acta Neurol Scand* (suppl) 1988; 117:47–51.
5. Mizrahi EM, Kellaway P. Grossman RG, et al: Anterior temporal lobectomy and medically refractory temporal lobe epilepsy of childhood. *Epilepsia* 1990; 31:302–312.
6. Hauser WA, Kurland LT: The epidemiology of epilepsy in Rochester, Minnesota, 1935 through 1967. *Epilepsia* 1975; 16:1–66.
7. Elwes RD, Johnson AL, Shorvon SD, et al: The prognosis for seizure control in newly diagnosed epilepsy. *N Engl J Med* 1984; 311:944–947.
8. Rodin EA: *The Prognosis of Patients with Epilepsy*. Springfield, Illinois, CC Thomas, 1968.
9. Mattson RH, Cramer JA, Collins JF, et al: Comparison of carbamazepine, phenobarbital, phenytoin and primidone in partial and secondarily generalized tonic-clonic seizures. *N Engl J Med* 1985; 313:145–151.
10. Gastaut H, Gastaut JL, Convales e Silva GE, et al: Relative frequency of different types of epilepsy: a study employing the classification of the International League Against Epilepsy. *Epilepsia* 1975; 16:457–461.
11. Dreifuss FE: Goals of surgery for epilepsy, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 31–49.
12. Schomer DL: Current concept in neurology. partial epilepsy. *N Engl J Med* 1983; 309:536–539.
13. Engel J Jr: *Diagnostic Evaluation in Seizure and Epilepsy*. FA Davis, Philadelphia 1989, pp 303–335.
14. Leppik IE: Intractable epilepsy in adults. NIH Consensus Development Conference: *J Epilepsy Res* 1991 (in press).
15. Schraeder PL, Lathers CM: Paroxysmal autonomic dysfunction, epileptogenic activity and sudden death. *Epilepsy Res* 1989; 3:55–62.
16. Jay GW, Leestma JE: Sudden death in epilepsy. a comprehensive review of the literature and proposed mechanisms. *Acta Neurol Scand* 1981; 82(suppl): 1–66.
17. Orlowski JP, Rothner AD, Luders H: Submersion accidents in children with epilepsy. *Am J Dis Child* 1982; 136:777–780.
18. Barraclough B: Suicide and epilepsy, in: *Epilepsy and Psychiatry*. London, Churchill-Livingstone, 1981, pp 72–76.
19. Lindsay J, Ounsted C, and Richards P: Long-term outcome in children with temporal lobe seizures. I: Social outcome and childhood factors. *Dev Med Child Neurol* 1979; 21: 285–298.
20. Dodrill CB, Troupin AS: Seizures and adaptive abilities. a case of identical twins. *Arch Neurol* 1976; 33:604–607.
21. Dodrill CB: Correlates of generalized tonic-clonic seizures with intellectual, neuropsychological, emotional and social function in patients with epilepsy. *Epilepsia* 1986; 27:399–411.
22. Trimble MR: Cognitive hazards of seizure disorder. *Epilepsia* 1988; 29(suppl1):S19–S24.
23. Glaser GH: Natural history of temporal lobe-limbic epilepsy, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 13–30.
24. Lesser RP, Luders H, Wyllie E et al: Mental deterioration in epilepsy. *Epilepsia* 1986, 27(suppl2):S105–S123.
25. Janz D: Neurological morbidity of severe epilepsy. *Epilepsia* 1988; 29(suppl1):SI–S8.
26. Thompson PJ, Oxley J: Socioeconomic accompaniments of severe epilepsy. *Epilepsia* 1988; 29(suppl1):S9–S18.
27. Dinner DS, Luders H., Rothner AD. et al: Complex partial seizures of childhood onset: a clinical and encephalographic study. *Cleve Clin Q* 1984; 51: 287–291.
28. Morrell F, Wada J, Engel J Jr: Appendix III: Potential relevance of kindling and secondary epileptogenesis in the consideration of surgical treatment of epilepsy, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 701–707.
29. Collaborative Group for Epidemiology of Epilepsy: Adverse reactions to

antiepileptic drugs: a follow-up study of 355 patients with chronic antiepileptic drug treatment. *Epilepsia* 1988; 29:787–793.

30. Dam M. Side-effects of drug treatment in epilepsy. *Acta Neurol Scand* 1988; 117(suppl):34–41.

31. Rausch R, Crandall PH: Psychological status related to surgical control of temporal lobe seizures. *Epilepsia* 1982; 23:191–202.

32. Ojemann LM: Natural history of drug-resistant seizures. clinical aspects. NIH Consensus Development Conference. *J Epilepsy Res* 1991 (in press).

33. Rasmussen T: Commentary. Extratemporal cortical excision and hemispherectory, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 417–424.

34. Andermann F: Identification of candidates for surgical treatment of epilepsy, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 51–70.

35. Gumnit RJ: Selection of adult patients for surgical treatment of epilepsy. *Acta Neurol Scand Suppl* 1988; 117:42–46.

36. Ojemann, GA, Engel J Jr: Acute and chronic intracranial recording and stimulation, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 263–288.

37. Ojemann GA: Surgical therapy for medically intractable epilepsy. *J Neurosurg* 1987; 66:489–499.

38. Wyllie E. Luders H, Morris HH III, et al: Clinical outcome after complete or partial cortical resection for intractable epilepsy. *Neurology* 1987; 37:1634–1641.

39. Cascino GD, Hirschorn KA, Jack CR, et al: Gadolinium-DTPA-enhanced magnetic resonance imaging in intractable partial epilepsy. *Neurology* 1989; 39:1115–1118.

40. Sharbrough FW, Jack CR, Cascino GD, et al: Presurgical quantitative magnetic resonance-based hippocampal volume measurements in patients undergoing temporal lobectomy for medically intractable seizures. *Epilepsia* 1989; 30:675.

41. Engel J Jr, Brown WJ, Kuhl DE, et al: Pathological findings underlying focal temporal lobe hypometabolism in partial epilepsy. *Ann Neurol* 1982; 12: 518–528.

42. Engel J Jr: The role of neuroimaging in the surgical treatment of epilepsy. *Acta Neurol Scand Suppl* 1988; 117:84–89.

43. Rowe CC, Berkovic SF, Sia ST, et al: Localization of epileptic foci with postictal single photon emission computed tomography. *Ann Neurol* 1989; 26: 660–668.

44. Dodrill CB: A neuropsychological battery for epilepsy. *Epilepsia* 1978; 19:611–623.

45. Dodrill CB: Commentary. psychological evaluation, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 51–70.

46. Dodrill CB, Wilkus RJ, Ojemann GA, et al: Multidisciplinary prediction of seizure relief from cortical resection surgery. *Ann Neurol* 1986; 20:2–12.

47. Milner B, Branch C, Rasmussen T: Study of short-term memory after intracarotid injecton of sodium amytal. *Trans Amer Neurol Ass* 1962; 87:224–226.

48. Blume WT, Grabow JD, Darle FL, et al: Intracarotid amobarbital test of language and memory before temporal lobectomy for seizure control. *Neurology* 1973; 23:812–819.

49. Wada J, Rasmussen T: Intracarotid injection of sodium amytal for the lateralization of cerebral speech dominance: experimental and clinical observations. *J Neurosurg* 1960; 17:226–282.

50. Rausch R: Psychological evaluation, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 181–195.

51. Penfield W, Mathieson G: Memory. autopsy findings and comments on the role of hippocampus in experiential recall. *Arch Neurol* 1974; 31:145–154.

52. Rausch R, Babb TL, Engel J Jr, et al: Memory following intracarotid amobarbital injection contralateral to hippocampal damage. *Arch Neurol* 1989; 46:783–788.

53. Ojemann GA, Dodrill CB: Verbal memory deficits after left temporal lobectomy for epilepsy. mechanism and intraoperative prediction. *J Neurosurg* 1985; 62:101–107.

54. Ojemann GA, Dodrill CB: Intraoperative techniques for reducing language and memory deficits with left temporal labectomy, in: Wolf P, Dam M, Janz D, Dreifuss FE (eds). *Advances in Epileptology, Vol. 16*. New York, Raven Press, 1987, pp 327–330.

55. Spencer SS: Depth versus subdural electrode studies for unlocalized epilepsy. *J Epilepsy* 1989; 2:123–127.

56. Cahan LD, Sutherling W, McCullough MA, et al: Review of the 20-year UCLA experience with surgery for epilepsy. *Cleve Clin Q* 1984; 51:313–318.

57. Rosenbaum TJ, Laxer KD: Subdural electrode recordings for seizure focus localization. *J Epilepsy* 1989; 2:129–135.

58. Pilcher WH, Ojemann GA: *Complications of Epilepsy Surgery*. New York, Thieme Medical Publishers, Inc. (in press).

59. Ojemann GA, Ojemann J, Lettich E, et al: Cortical language localization in left dominant hemisphere. an electrical stimulation mapping investigations in 117 patients. *J Neurosurg* 1989; 71:316–326.

60. Engel J Jr: Outcome with respect to epileptic seizures, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 553–571.

61. Milner B: Psychological defects produced by temporal lobe excision. *Res Pub Ass Res Nerv Ment Dis* 1958; 36:244–257.

62. Hermann BP, Wyler AR: Neuropsychological outcome of anterior temporal lobectomy. *J Epilepsy* 1988; 1:35–45.

63. Ivnik RJ, Sharbrough FW, Laws ER Jr: Effects of anterior temporal lobectomy on cognitive function. *J Clin Psych* 1987; 43:128–137.

64. Taylor DC: Psychiatric and social issues in measuring the input and outcome of epilepsy surgery, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 485–503.

65. Van Buren: Complications of surgical procedures in the diagnosis and treatment of epilepsy, in: Engel J Jr (ed): *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 465–475.

66. Luders H, Lesser RP, Dinner DS, et al: Commentary. chronic intracranial recording and stimulation with subdural electrodes, in: Engel J Jr (ed). *Surgical Treatment of the Epilepsies*. New York, Raven Press, 1987, pp 297–321.

67. Hendrick EB, Hoffman HJ, Hudson AR: Hemispherectomy in children. *Clin Neurosurg* 1969; 16:315–327.

68. Van Wagenen WP, Herren RY: Surgical division of commisural pathways in corpus callosum relation to spread of epileptic attack. *Arch Neurol Psychiat* 1940; 44:740–759.

69. Spencer SS: Corpus callosum section and other disconnection procedures for medically intractable epilepsy. *Epilepsia* 1988; 29(suppl2):S85–S99.

70. Novelly RA, Lifrak MD: Forebrain commissurotomy reinstates effects of preexisting hemisphere lesions; an examination of the hypothesis, in: *Epilepsy and the Corpus Callosum*. New York, Plenum Press, 1985, pp 467–500.

71. Chaudhry MR, Pond DA: Mental deterioration in epileptic children. *J Neurol Neurosurg Psychiat* 1961; 24:213–219.

72. Trimble MR, Cull CA: Antiepileptic drugs, cognitive function and behavior in children. *Cleve Clin J Med* 1988; 56(suppl1): S140–S146.

The non-psychiatric physician's responsibilities for the suicidal adolescent

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Suicidal behavior has traditionally been considered a problem for the psychiatrist, rather than for the pediatrician, internist, emergency room physician, or family practitioner. Few non-psychiatric physicians inquire about emotions or suicidal thoughts or feelings as a part of their routine health care examinations,[1] even though adolescents are more likely to die from suicide than any medical condition. Homicide and suicide are the second and third leading causes of teenage mortality. Accidents are the leading cause, and we have no idea how many of these "accidents" are really disguised suicides. Accidents, suicide, and homicide together account for 75% of all teenage deaths.

There is an evolving commitment in pediatric education to psychosocial aspects of pediatric care,[2] and pediatric services increasingly will provide for the emotional needs of children.[3] As stated by the American Academy of Pediatrics' Committee on Adolescence in 1988: "Pediatricians should include questions about suicidal thoughts in the routine medical history throughout the adolescent years. Such topics should be openly discussed with the adolescent."[2] There are no data to indicate that inquiry about suicide precipitates the behavior.

The number of children in need of help because of psychosocial and/or financial difficulties is increasing, with a relative decrease in resources and funds available to serve children.[4] Pediatricians, emergency room physicians, and family practice physicians are often the first to hear about suicidal acts or other signs of depression, including those disguised as "accidents," somatic complaints, or school problems. Thus, they are in an ideal position to assess, manage, and possibly prevent suicidal behaviors in their patients.[5] This is not to suggest that non-psychiatric physicians should necessarily undertake extensive counseling or therapy with self-destructive children. Rather, this article provides information that should help non-psychiatric physicians feel comfortable in screening, coordinating, and supporting appropriate referrals with their adolescent patients.

Demographics and Risk Factors

Four times as many 15–19 year-old girls as boys attempt suicide,[6] perhaps explaining why so many of the suicidal patients seen in the office, in the emergency room, and on medical floors are girls. However, five times as many 15–19 year-old boys as girls actually die by suicide.[7] It has been estimated that boys make three to four suicide attempts per completed suicide, whereas among girls, the ratio is 25–30 attempts to one completed suicide.[8,9]

According to Suter,[10] a suicide attempt occurs when a person is despairing about his/her own ability to cope but still has the hope of being helped by others. A suicide attempt can thus be viewed as a combination of feelings of personal helplessness with the wish of being rescued by another person. "Not having the guts to do it," or "being a coward" is a much more disturbing acknowledgment for males than females. Until 1973, boys were much more likely to use a lethal method, such as a firearm, than girls. It is hypothesized that all these factors may offer some explanation for the increased completed suicide rate among males. Additionally, in our society, males are less encouraged than females to acknowledge their feelings and/or ask for help; by the time they realize how bad they are feeling, it is likely that they are in a much more desperate state than their female counterparts.

Of the 30,000 completed suicides in the United States every year, 2,000 of these are among youngsters under the age of 20 years. Research suggests that for every completed suicide there may be anywhere from 30–100 attempts, yielding at least 60,000 attempted adolescent suicides per year.[11–14] If one considers that a minimum of ten family members and close friends may be affected by each attempted or completed suicide, we arrive at a potential figure of 620,000 people in a year who could have had first hand experience with an attempted or completed suicide of someone under the age of 20 years. If all ages of attempted suicides are included, a minimum of 9 million people in the United States could have had personal contact with an attempted or completed suicide in the last year.

Suicide rates for persons 15–19 years old by race and sex in the United States from 1970 to 1980 show that white males are at the greatest risk, followed by black and other males, white females, and then, at lowest risk, black and other females.[15] Socio-economic status and parental occupation do not significantly affect the suicide rate. Academic stress may have an impact on the suicide rate, since the completed suicide rate among female medical students, although not significantly different from male medical students, is two-to-three times higher than for the same age in the general population.[16,17] Other female professionals are also at elevated risk for suicide.[18]

Related Drug, Alcohol, and Psychiatric Problems

Alcohol and drug abuse are among the best predictors of suicide attempts and death by suicide in adolescence, whether or not a depressive illness is also present. The first

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Sponsored in part by a grant from the University Committee on Research, Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL.

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reported study of a large series of consecutive youthful suicides[19,20] suggests that 53% of the sample evidenced a substance abuse disorder at the time of death. All but two of these substance abusers met DSM-III (Diagnostic Standard Manual-III) criteria for drug abuse, and a smaller proportion (39%) of the same sample evidenced a mood disorder. Half the depressed suicide victims were substance abusers, and half were not. Shaffer (personnel communication) has indicated that the substance abuse rate in his large sample of New York City area adolescent suicides is similarly high. In addition, Peterson et al[21] found that half of 30 trauma center patients treated for self-inflicted gunshot wounds, most of which would have been fatal without emergency treatment, had used alcohol or drugs immediately before wounding themselves.

In a recent national adolescent student health survey of 11,419 students in 217 schools in 20 states,[22] it was found that 77% of eighth graders and 89% of tenth graders said they had used alcohol, and 31% reported that they had used it before or during the sixth grade. Twenty-six percent of the eighth graders, and 38% of the tenth graders reported having had five or more drinks on one occasion during the two weeks before filling out the survey. In a study conducted by Berman and Schwartz,[23] 200 of 298 (6%) adolescents who completed a questionnaire in a drug-abuse treatment program reported a history of suicidal ideation, and 89 (30%) reported making one or more suicide attempts. Forty percent used drugs within eight hours before the attempt. Berman and Schwartz concluded that drug education programs should address the issue of suicide risk recognition and management with the entire family of adolescents in treatment. According to Crumley,[24] it is likely that increasing rates of adolescent substance abuse are contributing to increasing rates in adolescent suicidal behavior.

In a systematic study of 200 adolescent psychiatric inpatients, Pfeffer et al[25] found that alcohol abuse was the single variable that retrospectively "predicted" a history of recent suicide attempts for both males and females. Two recent epidemiological studies[26,27] suggest that young persons who develop a mood disorder in childhood or adolescence are much more likely to develop a subsequent drug-use disorder when compared to their peers. Drug use is in essence self-medication of the mood disorder. With these youngsters, the first episode of major depression usually precedes the emergence of substance abuse by four years, and drug and alcohol use/abuse increases the risk of death by suicide. Many studies have found that depression is a primary psychiatric diagnosis (30%–80%) in those who die by suicide.

PSYCHOLOGICAL AUTOPSIES

A prospective analysis of the mental health problems of the completed suicide victims is almost always unavailable, and so Brent et al[28,29] used the "psychological autopsy method," in which structured interviews are given to friends and families of the deceased. They compared 27 completed adolescent suicide victims with 56 adolescents admitted to an inpatient psychiatric unit for suicidal attempts or ideation. In Brent et al's study, completed adolescent suicide victims were found to differ from suicidal psychiatrically hospitalized adolescent inpatients in several important ways. Suicide victims were more likely to have a bipolar disorder (such as major depression and mania or hypomania), a major affective disorder with a concomitant non-affective disorder (eg, anxiety disorder, conduct disorder), and have guns in their homes. Suicide victims were less likely to have had any contact with a mental health professional in their lifetime and to have been engaged in conflict with their parents. Furthermore, at the time of the attempted or completed suicidal act, approximately 90% of both groups met criteria for at least one major psychiatric diagnosis, most commonly, major depression. However, two thirds of those who died by suicide had never seen a mental health professional, and only 2 of the 27 suicide victims were receiving any treatment for their illness close to the time of death. Adolescents isolate themselves from potentially helpful adults and/or refuse help when the risk of suicide is greatest.

METHOD OF SUICIDE

The most common methods of suicide for adolescents are, in order of frequency, firearms, hanging, jumping, carbon monoxide intoxication, and poisoning.[30] Before 1971, the most frequent cause of death for females tended to be solid and liquid poisons; however, since 1973, the most frequently used method has become firearms, followed by solid and liquid poisons, and then hanging, strangulation, suffocation, and gaseous poisons. Given these figures, as well as Brent et al's[31] findings that significantly more suicide victims are likely to have had guns in their homes than hospitalized adolescents who are suicide attempters, asking about the presence of firearms in the home becomes a relevant part of taking a psychosocial history, when suicidal risk is suspected. Similarly, since 80%–90% of suicide attempts are by overdose,[32] asking about the kinds of medications kept in the medicine cabinet or elsewhere in the house becomes relevant, when suicidal risk is suspected. Aspirin is found in nearly every medicine cabinet and seems to be the preferred drug of ingestion by adolescents, along with hypnotics, sedatives, and painkillers (ie, whatever they find in a moment of anger, despair, or hopelessness). Just as parents are made aware of the danger of medication being within the reach of toddlers, so too must parents be made aware of the potential danger of medication in a household with an adolescent at risk for attempting suicide.

SUICIDAL IDEATION, SUICIDAL ACTS (GESTURES OR ATTEMPTS), AND COMPLETED SUICIDE

Clinicians often make distinctions among suicidal ideation, suicidal gestures or attempts, and completed suicide. Suicidal ideation usually refers to a person's conscious thought about or wish to end his or her own life intentionally, and may be represented by vague, hazy thoughts about suicide with no thoughts about implementation, ideas about a particular method to employ, or focused mental rehearsal of a suicidal act. This is often contrasted with suicidal acts, including suicidal "gestures" and "attempts." The phrase "suicide gestures" is usually reserved for suicidal acts that are presumed to be less dangerous, less serious, and of less concern than "suicide attempts." Calling a suicidal act a "gesture," then, is to make an interpretation that the act was not intended seriously—often a dangerous assumption to make. As defined by Murphy,[32] "attempted suicide is any act, including overdose, cutting, or otherwise producing tissue injury that is so labeled by the person doing it, or by those involved in that person's care." All suicidal "gestures" and "attempts" should be considered suicidal "acts" and taken seriously, especially as clinicians and researchers have not yet reached any consensus about how to rate the severity of or future suicide risk posed by specific suicidal acts.

Often there is a lack of agreement among the adolescent, his or her family, and the professional as to whether specific behaviors constitute a "suicidal" act or not. Not only is there considerable social stigma attached to suicide-related behavior, but also families and particularly parents often find it painful and/or difficult to admit or acknowledge that their child has tried to kill him or herself. Although good agreement can be reached about what constitutes a suicidal act, considerable ambiguity remains in the scientific literature about how best to define the intent or "seriousness" of the suicidal act, and hence, the clinician should be reluctant to minimize concern about any specific suicidal act by labeling it as a "gesture." It is for this reason that Murphy's definition is a good one because it describes the act itself rather than an interpretation of what the act means.

Non-fatal suicide attempts must also be carefully distinguished from completed suicide. The demographic and clinical profiles of adolescents who die by suicide and those who make non-fatal suicide attempts are markedly different, and the clinician should be cautious about generalizing from risk profiles for completed suicide to risk profiles for non-fatal attempts.[33] This is not to say, however, that adolescents who make non-fatal attempts should be treated as persons at relatively low risk for completed suicide. Adult studies of suicidal behavior, which are our best guide until comparable adolescent studies have been reported, consistently show the following: (a) 7%–10% of persons receiving treatment for non-fatal suicide attempts eventually die by suicide over the course of their lifetimes, and (b) 60% of persons who die by suicide had never made a previous non-fatal attempt before in their lifetimes. By implication, adolescents who have made non-fatal attempts are at high-risk for death by suicide, as their lifetime risk is approximately five times greater than those who have never made a non-fatal attempt. However, more than 90% of persons receiving treatment for non-fatal suicide attempts will never die by suicide, and more than half of all completed suicides occur among persons with no earlier history of suicidal behavior.

The interrelationship among suicidal ideation, suicidal acts, and completed suicide is both complicated and controversial. The early "single-population" view suggested that all suicidal thinking and behavior were related to one another and differed only in terms of severity of the underlying suicidal tendency: completers were simply more suicidal than attempters, and attempters were simply more suicidal than ideators. Under this single-population view, all suicide attempters and all completed suicides shared the classification of suicide ideators, and the differences among the three groups were minimized. This perspective fostered generalizations from clinical studies of ideators and non-fatal attempters, implying that persons who die by suicide are like the first two groups but more severely affected. More recent studies, however, have exposed the flaws in the single-population model. The differences between non-fatal attempters and those dying by suicide are consistently pronounced in studies of adolescents[28,29,33] and adults.[32,34,35]

In a "multi-population" alternative adult model posed by Linehan,[34] differences rather than similarities among the three groups are highlighted. Linehan hypothesizes that although there is certainly overlap among ideators, non-fatal attempters, and completers, the groups are more different than alike, and that a substantial number of "self-harmers" are not contemplating suicide when they injure themselves. The clinical value of the multi-population perspective is that it treats each of the three behaviors, suicidal ideation, suicide attempts or "self-harm," and completed suicide, as separate entities, and it further emphasizes the identification of different kinds of attempters, each requiring a different kind of treatment.

PHYSICIANS, NOT THE MENTAL HEALTH PROFESSIONAL, SEES THESE CHILDREN FIRST

From adult suicide autopsy data, we know that most completed suicides have never seen a mental health professional. However, at the same time, 50% had seen a physician for a medical complaint (ie, a non-psychiatric physician) within a month of their suicides, and, within 6 months of their suicides, 80% had seen a physician.[36,37] Murphy[38] found that for 49 adults who had seen a physician within six months of their death by suicide, more than two thirds of the adults had previously attempted or threatened suicide, but only 40% of their physicians were aware of it. Of the 27 adolescent suicide victims studied by Brent,[29,31] only one third had ever seen a mental health professional. It seems likely that non-psychiatric physicians in general, and pediatricians in particular, are in an excellent position to play a significant role in the detection of self-destructive thoughts and behaviors in their younger patients. The average adolescent sees a physician an average of two-to-three times a year for school, camp, or work physicals, illness, and so forth. It seems likely, then, that most youngsters will have seen their physician at least once within any six month time period, with those at risk for suicidal behavior perhaps visiting more frequently. Non-psychiatric primary care physicians are in a critical position to prevent suicidal behavior when trained how to identify at-risk adolescents and intervene appropriately.

It is important for physicians to understand that adolescents "talk with their feet;" if an adolescent did not have something to say, he or she would not be in the doctor's office. Adolescent's thoughts about, or attempts at, suicide might be best viewed as attempts at communication, albeit maladaptive ones. Adolescents are unlikely to walk into the office and spontaneously speak of their thoughts about dying. They are likely to present with some vague complaint such as headaches, stomachaches, dizziness, or problems at home or at school. Physicians must hear their words, but *listen* to what it is they are really trying to say. The clinician's task is to decipher the communication and determine the best mode of treatment for each individual case.

IF YOU DON'T ASK, YOU WON'T KNOW
IF YOU DON'T KNOW, YOU CAN'T HELP

In January 1988, the American Academy of Pediatrics stated that: "The pediatrician should not hesitate to question an adolescent directly about suicidal thoughts. There are no data indicating that inquiry about suicide precipitates the behavior." From the generic (ie, not exclusively adolescent) literature on completed suicides, asking or talking about suicidal thoughts or behaviors does not appear to promote nor induce suicidal thoughts nor behaviors. Rather, many afflicted adolescents are relieved to be asked because it gives them the opportunity, or permission, to discuss an often thought about but generally taboo topic.

Friedman et al[39] found that 60% of 380 high school students given an anonymous survey regarding their experience with suicidal behavior reported that they had thought about killing themselves. Almost 9% of these youngsters reported that they had actually made one attempt, and over half of these "attempters" had made at least two attempts. Less than half of these youngsters had ever been seen by a mental health professional. Berman and Schwartz[23] found that of the 89 adolescents in a drug-abuse treatment program who reported making one or more suicide attempts, 78% of the attempts occurred at home. However, 35% of the attempters reported that their parents never found out, and 19% reported that no one else ever knew. A recent survey conducted by the Centers for Disease Control[22] revealed that approximately one of three eighth and tenth graders had seriously contemplated suicide. This 1987 sampling of 11,419 students in 217 schools in 20 states found that 25% of the boys, and 42% of the girls reported that they had seriously thought about killing themselves. Of the girls, 18% and 11% of the boys reported that they had actually attempted to injure themselves in a way that could have resulted in their death. More than half of the students (53%) reported that they have known someone who tried to commit suicide.

A time ago, the American Academy of Pediatrics stated: ". . . suicidal thoughts or comments should never be dismissed as unimportant." It is not true that: "those who talk about suicide don't, or won't, try to do it." Murphy[13] reported that although an earlier suicidal act preceded one-third of the completed suicides, two-thirds to four-fifths of the completed suicides communicated thoughts about dying to more than one person, on more than one occasion, and in more than one way. It is reported that approximately 40% of completed suicides have given direct notice (ie, "I'm going to kill myself and jump in the river"). Adolescents, however, are equally likely to be indirect: "I would be better off dead," or "My family would be better off if I were dead or not around," or "Nobody would care or miss me anyway."

Of 27 adolescent suicide victims studied by Brent et al,[28,29] 83% talked about their suicidal state of mind with others within a week of their death. Half of those who communicated their intentions did so only to persons of the same age, such as siblings or friends. Results from the National Adolescent Student Health Survey[22] found that over 50% of eighth and tenth graders reported that it would be "hard" or "very hard" to tell someone in a friend's family, the school counselor, or a teacher if they suspected that a friend of theirs might be suicidal. Over 50% of these youngsters also reported that it would be "hard" or "very hard" to tell an adult if they had promised their friend that they would not.

Suicidal risk is one situation in which caregivers, who generally maintain confidentiality with their adolescent patients, must break confidentiality in the best interest of their patients. Parents must be informed and measures taken to ensure the safety of the adolescent. All talk of suicidal intent must be listened to and taken very seriously. Lines of communication must be opened to persons in a position to intervene, before it is too late.

MOST COMMON PRECIPITATING EVENTS

Some studies report a seasonal effect on suicide; however, the peak months reported vary with the study.[8,40] Many factors contribute to suicidal risk, such as broken homes, alcohol abuse by patient or a parent, history of physical abuse, history of a previous attempt by patient or another member of the family. These factors, although important, often do not differentiate suicide attempters from other adolescent psychiatric patients. However, "self esteem and poor interpersonal skills characterize almost all suicide attempters."[32]

Both Marks[41] and Schreiber and Johnson[3] found that parental conflict was the event most frequently stated to have precipitated a suicidal act, followed by romantic conflict, personal loss, and chronic depression. It is almost never helpful to tell one of these youngsters that they have "no good reason" for wanting to die. Rather, it is much more helpful to acknowledge their feelings, stating, "It is awful that you feel so badly that you would want to die." Children and adolescents do not have the perspective of time. They do not know that things will get better. They do not know that they will not always feel as badly as they do at present. They are egocentric in their thinking and cannot free themselves from their own subjective viewpoints and feelings to take another person's point of view. They do not know that they are not the only person in the world who feels so badly. They must be reassured. There must be empathy and not anger or scorn.

MODELING/MEDIA

The idea that published accounts of suicidal deaths promote imitation is an old one. For example, in 1774, the German poet Goethe published a romantic novel, *The Sorrows of Young Werthur,* about a gifted and artistic young man who shot himself in the head after a failed love affair. This novel was widely read in Europe, and because it was felt to have been the model used by young people in many countries to kill themselves in the months that followed, it was banned in several cities. In the 1840 annual report on births, deaths, and marriages in England, William Farr, the Registrar General of England, called for an end to publicizing deaths by suicide, murder, and bloodshed in the newspapers, as he argued that these acts are often committed by imitation.

In recent times, the seminal work of David Phillips, a sociologist at the University of California, San Diego, has shed new light on the frequency and nature of suicide contagion. In a series of studies, Phillips and Carstensen[42,43] have shown that the number of United States suicides increases 2.5% for two weeks after the story of a suicide is publicized on the front page of the *New York Times*, and similar effects can be documented in England for stories in major London newspapers. The larger the circulation of the newspaper and the more days the story is publicized, the greater is the increase in suicide rate. In addition, the number of United States suicides increases 7% for seven days after a suicide is publicized on two or more national network TV evening news programs. These newspaper- and television-related increases in suicides occur almost entirely within the adolescent population, regardless of the age of the suicide victim being reported. News reports of suicides are much more likely to be followed by an increase in suicides if the story is repeated on different days and on different television programs. Phillips' research indicates that media-related increases in deaths by suicide are not simply a matter of encouraging suicides that would have occurred sooner or later anyway.

Research on the impact of fictional made-for-TV movies about suicide, intended to have an educational and/or preventive impact with the help of expert consultants and advisors, has yielded contradictory findings. Although Gould and Shaffer[44] originally reported an increase in teen suicidal deaths and suicide attempts in the two weeks after the airing of four fictional movies on national network television, others have not replicated their findings.[45–47] Berman[45] suggests that TV dramas may influence the method of suicide youngsters employ, rather than the number of teen suicides.

Suicide "outbreaks" of all types have been reported and discussed, including psychiatric inpatient units, high schools, neighborhoods, Native American reservations, military bases, and prisons[48–57] There has been surprisingly little effort to develop quantitative epidemiological definitions of suicide clusters, in spite of the frequency with which these outbreaks have been reported to occur. The relatively low rate of suicide in the general population and the small numbers that make up a potential cluster make it difficult to distinguish between clusters of suicides that lie within the realm of chance aggregation and clusters representing significant aggregations that cannot be explained by chance alone. Gould et al[58] undertook a time-space cluster analysis of United States data on adolescent suicide and concluded that only 1% of all teen suicides occur in the context of a cluster event. Gibbons et al[59] found that in one large metropolitan county in the past 11 years, all clusters identified by the media or mental health professionals were randomly distributed, independent events. They propose their model to be a good epidemiological tool for discriminating between "real" and "pseudo" clusters, so that the mechanisms underlying the contagious communication of suicide can be studied further.

The word "epidemic" has been used to describe the problem of adolescent suicide. As with child abuse and sexual abuse, the problem of adolescents killing themselves has been around for a long time. However, now that we acknowledge that children and adolescents can be depressed, now that we acknowledge that they can have suicidal thoughts, suicidal behavior is more likely to be identified, and pediatricians and family practice physicians are in the ideal position to do so.

BIOLOGY OF SUICIDAL RISK

The promise and complexity of investigations into the biology of suicide are growing in parallel. Proposed correlates or markers of suicide investigated over the last 20 years have included urinary 17-hydroxycorticosteroids, cortisol levels in plasma and cerebrospinal fluid (CSF), response to dexamethasone challenge, 5-hydroxyindoleacetic acid in CSF, thyroid-stimulating hormone response to thyrotropin-releasing hormone, norepinephrine-to-epinephrine ratio in urine, 3-methoxy-4-hydroxyphenolglycol and homovanillic acid, tritiated imipramine binding in brain tissue, elevated CSF magnesium levels, and decreased prolactin response to fenfluramine. These research avenues constitute areas of promising developments but not the basis for clinical decision-making.[60–62] Small sample sizes, inadequate comparison groups, and insufficiently differentiated psychological characterization of research subjects has made it difficult to understand whether findings are more closely linked to suicidal behavior, violent tendencies, impulsivity, anxiety and agitation, or underlying psychiatric diagnosis. Furthermore, very little of the biological research on suicide has been conducted in adolescent samples. Thus, we do not know the degree to which adult research findings generalize to youth.

PHYSICIAN INTEREST AND BEHAVIOR

In a study conducted by Hodgman and Roberts,[1] 48 pediatricians were polled to determine their awareness of adolescent suicide and suicide attempts in Syracuse and Rochester, New York. Eighty-seven percent of practicing pediatricians who were sent a one-page questionnaire responded. Hodgman and Roberts found that although few actual deaths by suicide or "accidental" deaths attributed to suicide were encountered by pediatricians (10 total across 48 pediatricians and 979 total practice years represented), they did encounter a substantial number of suicidal attempts. One third of suicide attempts were preceded by medical consultation, and more than one half were routinely referred for psychiatric consultation. Some pediatricians reported that they ask about suicide "only if indicated," although for others, the question allegedly "never comes up" in their practices. It is important to remember that asking about suicide does not cause suicide, and asking about suicidal symptoms should be a part of routine history taking during the adolescent years. If we do not ask, we will not know; if we do not know, we can not help.

McIntire, Fine, and Fain[63] sent a questionnaire to 110 members of the Nebraska Chapter of the American Academy of Pediatrics. Of the 65 pediatricians, two thirds who responded stated that they did not manage suicide attempters, but rather, refered the patients elsewhere, usually to psychiatrists. As a group, these pediatricians were more likely to manage accident-prone children and abusive or neglectful parents than to manage suicide attempters. Seventy-nine percent of the pediatricians surveyed felt that a continuing medical education course on the subject of teen suicide and self-destructive behaviors would be extremely effective, with the remaining 21% concluding it would be somewhat effective. No one felt it would be ineffective or a waste of time. A city- or nation-wide survey of pediatricians' actual and current experiences with suicidal adolescents is badly needed.

Hergenroeder et al[64] reviewed 45 charts of children and adolescents who presented in the emergency room with suicidal thoughts or behavior. They found that 21 of these children were directly referred to the Child Psychiatry Service, with only minimal involvement by the pediatric resident. Of the remaining 24 cases, residents conducted a suicide assessment (ie, past suicide attempts and current ideation) in only about one fourth of the cases. The investigators recommend the need for pediatric programs to emphasize training in the assessment of suicidal risk and appropriate interventions for their adolescent population. Hergenroeder et al[64] offer the possibility that pediatricians are not more involved with suicidal adolescents because traditionally they have not been trained to do so. The investigators suggest that if pediatricians were perceived as more accessible and helpful to their adolescent patients for psychosocial needs, perhaps they would be more likely to be called on during times of stress.

Hergenroeder et al's[64] findings are consistent with those of Marks et al,[65] whose self-administered, anonymous questionnaires were completed by 649 high school students in a middle-class New York suburb. Of these adolescents, 49% reported that they see a pediatrician regularly, and 93% (85% for a "check-up") reported that they had been to a physician at least once within the year. Nevertheless, a majority of these students stated that they would not elect to go to their physician for care related to sexuality, substance abuse, or emotional difficulties. The investigators conclude: "If pediatricians are to meet the health care needs of adolescents, they must adapt their approach to this age group, including considerations of time and payment, privacy and confidentiality, and expertise"[65(p460)]. This adaptation has begun because pediatricians are increasingly asked to consult to schools after a serious suicide attempt or death by suicide and become participating members of ongoing crisis teams.[66]

FROM THE EMERGENCY ROOM: WHERE TO?

In a study conducted by Litt et al,[67] the charts of 27 adolescents who had attempted suicide and were brought to the emergency room at Stanford University were reviewed during a one-year period. Of these adolescents, 42% had poor relationships with parents; 30% had a previous history of having made more than one suicide attempt, two thirds of which were within the two weeks before the present attempt; and 67% had behavior, school, drinking, or sleeping problems. Seventy-eight percent of these attempts involved a drug overdose. All patients, whether discharged from the hospital (13 of 27 were hospitalized) or directly from the emergency room, were given outpatient follow-up appointments. For both groups, there was approximately 40% compliance with recommended follow-up within one year. Only one variable separated compliant from non-compliant patients: history of previous attempt. Sixty percent of those who were non-compliant with outpatient follow-up had made a previous attempt. Compliance is a common problem with medical regimens, whether psychiatric or medical. Adolescents who have attempted suicide for the first time, that is, with no history of previous attempts, are more likely to follow through with a therapeutic referral than those patients with a history of previous attempts, who are at a greater risk.

FROM THE PEDIATRIC FLOOR: WHERE TO?

The policy of the Division of Adolescent Medicine at Albert Einstein College of Medicine is to admit to a general medical adolescent inpatient medical unit all adolescents presenting to the emergency room or outpatient service after a suicide attempt.[41] This policy permits prompt attention to physiologic and psychologic disturbances, separation of the adolescent from the environment which generated the suicidal behavior, and evaluation of follow-up needs. Seventy percent of the first 100 suicide attempters admitted to the adolescent unit were females, with ingestion of drugs or toxic substances involved in 95% of the attempts. Most frequently used were aspirin, barbiturates, and other sedative hypnotics commonly found in the home. No suicide attempts or self-abusive acts occurred during hospitalization. However, 11 patients remained actively suicidal for the duration of their stay and were transferred to inpatient psychiatric units. Of the 89 patients judged not to be at acute suicidal risk, approximately half were discharged to outpatient psychiatric follow-up, with 16 discharged to an adolescent ambulatory program to receive follow-up from a pediatrician or a social worker. Although only a few patients required transfer to inpatient psychiatry, all adolescents were judged in need of further professional attention.

A similar study was conducted by Schreiber and Johnson.[3] During a six-month period, 38 adolescents, 50% male and 50% female, were medically admitted for acute drug ingestion. Of the 38 admissions, a psychiatric consultation was requested in only 18 of the cases, and 72% of this group was female. In the 20 who received no consultation, 70% were male, and there were 12 cases of drug ingestions that reportedly occurred during an episode of drug abuse. Furthermore, most of the non-consult group were adolescents actively involved in the abuse of drugs and reported having overdosed or suffered a side-reaction to a drug in an attempt to get high. In light of the high percentage of substance abuse in adolescents who die by suicide, all adolescents who reportedly overdose as a consequence of substance abuse should also receive mental health consultation.

YOU DON'T HAVE TO BE A PSYCHIATRIST TO ASSESS SUICIDAL RISK

McIntire et al[14] found that using a structured interview with a pediatric perspective, specially trained paraprofessionals arrived at estimates of lethality, risk of recidivism, and current stress that correlated at 95% with ratings by psychiatrists. Prediction of risk was 100% reliable in low-risk subjects. With some training and interest, physicians can establish a database (eg, suicidal intent, medical lethality) and estimate the likelihood of compliance with follow-up recommendations and the probability of subsequent attempts.

The assessment of suicidal thoughts and intent can be readily learned. In a study conducted by Patterson,[68] two groups of third-year medical students were given a lecture and one week later were asked to rate the suicidal risk of two patients who were presented in videotaped interviews. One group was given the lecture and an acronym, and one group was given the lecture with no acronym. Three senior psychiatrists viewed the films and assessed each of the two patients' suicidal risk on a scale of 0 to 10, with 10 as highest risk. One patient was given a rating of 3 by the three psychiatrists; the control group, with no acronym, overrated suicidal risk at 5.6, whereas the investigational group with the acronym gave the patient a risk rating of 3.06. Similarly, the second patient was given a rating of 5 by the three psychiatrists; the control group, with no acronym, again overrated the risk at 8.15, whereas the investigational group with the acronym assessed risk at 4.96. There was no significant difference between the investigational group with acronym and the psychiatrists' ratings; the control group overrated suicidal risk in both cases. The acronym used was SAD-PERSONS (S = Sex; A = Age; D = Depression; P = Previous attempt; E = Ethanol abuse; R = Rational thinking loss; S = Social supports lacking; O = Organized plan; N = No spouse; S = Sickness). SAD-PERSONS is an acronym that is easily remembered and can help physicians to determine and evaluate suicidal risk factors more accurately. For adolescents, the acronym can be revised to SAD-PERSONS-R, where S = Sex; A = Adolescents; D = Depression; P = Previous attempts; E = Ethanol and drug abuse; R = Rational thinking loss; S = Social supports lacking (parents and peers); O = Organized plan; N = No one to talk to; S = Sickness or somatization; and R = Risk-taking behaviors (eg, accidents).

SUMMARY

Pediatricians, internists, emergency-room physicians, family practice physicians, and other non-psychiatric physicians, often trusted allies of the child and family, are in an excellent position to assess, manage, and even prevent suicidal behaviors in their younger patients.[5] This is not to suggest that these physicians should necessarily undertake extensive counseling or therapy with self-destructive children and adolescents; rather, they can help to screen and coordinate appropriate referrals and reinforce and/or support corrective efforts.[5]

PRECAUTIONS: ELEVEN POINTS TO REMEMBER

1. *P*arental and romantic conflict, personal loss, and chronic depression are the most frequent precipitants of adolescent suicidal behavior. These adolescents need empathic support and direction.
2. *R*outine history taking during the adolescent years should include direct questions about suicidal thoughts and behavior.
3. *E*xplain to parents the potential risk of having medications and firearms in the home and share information, when suicidal risk is suspected.
4. *C*hronic low self-esteem and poor interpersonal skills characterize almost all suicide attempters.
5. *A*dolescents are more likely to die from suicide than any medical condition.
6. *U*nder all conditions, suicidal "gestures" and "attempts" should be considered suicidal "acts," taken seriously, and followed up and/or referred to a mental health professional.
7. *T*raining non-psychiatrists to assess suicidal risk has proved effective. Using a structured interview with a pediatric perspective, trained paraprofessionals arrived at estimates of lethality, risk of recidivism, and current stress that correlated at 95% with ratings by psychiatrists.
8. *I*dentify a colleague in the mental health profession (psychologist, social worker, psychiatrist, and so forth) with whom you feel comfortable discussing and/or referring patients as indicated.
9. *O*ver four times as many 15–19-year-old girls as boys attempt suicide; five times as many boys as girls die by suicide.
10. *N*on-psychiatric physicians in general, and pediatricians in particular, are in an excellent position to play a significant role in the detection of self-destructive thoughts and behaviors in their young patients. Adolescents are more likely to present with vague complaints such as headaches, stomach aches, problems at home or at school than to describe suicidal thoughts and/or behaviors spontaneously. The clinician's task is to decipher the communication and determine the best mode of treatment for each individual patient.
11. *S*ubstance abuse, that is alcohol and drug abuse, are among the best predictors of suicide attempts and death by suicide in adolescence, whether or not a depressive illness is also present. All adolescents who overdose, even if reportedly a consequence of substance abuse, should receive mental health consultation.

Acknowledgments. The authors would like to thank Samuel P. Gotoff, MD, for his encouragement and thoughtful comments throughout the preparation of this manuscript and also Barbara Kienker for typing this manuscript.

REFERENCES

1. Hodgman CH, Roberts FN: Adolescent suicide and the pediatrician. *J Pediatr* 1982; 101:118–123.
2. Committee on Adolescence: American academy of pediatrics. suicide and suicide attempts in adolescents and young adults. *Pediatr* 1988; 81(2);322–324.
3. Schreiber TJ, Johnson RL: The evaluation and treatment of adolescent overdoses in an adolescent medical service. *J Natl Med Assoc* 1986; 78:101–108.
4. Preston SH: Children and the elderly in the U.S. *Sci Am* 1984; 251:44–49.
5. McKenry PC, Tishler CL, Kelley C: Adolescent suicide. a comparison of attempters and nonattempters in an emergency room population. *Clin Pediatr* 1982; 21:266–270.
6. Garfinkel B: Major affective disorders in children and adolescents, in Winokur G, Clayton P: *The Medical Basis of Psychiatry.* Philadelphia, WB Saunders Co, 1986, pp 308–330.
7. Shaffer D: The epidemiology of teen suicide. an examination of risk factors. *J Clin Psychiatry* 1988; 49(Suppl):36–41.
8. Otto U: Suicidal acts by children and adolescents. a follow-up study. *Acta Psychiatr Scand* (suppl)1972; 233:1–123.
9. Waldenstron J, Larson T, Ljungstedt N: *Suicide and Attempted Suicide.* Stockholm, Nordiska Bokhandelns Forlag, 1972.
10. Suter B: Suicide and women, in: Wolman BB, Krauss HH (eds). *Between Survival and Suicide.* New York, Gardner Press, 1976.
11. Jacobziner H: Attempted suicides in children. *J Pediatr* 1960; 56:519–525.
12. Jacobziner H: Attempted suicides in adolescence. *JAMA* 1965; 191:101–105.
13. Tuckman J, Connon HE: Attempted suicide in adolescents. *Amer J Psychiatr* 1962; 119:228–232.
14. McIntire MS, Angle CR, Wilcoff RL, et al: Recurrent adolescent suicide behavior. *Pediatr* 1977; 60:605–608.
15. Department of Health & Human Services, Public Health Service, CDC, *Youth Suicide in the U.S.* 1970–1980; Atlanta, 1986.
16. Pepitone-Arreola-Rockwell F, Rockwell D, Cone N: Fifty-two medical student suicides. *Am J Psychiatr* 1981; 138:198–201.
17. Roy A: Suicide in doctors. *Psychiatr Clin North Am* 1985; 8(2):377–387.
18. Clayton P, Marten S, Davis M, et al: Mood disorder in women professionals. *J Affective Disord* 1980; 2:37–46.
19. Rich CL, Young D, Fowler RC: San Diego Suicide Study. I. young vs. old subjects. *Arch Gen Psychiatry* 1986; 43:577–582.
20. Fowler RC, Rich CL, Young D: San Diego Suicide Study. II. substance abuse in young cases. *Arch Gen Psychiatry* 1986; 43:962–965.
21. Peterson LG, Peterson M, O'Shanick GJ, et al: Self-inflicted gunshot wounds. lethality of method versus intent. *Am J Psychiatry* 1985; 142:228–231.
22. Collins JL, Appel EM, Popham WJ: The 1987 National Adolescent Student Health Survey. *MMWR* March 10, 1989; 38:147–150.
23. Berman AL, Schwartz RH: Suicide attempts among adolescent drug users. *Am J Dis of Child* 1990; 144:310–314.
24. Crumley FE: Substance abuse and adolescent suicidal behavior. *JAMA* 1990; 263:3051–3056.
25. Pfeffer CR, Newcorn J, Kaplan G, et al: Suicidal behavior in adolescent psychiatric inpatients. *J Am Acad Child Adolesc Psychiatry* 1988; 27:256–261.
26. Deykin EY, Levy JC, Wells V: Adolescent depression, alcohol, and drug use. *Am J Public Health* 1987; 76:178–182.
27. Christie KA, Burke JD, Regier DA, et al: Epidemiologic evidence for early onset of mental disorders and higher risk of drug abuse in young adults. *Am J Psychiatry* 1988; 145:971–975.
28. Brent DA, Perper JA, Allman CJ: Alcohol, firearms, and suicide among youth. *JAMA* 1987; 257:3369–3372.
29. Brent DA, Perper JA, Kolko DJ, et al: The psychological autopsy method: methodological considerations for the study of adolescent suicide. *J Am Acad Child Adolesc Psychiatry* 1988; 27:362–266.
30. Shaffer D, Fisher P: The epidemiology of suicide in children and young adolescents. *J Am Acad Child Psychiatry* 1981; 20:545–565.
31. Brent DA, Perper JA, Goldstein CE, et al: Risk factors for adolescent suicide. a comparison of adolescent suicide victims with suicidal inpatients. *Arch Gen Psychiatry* 1988; 45:581–588.
32. Murphy GE: Suicide and attempted suicide, in: Winokur G, Clayton P (eds). *The Medical Basis of Psychiatry.* Philadelphia, WB Saunders, 1986, pp 562–579.
33. Brent DA: Suicide and suicidal behavior in children and adolescents. *Pediatr Rev* 1989; 10:269–275.
34. Linehan MM: Suicidal people. one population or two? *Annals NY Acad Sci* 1986; 487:16–33.
35. Fawcett J, Scheftner WA, Fogg L, et al: Time-related predictors of suicide in major affective disorder. *Am J Psychiatry* 1990; 147:1189–1194.
36. Robins E, Murphy GE, Wilkinson RH Jr, et al: Some clinical considerations in the prevention of suicide based on a study of 134 successful suicides. *Am J Public Health* 1959; 49:888–899.
37. Murphy G: The physician's responsibility for suicide. I: an error of commission. II: errors of omission. *Annals Internal Med* 1971; 82:302–309.
38. Murphy GE: The physician's responsibility for suicide. II: errors of omission. *Ann Intern Med* 1975; 82:301–304.
39. Friedman JMH, Asnis GM, Boek M, et al: Prevalence of specific suicidal behaviors in a high school sample. *Am J Psychiatry* 1987; 144:1203–1206.
40. Hawton K, Goldacre M: Hospital admissions for adverse effects of medicinal agents (mainly self-poisoning) among adolescents in Oxford region. *Br J Psychiatry* 1982; 141:106–170.
41. Marks A: Management of the suicidal adolescent on a nonpsychiatric adolescent unit. *J Pediatr* 1979; 95:305–308.
42. Phillips DP, Carstensen LL: Clustering of teenage suicides after television news stories about suicide. *N Engl J Med* 1986; 315:685–689.

43. Phillips DP, Carstensen LL: The effect of suicide stories on various demographic groups, 1968–1985. *Suicide Life Threat Behav* 1988; 18:100–114.

44. Gould MS, Shaffer D: The impact of suicide in television movies. evidence of imitation. *N Engl J Med* 1986; 315:690–694.

45. Berman AL: Fictional depiction of suicide in television films and imitation effects. *Am J Psychiatry* 1988; 145:982–986.

46. Gould MS, Shaffer D, Kleinman M: The impact of suicide in television movies. replication and commentary. *Suicide Life Threat Behav* 1988; 18:90–99.

47. Phillips DP, Paight DJ: The impact of televised movies about suicide. a replicative study. *N Engl J Med* 1987; 317:809–811.

48. Anonymous: A suicide epidemic in a psychiatric hospital. *J Diseases Nervous System* 1977; 38:327–331.

49. Sacks M, Eth S: Pathological identification as a cause of suicide on an inpatient unit. *Hosp Community Psychiatry* 1981; 32:36–40.

50. Robbins D, Conroy RC: A cluster of adolescent suicide attempts. is suicide contagious? *J Adoles Health Care* 1983; 3:253–255.

51. Ashton VR, Donnan S: Suicide by burning as an epidemic phenomenon: an analysis of 82 deaths and inquests in England and Wales in 1978–1979. *Psychol Med* 1981; 11:735–739.

52. Walton EW: An epidemic of antifreeze poisoning. *Medi Sci Law* 1978; 18:231–237.

53. Rubinstein DH: Epidemic suicide among Micronesian adolescents. *Soc Sci Med* 1983; 17:657–665.

54. Ward JA, Fox J: A suicide epidemic on an Indian reserve. *J Canadian Psychiatr Ass* 1977; 22:423–426.

55. Hankoff LD: An epidemic of attempted suicide. *Comp Psychiatry* 1961; 2:291–298.

56. Farrell, JW, Petrone ME, Parkin, WE et al: Bergenfield suicide cluster report. *MMWR* 1988; 37:213–216.

57. Coleman L: *Suicide Clusters*. Winchester MA, Faber & Faber Inc, 1987.

58. Gould MS, Wallenstein S, Kleinman M: Time-space clustering of teenage suicide. *Am J Epidemiol* 1990; 131:71–78.

59. Gibbons RD, Clark DC, Fawcett J: A statistical method for evaluating suicide clusters and implementing cluster surveillance. *Am J Epidemiology* 1990; 132(suppl):183–191.

60. Motto J: Clinical considerations of biological correlates of suicide. *Suicide Life Threat Behav* 1986; 16:83–102.

61. Stanley M, Mann JJ: Biological factors associated with suicide, in Frances AJ, Hales RE (eds). *American Psychiatric Press Review of Psychiatry,* Washington DC, American Psychiatric Press 1988; 7:334–352.

62. Brown GL, Linnoila MI: CSF serotonin metabolite (5-HIAA) studies in depression, impulsivity, and violence. *J Clin Psychiatry* 1990; 51(suppl):31–43.

63. McIntire MS, Fine P, Fain PR: Early indicators of self-destructive behaviors in children and adolescents, in: Sudak HS, Ford AB, Rushforth NB (eds). *Suicide in the Young,* Boston, John Wright, 1988, pp 259–271.

64. Hergenroeder AC, Kastner L, Farrow JA, Deisher RW: The pediatrician's role in adolescent suicide. *Pediatr* 1986; 15:11:787–798.

65. Marks A, Malizio J, Hock J, Brody R, Fisher M: Assessment of health needs and willingness to utilize health care resources of adolescents in a suburban population. *J Pediatr* 1983; 102:456–460.

66. Adler RS, Jellinek MS: After teen suicide. issues for pediatricians who are asked to consult to schools. *Pediatr* 1990: 86(6): 982–987.

67. Litt IF, Cushey WR, Rudd S: Emergency room evaluation of the adolescent who attempts suicide. *J Adoles Health Care* 1983; 4:106–108.

68. Patterson WM, Dohn HH, Bird J, et al: Evaluation of suicidal patients. The SAD PERSONS scale. *Psychosomatics* April 1983; 24:343–349.

SPECIAL ARTICLE

The challenge to psychiatry by psychologists: Economic versus professional issues

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These days, all agree that the practice of medicine is undergoing profound and difficult changes. There is the increased pressure to attend to the business of clinical practice, with burgeoning demands for paperwork documentation. The avalanche of new developments in medical technology has led not only to more precise diagnosis and effective treatment, but also to increased patient expectations. Coupled with our current environment of adversarial consumerism and the defensive medicine it has engendered, these miracles of modern medicine have paradoxically enhanced the growing crisis in the financing of medical care in this country. Clearly, challenges have been raised to the traditional definition of the physician's role, responsibility, and accountability by a demanding consumer population.

Superimposed on this backdrop, there is the emergence of territorial disputes within some clinical patient-care domains, resulting in a challenge to some of the traditional delineations made between different health-care-providing disciplines.

Regarding this last point, the current paper focuses on the initiatives by clinical psychologists through the offices of their national organization, the American Psychological Association.

THE ISSUE

Psychologists have organized an attack on certain traditional areas of medical/psychiatric practice. They assert that they should be granted full-admitting privileges to hospitals and the right to prescribe psychoactive medications. With regard to the first of these claims, they deny that their concern is a guild issue or a fight over professional territory. They insist, instead, that they are fully qualified for, and deserving of, hospital privileges and should be permitted to admit, treat, and discharge patients without supervision of a physician. This paper first reviews these claims and the response of the American Psychiatric Association (APA) to them and then discusses the issues raised.

ASSERTIONS OF THE AMERICAN PSYCHOLOGICAL ASSOCIATION

In 1979, clinical psychologists brought suit against the Neuropsychiatric Society of Virginia over practice-venue issues.[1] Five years later, the California Association of Psychological Providers (CAPP) sued the State of California and the director of its Department of Human Services for alleged discrimination against psychologists.[2] In that suit and in subsequent position papers, CAPP and the American Psychological Association protested the state's insistence on *psychiatric* supervision of care rendered by psychologists to hospitalized patients. Although a superior court in California initially found for CAPP, a California appeals court in June 1988 reversed the lower court finding.[3,4] The higher court ruled that psychologists may not practice medicine and that physicians may supply psychological services. On June 25, 1990, however, in a 5-to-4 split decision, the California Supreme Court ruled in favor of the psychologists and, notably, stated that it is up to the legislature, not the courts, to decide on criteria for competence for overall patient care.[5]

Before this ruling, a position, supported by a document entitled *The Independent Practice of Psychology in Hospitals: Advantages to Professionals and Consumers* was presented to members of the New York State Legislature by spokespersons for the American Psychological Association, through the New York State Psychological Association. The paper was couched in assertions claiming discrimination. This document requested the legislature to "grant psychologists the right to independent practice in hospitals. . . ."[6] It was based on the following assumptions:

- That a civil rights issue ("equality" of access to practice in hospitals) and a training, experience, and competency issue ("equivalency") between psychiatrists and psychologists are one and the same;
- That independent clinical practice in a hospital setting means nonaccountability and exists only for staff physicians;
- That there exists a clear and unambiguous meaning for the term "licensure," when applied to psychologists;
- That outpatient and hospital-based psychological practice are the same with no differentiation among different types of inpatient services.

The psychologists concluded their argument claiming that when it comes to patient care management within an acute, inpatient setting, psychologists are co-equal, if not superior, to psychiatrists with regard to experience and training.[7] Notably, they deny that any economic turf considerations underlie their position.

In response to this position paper, an act was sponsored in the New York State Legislature with the stated purpose of overcoming ". . . the practice in hospitals and similar institutions restricting membership, and therefore full staff privileges, solely to physicians."[8] Curiously, the sponsors of the act included only those institutions that provide services to the mentally disabled. By this restriction, they focused specifically on the issue of including psychologists for clinical staff membership with full staff privileges.

The problems that the American Psychological Association listed as a consequence of the restriction on psycholo-

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gists to admit their patients to hospitals *without* medical supervision were the following:

- Patients are denied freedom of choice in choosing a care provider; hospital care provided by a physician, who is different from the psychologist rendering outpatient care, necessarily results in a disruption in patients' continuity of care, a waste of resources, increased hospital costs, and restriction on the therapeutic services available; hospitals are currently run without "multidisciplinary involvement," there is no effective collaboration among different treating disciplines, and because of these restrictions, psychologists are denied access to leadership positions.[6]

POSITION OF THE APA

On March 16, 1988, the APA submitted a statement to the New York Senate and State Assembly standing committees, regarding the proposal to grant hospital privileges to psychologists and other non-medical mental health practitioners.[9] Two issues emerged as the basis of the APA's stand in opposition to the psychologists' claims: (1) the biologic and biochemical substrate underlying mental illness is, correctly, the professional territory of the psychiatric physician, by virtue of his/her medical training and experience, and (2) the psychiatric physician is uniquely qualified to deliver the specific quality of care required by the severely ill patient who requires in-hospital treatment.

The APA's position was then amplified to include the following points:

- Psychiatrists and psychologists are not competing providers of mental health services. Psychologists have none of the psychiatrist's medical training and experience. Their training is non-medical, graduate-school-based, and the required post-doctoral year is not necessarily hospital-based.
- Psychologists diagnose and treat patients with only psychological techniques and therefore do not have training or experience with the medical armamentaria necessary in the clinical management of hospitalized patients.
- Since there is a close relationship between mental health and physical health, with a wide variety of physical disease masquerading as psychiatric disorders, the presence of behavioral symptoms does not necessarily imply the presence only of a psychological disorder, and psychotherapy, or other psychological interventions, cannot be blindly applied without regard to diagnosis.
- The granting of "independent hospital practice" status to psychologists will *increase* the costs of providing mental health care because psychologists would be required to seek ongoing medical consultation, lengthening patient hospital stays and involving fees to more than one professional treating the patient.
- Most hospitals already grant psychologists membership to their "allied health," "ancillary," "affiliate," or "courtesy" staffs to permit them to provide care to their hospitalized patients.[9–12]

The right of hospitals to determine the composition of their medical staffs has been repeatedly upheld by the courts and mandated by the Joint Commission on Accreditation of Health-Care Organizations (JCAHO). Hospitals are responsible for the care given to their patients, and therefore hospitals exercise selective care in the composition of their medical staffs. Even physicians, simply because they are physicians, do not have the right to admit or treat their patients to any hospital at all.

DISCUSSION

In response to the challenge presented by spokespersons for the American Psychological Association, the APA has argued that mental illness, although manifested by aberrant behavior, is *disease* and therefore must be treated as such by those specifically trained in the treatment of disease-based processes, namely physicians. Unfortunately, although having some merit, this argument might be made more strongly by addressing underlying issues regarding the delivery of mental health care. If left unchallenged and unexamined, these very issues could lead to the discarding of the "science" of behavioral science, with the ultimate trivialization of psychotherapy and associated mental health care. The danger is that we may be about to do to psychotherapy and the mental health profession what has been done to many teachers and public education systems across the country—devaluing them through an anti-intellectual and bureaucratic homogenizing process, dispensing with all interdisciplinary distinctions.

It seems as if this is the approach taken by the psychologists. One example of important clinical and therapeutic features that are blurred in the polemical approach of the American Psychological Association can be seen in the generality of their statements regarding the hospital care of patients. Instead, it must be appreciated that the practice of hospital psychiatry is a very particular and complex form of care delivered to patients whose mental illness is severe enough to require 24-hour hospitalization. The hospital milieu is a specific treatment setting that encompasses a multiplicity of therapeutic options. It is not a one-dimensional system, as implied by the position papers presented by the psychologists. Furthermore, it is primarily a *medical milieu*. Acute and emergency clinical decisions must be made, dealing with inputs from diverse sources. Here, the physician has been trained and functions as the leader of a diagnostic-treatment *team*.[13]

Particularly today, when case management is seen by some as the means by which the containment of medical care costs can be accomplished along with improved health care delivery, the following question is raised: who, in a hospital setting, is best able to assess the patient and coordinate and monitor care? Who is most competent to document the treatment plan and the clinical decisions made, supply relevant information, and act as the conduit for informed consent, while providing liaison with a consultant staff as necessary? To date, because of both training and experience, this has been the role of the physician. In the specialty area of mental health care, it has been the role of the psychiatrist:

> "Psychiatry is a medical specialty. A psychiatrist is a physician educated and trained to provide direct service, leadership, and responsibility for the diagnosis, treatment, and management of those who have disorders of behavior, thought, feeling, or action . . . specifically trained in the simultaneous relevance of biological, psychological, and social factors."[14]

In this regard, it is important to note that at the root of the California Supreme Court's decision in June 1990 is the faulty *legislative* description of the scope of practice limitations of psychologists. Specifically, Section 2903 of the California Business and Professional Code merely states, without qualifications, that psychologists can "diagnose and treat." Nowhere does any wording reflect what this means nor what the educational and experiential prerequisites are on which such practice criteria rest. Here, poorly worded legislation and meagre regulations not only give the mistaken impression that the public is protected, but also mislead justices.[5]

It seems, from the actions of the American Psychological Association pertaining to the hospital-admitting-privilege issue, that similar weaknesses in legislative-appreciation of complex health-care issues is being played upon. For instance, in New York State, the State Psychological Association sent a letter to its membership conveying a message considerably different from what its public stance had been. In a review of *CAPP v Rank* by Charatan, this letter was cited and revealed the following statement regarding the motives underlying the demands of psychologists for unsupervised hospital admission privileges:

> The impact, if our goal is obtained, will go far beyond the practice of psychology in hospitals. This clarification of our status and role as fully autonomous professionals and independent providers will make us more competitive with other mental health professionals for jobs, promotions, and improved salaries and benefits."[15]

To legislators and the public, however, the published position of organized psychology seems to have camouflaged this claim for new professional "turf" by couching it in terms of a struggle for equality. Additionally, the authors of such position papers have asserted that without these changes in privileges, the public consumer will suffer. It seems that they have tailored the language of their demands to fit a tone consistent with a current populist spirit of anti-establishment "awakening" and cloaked their demands in pro-consumerist clothing.

To the authors here, the principal issue is one of professional boundaries, hinging on factors such as intrinsic training and experience and clinical expertise. It is far from an issue of the civil rights of a deprived or underprivileged group. However, it should be noted that for states like New York there is *no* common law or statutory law entitling *anyone*—psychiatriat, psychologist, or anyone else—to hospital privileges. Therefore, for New York State, the results of *CAPP v Rank* are irrelevant.[16]

To return to the question of the components and procedures of the hospital care of the mentally ill, organized psychology has suggested that by not allowing psychologists to admit their patients to hospitals directly, a waste of resources and an obstruction to the smooth delivery of care result. They have claimed that the admitting limitation creates "discontinuities" of care. We see this argument as erroneous in several ways. For example, "continuity-of-care" is not a simple issue, the presence or absence of which necessarily defines good clinical practice. In fact, there are, at times, specific indications for not only hospitalizing a patient, but also for interrupting the ongoing outpatient therapy and replacing the current practitioner with a new treatment team. Among these indications are (1) the presence of a medical-psychiatric illness in need of short-term stabilization with medications, as well as interpersonal intervention; (2) the presence of family, work, or community stressors that malignantly affect the individual's clinical course outside the hospital setting; and (3) the breakdown in effectiveness of ongoing outpatient therapy.

In the course of addressing these patient care needs, it is the general practice of many hospitals to give "emergency" or "temporary" hospital privileges to the patient's private practitioner so that an orderly procedure occurs in response to the specific and unique needs of a particular situation. This is especially true when a practitioner can present compelling clinical justification for not interrupting the ongoing treatment relationship. Under these circumstances, the outside practitioner may hold a PhD or comparable credential, and a responsible and systematic procedure for review and accountability is provided, ensuring that the best possible care is given within the broadest range of treatment modalities available. The issue, then, regarding "continuity-of-care" and "independent health care providers" is only, and specifically, about what these phrases mean, and under what category of services do they relevantly apply so that the best health care may be achieved.

We suggest that determining this is not accomplished by arguing about theories held dear by either the field of psychiatry or the field of clinical psychology but by a careful examination of the practitioner's training and experience that reveals important differential features between the two professions. These substantive features can then be matched with the expertise needed for the relevant health care required within the overall delivery system.

It should be noted that neither psychiatry nor psychology represent a stagnant field. Neither has established a lock on "the truth" with regard to the data of the behavioral sciences. Neither is mutually exclusive of the other, and neither is sufficient without the other. However, they are not interchangeable disciplines! They are complementary. They overlap in places. They certainly cross-fertilize each other. However, they are not identical. Psychologists do share with psychiatrists an interest in the broad field of the behavioral sciences, but this does not mean that their distinctive training, experience, or expertise is interchangeable.

Treatment in a hospital setting is structured in terms of a "medical team" organization. There is no opposition to the integration of diverse but complementary disciplines in the organized management of patient care. It is, however, organized in a "medical" context. If the desire is now to reorganize this system of care delivery, one has to rethink certification procedures so that the requisite degree of quality care can be assured. Certification must be determined by academic training and supervised experience, according to *what* is being certified. For instance, the designation of "qualified psychologist" gives no real, pertinent information to the administrating body of the health care facility regarding subspecialty expertise and level of clinical competence.

The question at hand pertains to the practical delivery of care to a patient population in a unitary and coherent fashion, incorporating the best of all that is known regarding the clinical problem. Ideological splits must be worked out in terms of substantive clinical and research issues not via a process of advocacy, where patients themselves may become inadvertant participants in the struggle.

The purpose of the December 1987 issue of the *American Psychologist* (the official journal of the American Psychological Association) was to summarize a conference held at the University of Utah examining and questioning the whole system of graduate education in psychology.[17] As these proceedings readily reflect, psychology is not a unitary discipline. Therefore, if we are to support the privilege to admit patients to hospitals by psychologists, it is essential to answer the critical question: What is the common core training of a "psychologist" by which hospital authorities can determine what qualifications have been achieved that are consistent with what curriculum and what standard of care? In order that hospitals may have some control over the quality of care they provide, how are they to evaluate a practitioner's professional training in psychology to ensure an adequate expertise to treat patients?

Within the American Psychological Association itself, the ranks of academic, research, and clinical psychologists are wrestling with each other.[17] Furthermore, Holden[18] notes that a splinter group of psychologists has become increasingly frustrated with their national organization. They have protested that a disproportionate amount of the organization's resources have been directed into guild issues such as reimbursement, prescription-writing privileges, hospital admission privileges, and licensing and certification for clinical psychologists.

Legislative recourse to the resolution of the territorial issue (and the unspoken economic one) propounded by the American Psychological Association is neither sound nor appropriate for the reasons noted above. A full understanding of the differential characteristics of the two disciplines and an appreciation of the complex and subtle relationships "between the apparent clinical presentation and the treatment or management strategy prescribed"[19] needs to be addressed.

Kissick points out that systems need methods for evaluating the performance of their professionals, and as patients become subscribers and beneficiaries of such systems, there is an increasing degree of reciprocal accountability between those seeking care and those providing it.[20] As aspects of the psychiatrist's role in society continue to evolve, we must not lose sight of the important qualities we possess and the distinctions that make our specialty a "profession." No marketplace pressures nor industry's mentality should be allowed to cloud these important considerations. Under all circumstances, we must avoid placing our patients in the position of middle-men, courted as advocates for one side or the other, and caught between the two warring factions in an emotional tug-of-war.

CONCLUSIONS

The mental health care delivery systems in this country have traditionally been medically based and interdisciplinarilly integrated. Within these systems, it is generally agreed that case-management is a desirable feature and that overall responsibility for such should fall on those with the broadest perspective and training.

Legislative fiat is neither the route for thoughtful examination and evaluation of a complex social system, such as mental health care delivery, nor is it necessarily the best protector of society's interests.

Emotional claims for equality in "privileges" by law rather than by demonstrated criteria of training and competence, denial of alternative motives behind the public outcry for "fairness," self-identification as victim of an authoritarian and paternalistic superstructure, and unidimensional and simplistic criticism of isolated issues taken out of their larger context are all nonproductive and unprofessional ways of proceeding to affect changes.

Psychiatry and psychology are different disciplines with respect to focus, training, experience, and societal expectations. Although there is overlap and cross-fertilization between them, they are distinct disciplines.[21] It follows therefore that the simplistic formula, "fusing" an internist and a psychologist to "equal" a psychiatrist, is not only misleading, but it also fails to reflect a realistic appreciation of the disciplines involved and of the requirements of the health care delivery systems.

The hospital milieu is structured along the lines of a "medical team" model, and as in all medical care systems, must have inherent mechanisms for evaluating and monitoring its constituent parts. Hospital medical boards, for instance, must have consistent and reliable criteria by which they can evaluate who might be best suited for staff participation when measured against quality-of-care and accountability standards. Additionally, as Kissick noted,[20] although there are standards set by the JCAHO requiring hospitals to consider allied professionals for medical staff privileges, to the extent permitted by state law, there is concomitantly an increasing trend toward "incorporization"—the "corporation" being a social and economic tool in our society needing to have its own coherent infrastructure.

Psychologists are a very heterogeneous group of professionals, with considerably different graduate school tracks (some clinical, some educational, others experimental or academic) without one uniform set of criteria available for assessing competence. In contrast, medical school training, internship, and residency programs of the psychiatrist are specifically geared to training for the assumption of clinical responsibility in a variety of settings. Within certain limits, these training programs contain core curricula, the content of which is reasonably consistent from training program to training program. These curricula are subject to a tradition of assessment by measures of academic and clinical competence that are used in common across different programs.

To the contrary of what the psychologists claim in their position paper, continuity-of-care is not a political issue. Instead, it is a treatment issue that remains to be decided on a case-by-case basis. It should not be confused, either directly or by manipulative inference, with quality-of-care issues, which apply to all cases.

Patient care and interdisciplinary management are at the heart of the training of the psychiatrist. They are not added on adjunctively but are an integral part of the intensive and lengthy training program, encompassing both the biological and psychological approaches to understanding and treating mental disorders.

REFERENCES

1. *Virginia Academy of Clinical Psychologists v Blue Shield of Virginia,* 469 FSupp 552 (ED Va 1979).
2. *California Assoc of Psychology Providers v Rank,* 214 Cal App 3d 1207, 247 C 641 (Cal App 2 Dist 1988), *rev'd* 51 Cal 3d 1, 270 C 796 (1990).
3. Plows CW: *CAPP v Rank. Medical staff privileges for clinical psychologists: Legal Memorandum, California Medical Association:* December 20, 1990.
4. California Court of Appeal: *CAPP v Rank B020113:* June 2, 1988.
5. Supreme Court. State of California: *California Assoc of Psychology Providers v Rank,* 51 Cal App 3d 1, 270 C 796 (1990).
6. New York State Psychological Association: *The Independent Practice of Psychology in Hospitals: Advantages to Professionals & Consumers* May, 1986.
7. Welch B: *Testimony Regarding S.2818 & A.3718 on Behalf of the American Psychological Association* Before the New York Senate Standing Committee on Mental Hygiene and New York Assembly Standing Committee on MH, MR, and DD: March 16, 1988.
8. Senators Spano and Padavan and Member of the Assembly Connelly: *Sponsor's Memorandum S.2818/A.3718.*
9. Sabshin M: *Statement of the American Psychiatric Association on Hospital Privileges for Psychologists and Other Mental Health Professionals S.2818/A.3718* Before the New York Senate Standing Committee on Mental Hygiene and the New York Assembly Standing Committee on MH, MR, and DD: March 16, 1988.
10. Sabshin M: *Letter to Dr Wm Mayer, Department of Defense* October 31, 1988.
11. Sabshin M: *Statement of the American Psychiatric Association on Hospital Staff Privileges for Psychologists SB 638* Before the State of Maryland Senate Finance Committee: March 2, 1989.
12. American Psychiatric Association/American Medical Assocation: *Amici Brief Before the Supreme Court of California,* S002524: January 9, 1989.
13. Board of Trustees, American Psychiatric Association: Position statement on hospital privileges for psychologists. *Am J Psychiatry* 1971; 127:1456.
14. Fink P: Response to the Presidential Address: Is "biopsychosocial" the psychiatric shibboleth? *Am J Psychiatry* 1988; 145:1061–1067.
15. Charatan FB: A review of CAPP v Rank. *The Bulletin,* Area II Council, APA, 1988; 31:1.
16. New York Public Health Law 2801-B (McKinney 1991).
17. Spence JT: Centrifugal vs centripetal tendencies in psychology. *Am Psychol* 1987; 42:1052–4.
18. Holden C: Research psychologists break with APA. *Science* 1988; 241: 1036.
19. Gutheil TG: Medicolegal pitfalls in the treatment of borderline patients. *Am J Psychiatry* 1985; 142:9–14.
20. Kissick W: A glimpse into the 21st century. *Pa Med* 1988; :19–23.
21. Kingsbury S: Cognitive differences between psychologists and psychiatrists. *Am Psychol* 1987; 42:152–156.

Recombinant tissue-plasminogen activator for acute Budd-Chiari syndrome secondary to paroxysmal nocturnal hemoglobinuria

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Paroxysmal nocturnal hemoglobinuria (PNH) is an acquired hemolytic anemia characterized by increased sensitivity of erythrocytes to complement-mediated lysis. The increased erythrocyte susceptibility to complement in PNH is believed to be the result of a membrane defect.[1] The leading cause of death in patients with PNH is the formation of abdominal intravascular thrombus.[2] The platelets in patients with PNH are also complement-sensitive and may be partly responsible for the thrombus formation.[3] Budd-Chiari syndrome in PNH is well documented and is almost always fatal.[4,5,6] In the experience of Hartmann et al,[2] nine out of 32 patients with PNH were found to have Budd-Chiari syndrome. Thrombolytic therapy using streptokinase or urokinase[7,8] has been used in the past for this complication. We report a patient with transient improvement of the symptoms and signs of hepatic vein thrombosis after administration of recombinant tissue-plasminogen activator (rt-PA).

CASE REPORT

A 47-year-old women of East Indian ancestry from Guyana who presented in 1985 with hemolytic anemia. A diagnosis of PNH was subsequently made on the basis of a positive sucrose-water test and a Ham test. The patient remained well until seven days before this admission, when abdominal pain with increasing abdominal girth, and yellowing of her eyes developed.

On admission, the physical examination showed a markedly jaundiced patient with ascites and a prominent venous pattern. Examination of the extremities showed 2+ leg edema. Pertinent laboratory data included a white blood cell (WBC) count of 14,800/mm^3; hematocrit, 28.9%; platelet count, 80,000/mm^3; prothrombin time, 17.9 sec; albumin, 2.4 mg/dL; alkaline phosphatase, 192 U/L; serum glutamic oxaloacetic transaminase (SGOT), 1,020 U/L; serum glutamic pyruvic transaminase (SGPT), 1,860 U/L; total bilirubin, 15.0 mg/dL; direct bilirubin, 14.2 mg/dL; and lactate dehydrogenase, 1,691 U/L.

Paracentesis showed a WBC count of 1,050/mm^3; red blood cell count, 3,250/mm^3; amylase, 50 mg/dL; protein, 3.0 mg/dL. Gram stain, and bacterial and mycobacterial cultures were negative. An abdominal sonogram showed no evidence of abnormality of the biliary tree. A liver scan with ^{99}Tc revealed diminished uptake of both hepatic lobes, with only the caudate remaining relatively normal. The hepatic venogram showed a compressed inferior vena cava at the level of the liver and a "spider web" pattern in the caudate lobe consistent with the Budd-Chiari syndrome (Fig 1).

The patient was given a total of 100 mg of rt-PA (Activase, Genentech, Inc, South San Francisco, CA) intravenously over a three hour period. Heparin was given concomitantly with rt-PA but was discontinued six hours later because of a hemorrhagic diathesis. Over the next 24 hours, the ascites, pedal edema, urine output, body weight, and hepatic enzymes markedly improved (Fig 2). However, ten days later, the patient's clinical status began to deteriorate, with worsening of the ascites and hepatic enzyme levels. A portal-systemic shunt was performed, but the patient expired.

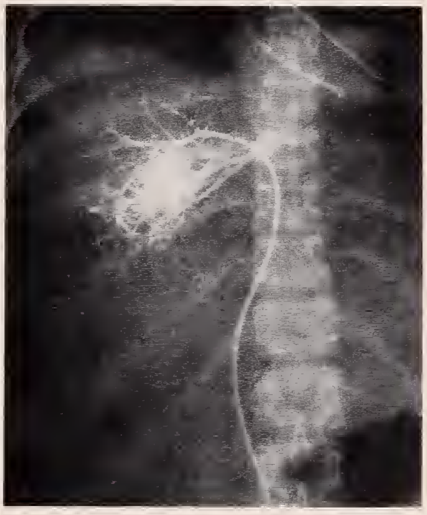

FIGURE 1. Hepatic vein angiography showing the "spider-web" pattern.

DISCUSSION

Treatment of patients with acute Budd-Chiari syndrome has historically involved surgery, mainly liver decompression by a shunting procedure or anticoagulation using high dose heparin.[9] Thrombolytic therapy such as streptokinase and urokinase has been used for treatment of this syndrome, but with a varied success rate.[7,8] rt-PA has several theoretical advantages. First, rt-PA is clot-selective because it requires the presence of the plasminogen-fibrin complex for avid binding to occur. Second, rt-PA has a very short half life, about five minutes, and may be discontinued in the event of bleeding. Third, rt-PA is a natural human enzyme with no known allergenicity.[10]

Administration of rt-PA was followed by dramatic clinical and biochemical improvement. Alternatively, the improvement could be attributed to spontaneous thrombolysis. Although a follow-up hepatic venogram was not done, the patient's status remained stable after rt-PA administration, suggesting drug efficacy.

Since there was no precedent for the use of rt-PA in the Budd-Chiari syndrome, the dose administered was that commonly used for acute coronary thrombosis, which may greatly differ from that required for hepatic vein thrombolysis. Drug intervention occurred at approximately two weeks after the onset of symptoms, and this too may have precluded complete clot resolution. In addition, the premature termination of

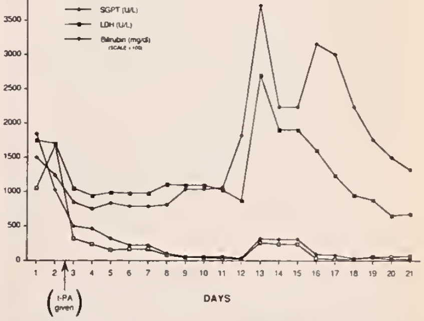

FIGURE 2. Liver function parameters before and during rt-PA administration.

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heparin could theoretically have increased the chance of re-occlusion, as suggested by the recent thrombolytic trials in myocardial infarction.[11,12]

It is clear that rt-PA did not worsen the patient's course. Certainly the transient improvement in this patient would justify further clinical trails of rt-PA, especially in view of the refractory nature of Budd-Chiari syndrome to standard therapy.

References

1. Dessypris EN, Clark DA, Mckee Jr C, et al: Increased sensitivity to complement of erythroid and myeloid progenitors in paroxysmal nocturnal hemoglobinuria. *N Engl J Med* 1983; 309:690–693.
2. Hartmann RC, Luther AB, Jenkins D Jr, et al: Fulminant hepatic venous thrombosis (Budd-Chiari syndrome) in paroxysmal nocturnal hemoglobinuria: Definition of a medical emergency. *John's Hopkins Med J* 1980; 146:247–254.
3. Peytremann R, Rhodes RS, Hartmann RC: Thrombosis in paroxysmal nocturnal hemoglobinuria (PNH) with particular reference to progressive, diffuse hepatic vein thrombosis. *Series Haematologica* 1972; 5:115–36.
4. Valla D, Dhumeaux D, Babany G, et al: Hepatic vein thrombosis in paroxysmal nocturnal hemoglobinuria: A spectrum from asymptomatic occlusion of hepatic venules to fatal Budd-Chiari syndrome. *Gastroenterology* 1987; 93:569–575.
5. Horler AR, Shaw MT, Thompson RB: Budd-Chiari syndrome as a complication of PNH. *Post Grad Med J* 1970; 46:618–622.
6. Falchuk ZM, Leventhal BG: Budd-Chiari syndrome in a patient with paroxysmal nocturnal hemoglobinuria. *Am J Dig Dis* 1973; 18:900–904.
7. Greenwood LH, Yrizarry JM, Hallett JW Jr, et al: Urokinase treatment of Budd-Chiari syndrome. *AJR* 141:1057–1059.
8. Sholar PW, Bell WR: Thrombolytic therapy for inferior vena cava thrombosis in paroxysmal nocturnal hemoglobinuria. *Ann Intern Med* 1985; 103:539–541.
9. Rosse WF: Treatment of paroxysmal nocturnal hemoglobinuria. *Blood* 1982; 60:20–23.
10. Tissue-type plasminogen activator for acute coronary thrombosis. *Med Lett* 1987; 29:107–109.
11. Hsia J, Hamilton WP, Kleiman N, et al: A comparison between heparin and low dose aspirin as adjunctive therapy with tissue plasminogen activator for acute myocardial infarction. *N Engl J Med* 1990; 323:1433–1437.
12. Bleich SD, Nichols T, Schumacher R, et al: The role of heparin following coronary thrombolysis with tissue plasminogen activator (t-PA). [abstract] *Circulation* 1989; 80(suppl II):13.

Gouty arthritis following gunshot wound: A case report

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The association of lead toxicity and gout is well established.[1,2] Sources of lead toxicity in adults are usually divided into two major categories: occupational exposures (including mining, welding, working with storage batteries) and environmental exposures ("moonshine alcohol," paint remover, food and water contamination, ceramics and possibly even crystal decanters and glasses.)[3] Saturnism resulting from lead bullets, although known for some time, is a rare phenomenon, and in none of the reported instances has been gout identified as a complication.[4–6] We present here what appears to be the first report of a case of gouty arthritis associated with retained bullets and elevated blood lead levels.

Case Report

A 47-year-old male presented to our clinic complaining of pain and swelling in left knee, ankle, elbow, and wrist for one week. He denied fever, headaches, nausea, vomiting, abdominal pain, or diarrhea. He had been having intermittent episodes of joint pains and swelling for approximately ten years, occurring every few months and lasting several days, with some response to ibuprofen. The patient had suffered multiple gunshot wounds 12 years earlier.

The patient was married with three children and had stopped working as a truck driver since sustaining the gunshot wounds. He acknowledged moderate alcohol consumption (four to six beers per week) but denied smoking or intravenous drug abuse. There was no family history of gout and no known history of exposure to environmental sources of lead.

On physical examination, he appeared in distress because of pain. His vital signs were within normal limits; weight was 70 kg, and height 172 cm. The left knee was swollen with a large effusion and was warm and tender, with significant reduction in range of motion. The left wrist, elbow, and ankle were also tender and moderately swollen.

The remainder of the physical examination was remarkable only for gunshot scars over the right chest and forearm. A left knee arthrocentesis yielded over 80 mL of cloudy, straw-colored fluid with 4.2×10^9/L white cells, 96% polys, 4% lymphs. On polarizing microscopy, many intracellular needle-shaped crystals were identified, with negative birefringence.

Other laboratory results included a white blood cell count of 10.8×10^9/L with 80% polys, 3% bands, and 16% lymphs; hematocrit was 43.5%. The mean corpuscular volume was 94 fL; the platelet count was 381×10^9/L, and the erythrocyte sedimentation rate was 107 mm/h. The uric acid level was 541 μmol/L (normal: 208–476 μmol/L); blood urea nitrogen was 5.7 mmol/L (normal: 2.9–8.9 mmol/L); and creatinine was 79 μmol/L (normal:53–133 μmol/L). Electrolytes and liver enzymes were normal.

Chest X-rays showed numerous pellets in the right chest wall and lung (Fig 1); X-rays of other joints were unremarkable.

A 24-hour urine collection for uric acid showed a total output of 700 mg/dL (4.14 mmol/dL) (normal:250–750 mg/dL [1.48–4.43 mmol/dL]). The blood lead level was 30 μg/dL (1.44 μmol/L) (normal: <25 μg/L [<1.2 μmol/L]); and 31 μg/dL (1.49 μmol/L) 6 weeks later. A free erythrocyte protoporphyrin blood measurement was 29 μg/mL (normal: 0–34 μg/mL). The patient's wife, and children had normal blood lead levels.

Discussion

This patient represents a typical case of gout with an unusual association, retained bullets presumably leaching lead as reflected in an increased blood lead concentration but without overt lead toxicity.

Blood lead levels, the single best test for assessing the likelihood that a clinical syndrome is related to lead,[7] were mildly but persistently elevated. There was no evidence of encephalopathy, abdominal pain, anemia, basophilic stippling, or renal dis-

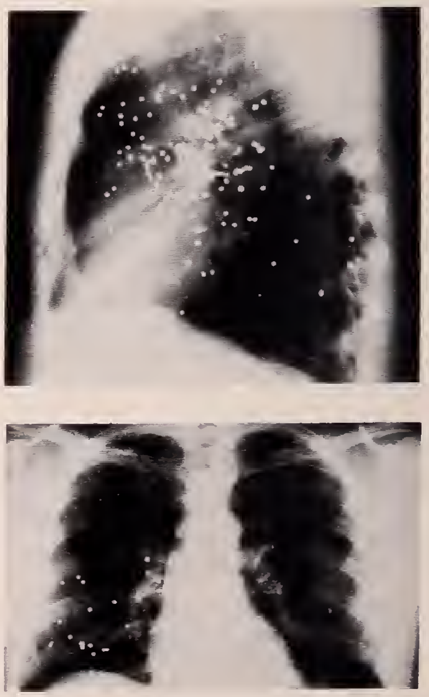

FIGURE 1. PA and lateral of the chest showing multiple pellets in right chest wall and lung.

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ease because these findings are rare in individuals with levels below 40 µg/dL.[7]

Lead intoxication from gunshot wounds has mostly been related to absorption of the metal from bullets that are retained within joints because the synovial fluid has been shown to enhance lead dissolution.[10] Nevertheless, there are reports of patients[11] who sustained multiple pellet gunshot wounds without concomitant joint involvement, as in our patient, and who developed lead toxicity as well. The large surface area of the numerous pellets may have favored increased absorption.

Environmental exposure as a cause of high lead levels in this patient was unlikely in view of a negative history and normal lead levels in his children and wife. Can the elevated lead level be postulated in this case as a contributing factor for the appearance of gout, even in the absence of systemic toxicity? We believe so, on the basis of the following evidence.

Subclinical lead toxicity as a cause of gout is not a new concept.[9] At least two mechanisms have been postulated. First, lead is thought to induce hyperuricemia, even in the absence of renal failure, by inhibiting tubular urate transport and decreasing urate excretion.[1] This mechanism could be operative in our patient, who had "normal" urinary excretion of urate in the face of significant elevation of serum uric acid. Second, there is some evidence suggesting that lead may enhance the crystallization of uric acid.[8]

It is of interest that patients with gout and no history of lead exposure were found to have higher blood lead levels than age- and sex-matched controls.[9] More recently, a group of patients with gout, again without history of lead exposure, was found to have greater ethylenedinitrilotetraacetc acid lead excretion values than subjects with neither gout nor lead exposure.[12] All this suggests that even minimal elevations of blood lead could be involved in the pathogenesis of apparently primary gout. This is a concept that has not been fully explored and deserves further investigation.

In conclusion, this case is of interest because of the association of gouty arthritis with a poorly recognized predisposing factor, "subclinical" lead toxicity, related to an unusual source, retained bullets. This, in fact, may not be so unusual in our increasingly violent society.

REFERENCES

1. Poor G, Mituszova M: Saturnine gout. *Baillieres Clin Rheumatol* 1989; 3:51–61.
2. Halla JT, Ball GV: Saturnine gout: A review of 42 patients. *Semin Arthritis and Rheum* 1982; 11: 307–314.
3. Graziano JH, Blum C: Lead exposure from lead crystal. *Lancet* 1991; 337:141–142.
4. Switz DM, Elmorshidy ME, Deyerle WM: Bullets, joints and lead intoxication. A remarkable and instructive case. *Arch Intern Med* 1976; 136:939.
5. Windler EC, Smith RB, Bryan WJ, et al: Lead intoxication and traumatic arthritis of the hip secondary to retained bullet fragments. A case report. *J Bone Joint Surg* 1978; 60:254.
6. DiMaio VJM, Garriot JC, DiMaio SM, et al: A fatal case of lead poisoning due to a retained bullet. *Am J Forensic Med Pathol* 1983; 4:165.
7. Cullen MR, Robins JM, Eskenazi B: Adult inorganic lead intoxication: Presentation of 31 new cases and a review of recent advances in the literature. *Medicine* 1983; 62:221–247.
8. Tak HK, Wilcox WR, Cooper SM: The effect of lead upon urate nucleation. *Arthritis Rheum* 1981 24:1291–1295
9. Campbell BC, Moore MR, Goldberg A, et al: Subclinical lead exposure: A possible cause of gout. *Br Med J* 1978; 2:1403.
10. Leonard MH: The solution of lead in synovial fluid. *Clin Orthop Rel Res* 1969; 64:255.
11. Glakin VM: Case of acute lead poisoning after a gunshot wound. *Klin Med* 1972; 50:145–147.
12. Craswell PW, Price J, Boyle PD, et al: Patterns of lead excretion in patients with gout and chronic renal failure: A comparative German and Australian study. *Sci Total Environ* 1987; 66:17–28.

Facial necrotizing fasciitis due to *Streptococcus pyogenes*

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Necrotizing fasciitis was described in detail by Meleny[1] in 1924 as an entity separate from erysipelas and synergistic gangrene. It is a mixed anaerobic or aerobic infection that dissects deep fascial planes rapidly. Common predisposing factors include diabetes, antecedent injury to soft tissue, abdominal surgery, pilonidal cyst, perirectal abscess, and decubitus or diabetic ulcers. It may become a rapidly progressive, life-threatening infection if the diagnosis is not made promptly and if antibiotic or adjuvant surgical treatment is delayed.[2-4] The syndrome occurs most commonly in the extremities or abdomen[2,3] and has only rarely been described as involving the face or oral structures.[2-7] We present a report of two cases of facial necrotizing fasciitis caused by *Streptococcus pyogenes*.

CASE REPORT

Case 1. A 52-year-old man was admitted for erythema of his left ear that had progressed over 16 hours to involve both sides of the face and neck. The patient denied any history of facial trauma or infection. He had had influenza-like symptoms during the five days before admission, for which he took aspirin, outdated ibuprofen tablets, and an unknown "decongestant." His medical history was unremarkable.

At initial presentation, the patient's oral temperature was 105.8°F (41.0°C), his pulse was 144/min, and his blood pressure was 94/40 mm Hg. The facial findings included severe generalized erythema and induration and edema of both eyelids, forehead, and cheeks. The affected area was warm and non-tender. The lips and oral mucosa were edematous. Blisters, which oozed a clear fluid, were present over the right side of the face. The conjunctivae were swollen. Speculum examination of the nasal cavity and examination of the oropharyngeal cavity did not reveal abscesses. No other significant physical findings were present.

On admission, the white blood cell (WBC) count was 6,000/mm^3 with 13% band forms; the rest of the laboratory values were within normal limits.

The initial diagnosis was drug reaction, and a single dose of dexamethasone, 16 mg intravenously (IV), was given. Within the first 16 hours of his hospital stay, he received ceftazidine, 500 mg IV every 12 hours, following a 1 g loading dose, and vancomycin, 500 mg IV every 12 hours, following a 1 g loading dose. The erythema and edema progressed, and the patient developed respiratory insufficiency. Tracheotomy was performed, and the patient was placed on ventilator support.

On day two, the WBC count was 14,000/mm^3. The respiratory insufficiency continued, and disseminated intravascular coagulation developed. Gangrene appeared in the eyelids, cheeks, and chin (Fig 1). Penicillin, 5,000,000 units IV every six hours, was begun.

Blood cultures taken on admission were positive for Group A *S pyogenes*. The skin biopsy from the margins of the lesion showed extensive epidermal and dermal necrosis associated with multiple fibrin thrombi within small blood vessels, extravasation of red blood cells, and colonies of gram-positive cocci. A culture of the wound yielded Group A *S pyogenes*, sensitive to vancomycin and penicillin. The Bactec™ culture system was used; species identification was performed using the Vitec™ system; antimicrobial susceptibility was established using growth curves.

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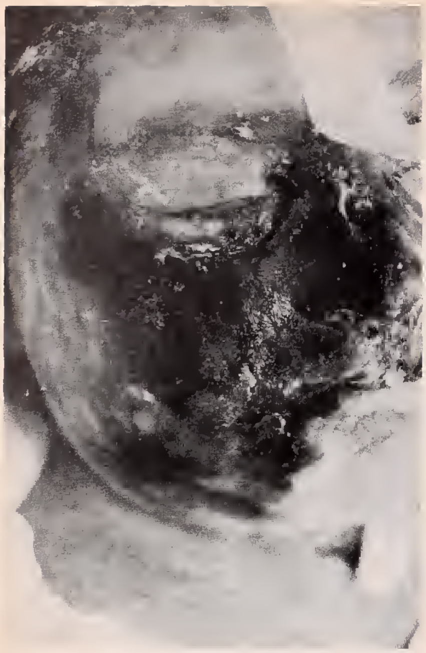

FIGURE 1. Case 1: Gangrenous changes involving the face, nose, and lips.

The patient was referred to the reconstructive surgery service. After stabilization of his medical condition, including the coagulopathy, and weaning from the respirator, he received debridement and repair of defects caused by the facial gangrene.

Case 2. A 67-year-old woman came to the emergency room because of facial edema and erythema. Three days before, she had influenza-like symptoms. The patient had had a left mastectomy ten months previously for breast cancer; she had declined chemotherapy or radiotherapy. On the day of admission she had nausea and vomiting and had taken outdated prochlorperazine. She noticed erythema of the lower left eyelid. This rapidly progressed to edema, closing the left eye. The patient noted that the edema and erythema spread to her forehead and right eyelid within three hours.

Examination revealed an obese women with an oral temperature of 101°F (38.3°C), blood pressure of 130/80 mm Hg, respiratory rate of 22/min, and a pulse rate of 80/min. The entire face was erythematous and edematous. Both eyes were closed caused by the edema. The patient had a purulent discharge from a pustule on the left lower eyelid. No nodes were palpable. The liver was enlarged and extended to 3 cm below the right rib margin. No other physical abnormalities were present. On admission, the WBC count was 6,600/mm^3, and the serum potassium level was 2.8 mEq/L; there were no other laboratory abnormalities.

On admission, the patient was treated with cefoxitin, 1 g IV every six hours, a single dose of gentamicin, 120 mg IV, and prednisolone, 125 mg IV as a loading dose, followed by 60 mg every eight hours. The admitting diagnoses were cellulitis or drug reaction. Within 24 hours after admission, the patient was afebrile and had only minimal facial pain.

Blood cultures (Bactec™) taken at admission remained sterile. A culture of the pustule yielded Group A *S pyogenes* (Vitek™). Computed tomography of the head showed an air-fluid level in the left sphenoid sinus and left paraseptal cellulitis. A bone scan was performed, with results consistent with metastatic spread of the patient's tumor to the cervical spine, left sternoclavicular joint, and lumbar spine.

The edema and erythema had resolved by hospital day five. By day six, there was no purulent discharge. On day seven, the facial lesions had improved to the point where there was a residual 5 × 2-cm area of necrosis on her left lower eyelid and marked vascularity on both cheeks (Fig 2). Intravenous antibiotics were stopped on day six, and oral therapy with ciprofloxacin was begun. The patient was discharged on day seven.

DISCUSSION

In the early stages of necrotizing fasciitis, diagnosis may be difficult, yet the prognosis depends on how quickly the diagnosis is made. If treatment is delayed, the infection rapidly spreads, dissecting through tissue planes, ultimately involving the subcutaneous tissue and fascia, usually within 48 hours. Mortality rates may be as high as 30% when there is a delay in diagnosis or treatment. The most significant disease related to necrotizing fasciitis is diabetes mellitus, which can significantly increase mortality.[8] Death is usually due to sepsis, respiratory failure, and multi-organ failure. Early reports described hemolytic streptococci as the causative agent of necrotizing fasciitis. However, sophisticated culture techniques have shown that several bacterial species, including anaerobes, can underlie the syndrome.[9]

Brun-Buisson et al[10] and Krige et al[11] have described an association between the use of non-steroidal anti-inflammatory drugs (NSAIDs) and the occurrence of necrotizing fasciitis. Krige et al[11] suggested that depression of lymphocyte number and function associated with use of diflunisal might have contributed to the occurrence of fasciitis in their patient. One of the patients described here had been using NSAIDs (aspirin and ibuprofen) at the onset of his fasciitis. These drugs are used very commonly in the general population. Therefore, we do not believe this case alone proves that use of NSAIDs facilitates onset of fasciitis.

Because of the high mortality rate of necrotizing fasciitis, it must quickly be differentiated from erysipelas, acute cellulitis, and synergistic gangrene. The lesion of erysipelas is hot, bright red, and elevated; its margins are sharply demarcated.[12] The lesions in acute cellulitis are red and warm; the borders are not elevated or well defined. Acute cellulitis is almost always associated with leukocytosis.[12] Synergistic gangrene is a gangrenous ulceration of the skin caused by a mixed bacterial infection; it usually occurs following abdominal infection, trauma, or surgery. The lesion is red, tender, and swollen, with a painful, superficial ulcer surrounded by a rim of gangrenous skin. The latter, in turn, is encircled by a zone of purple erythema, which blends into a peripheral pink edematous area. The process extends slowly but progressively and may result in enormous ulcerations.[12]

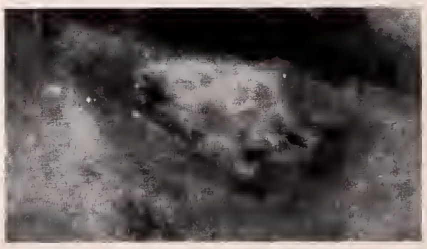

FIGURE 2. Case 2: Skin changes, left lower eyelid.

Necrotizing fasciitis usually occurs in the extremities or abdomen; rarely does it involve the face.[2–7,13] In the head and neck, the structures most commonly affected are the eyelids. This was the presentation in one of the cases presented here. The infection spreads with alarming rapidity; a whole limb may be involved within 24 hours. In these two cases, the whole face was involved in less than 18 hours. Generally, fluid-filled pustules will appear, followed by frank gangrene. The velocity and outcome of necrotizing fasciitis is due to the involvement of blood vessels with thrombosis, subcutaneous necrosis, and collagen fragmentation.[5] Surgical debridement of necrotic tissue is important in treatment of necrotizing fasciitis.[2–4,12] Swartz and Weinberg[12] recommended that all necrotic fat and fascia be removed, with the wound left open, and noted that "second-look" procedures may prove to be necessary.

In the cases of necrotizing fasciitis reported here, the presentation was unusual. The face is not commonly involved in this syndrome. Neither patient was diabetic or on immunosuppressants; neither had a history of facial trauma or dental disease. Both had a history of ingestion of outdated medication and one had been using NSAIDs. The second patient had a malignancy. The differential diagnoses in both cases included allergic reactions, erysipelas, acute cellulitis, and synergistic gangrene. Cellulitis and erysipelas, if untreated, may lead to necrotizing fasciitis with sepsis. When immunofluorescence techniques become widely available it may be possible to confirm suspected streptococcal infections early and start therapy, improving the prognosis. The cutaneous signs of streptococcal disease may be atypical, making correct diagnosis and treatment more difficult.

Late recognition of the necrotizing fasciitis, combined with its aggressive nature, is responsible for the current high mortality and morbidity rate, in spite of use of modern antibiotic therapy. Failure to seek medical attention early is a key factor in the development of the disease, with subsequent sepsis, acute renal failure, and respiratory failure. Therapy in both of the present cases was adequate, but the difference in outcome was associated with the speed with which treatment was begun. The second patient immediately received antibiotic therapy and had a much better response.

REFERENCES

1. Meleny FL: Hemolytic streptococcus gangrene. *Arch Surg* 1924; 9:317–364.
2. Casali RE, Tucker W, Petrino R, et al: Post-operative necrotizing fasciitis of the abdominal wall. *Am J Surg* 1980; 140:787–790.
3. Freeman HP, Oluwole SF, Ganepola GAP, et al: Necrotizing fasciitis. *Am Surg* 1981; 18:416–431.
4. Seal D, Leppard B, Widdowson J, et al: Necrotizing fasciitis due to Streptococcus pyogenes. *Br Med J* 1980; 280:1419–1420.
5. Bahna M. Canalis RF: Necrotizing fasciitis (Streptococcal gangrene) of the face. *Arch Otolaryngol* 1980; 106:648–651.
6. Kubo S, Abe K, Oka M: Necrotizing fasciitis of the middle third of the face: A case report. *J Craniomaxillofac Surg* 1989; 17:92–95.
7. Reifler JF III: Necrotizing fasciitis of the lip due to group A streptococcus. *Infections Med* 1991; 7:16–23.
8. Rea WJ, Wyrick WJ: Necrotizing fasciitis. *Ann Surg* 1970; 172:d957–964.
9. Guiliano A, Lewis F, Hadley K, et al: Bacteriology of necrotizing fasciitis. *Am J Surg* 1977; 134:52–57.
10. Brun-Buisson CL, Saada M, Trunet P, et al: Haemolytic streptococcal gangrene and non-steroidal anti-inflammatory drugs. *Br Med J* 1985; 290: 1786.
11. Krige JEJ, Spence RAJ, Potter PC, et al: Necrotizing fasciitis after diflunisal for minor injury. *Lancet* 1985; 2:1432.
12. Swartz MN, Weinberg AN: Infections due to gram-positive bacteria, in Fitzpatrick TB, Eisen AZ, Wolff K, et al (eds): *Dermatology in General Medicine*, 3rd ed. New York, McGraw-Hill, 1987, pp 2184–2188.
13. Lloyd RE: Necrotizing fasciitis of the orbit. *Br J Oral Maxillofac Surg* 1987; 25:323–327.

Pneumococcal cellulitis and dysgammaglobulinemia

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In spite of the availability of effective antibiotic therapy, *Streptococcus pneumoniae* continues to be an important cause of morbidity and mortality.[1–3] Pneumococcal infection of the skin, however, is extremely rare.[4–12] We describe a case of this rare infection, and present a review of the relevant literature.

CASE REPORT

A 69-year-old man presented with sudden onset of pain and swelling of his right foot associated with chills and fever. The patient's medical history included chronic lymphocytic leukemia (CLL) with hypogammaglobulinemia diagnosed five years before this admission, and type II diabetes mellitus, treated with glyburide 5 mg/day, diagnosed three years before admission. He had been treated with oral chlorambucil, 5-10 mg/day for several years for the CLL. A few months before admission, intravenous cyclophosphamide, 400 mg/day, was administered for five days, when he became unresponsive to chlorambucil. However, he was not on any chemotherapy at the time of admission. His dysglobulinemia had progressed from 2.1 g/dL of serum globulin with moderately depressed gamma globulin on serum protein electrophoresis, at the time of initial diagnosis of CLL, to 1.8 g/dL of serum globulin with severely depressed gamma globulin on serum protein electrophoresis, a few months before admission.

At the time of presentation, he was awake but disoriented. He had a temperature of 39°C; pulse, 90 beats/min; respiratory rate, 20/min. The blood pressure was 110/60 mm Hg. Examination of the head and neck was unremarkable. No lymphadenopathy was detected. The abdomen was soft and nontender, and there was no hepatomegaly or splenomegaly; minimal shifting dullness of the abdomen was present. Peripheral pulses, including pedal pulses, were palpable on both sides. There was bilateral pitting edema of the lower extremities. The right foot was erythematous, swollen, and tender and this inflammation extended to the lower part of the leg. The plantar aspect of the right foot had a purple hue and was also exquisitely tender.

The total leukocyte count was 7,100/µL. A blood smear revealed 4% polymorphonuclear leukocytes, 6% band forms, 82% mature-appearing lymphocytes, 3% myelocytes, and 2% metamyelocytes. The hemoglobin count was 6 mmol/L (9.6 g/dL), and the hematocrit was 29%. The blood glucose level was 16.5 mmol/L (300 mg/dL). A venogram of the right lower extremity did not show any evidence of venous thrombosis. The total serum protein count was 3.5 g/dL, and the albumin was 1.8 g/dL. Serum protein electrophoresis showed markedly reduced gammaglobulin and albumin. Alpha-1 and alpha-2 globulin levels were within normal limits and beta globulins were slightly reduced. Levels of liver enzymes and serum bilirubin were within normal limits.

The patient was admitted with a diagnosis of right foot cellulitis, and therapy was begun with intravenous antibiotics: ceftazidime, 1 g every 12 hours; tobramycin, 60 mg every eight hours (after a loading dose of 100 mg); and metronidazole, 400 mg every six hours. Surgical exploration of an area of skin discoloration over the medial aspect of the right mid-arch revealed a small cavity filled with thin fluid; cultures were obtained from this area. No pus or evidence of fasciitis or pyomyositis was found. On the third hospital day, preliminary results of blood cultures showed gram-positive cocci. Ceftazidime was changed to cefazolin, 1 g intravenously every eight hours. Tobramycin and metronidazole were continued. On the fourth hospital day, the gram-positive cocci in both blood cultures were identified as *S pneumoniae*. Cefazolin was discontinued. Penicillin G was begun intravenously in doses of 4 million units every four hours and gamma globulin, 24 grams intravenously, was administered. Cultures from the foot also grew *S pneumoniae* which was found to be sensitive to Pencillin G, as was the *S pneumoniae* isolated from the blood. However, in addition to penicillin G, tobramycin, and metronidazole were also continued, in view of the continuing neutropenia and to avoid the possibility of superinfection on discontinutation of these antibiotics. On the fifth hospital day, the patient's temperature returned to normal.

During the next ten days the patient had a downhill course. Although he remained afebrile, his right foot became progressively more discolored. The left foot and lower leg also began to exhibit signs of inflammation. Pedal pulses continued to be detectable by Doppler ultrasound. Roentgenograms and computed tomography (CT) scans of the lower extremities failed to show any fluid or gas. Septic emboli were considered. Ultrasound examination of the abdominal aorta and the common femoral arteries was performed to look for an aneurysm as a source of septic emboli; none was found. Echocardiography did not show any valvular vegetations. Blood cultures were repeated on the fourth hospital day and were sterile. Serial chest films did not show any pneumonia. Gangrene developed in the right foot and progressed to involve the distal third of the right leg. Amputation was not allowed by the family, and the patient died on the 15th hospital day.

DISCUSSION

Pneumococcal cellulitis is a rare entity. Of the previously reported cases, four were associated with intravenous drug abuse,[4,5] two with nephrotic syndrome,[6,7] one with rheumatoid arthritis,[8] and one with systemic lupus erythematosus.[5] For another three patients with pneumococcal cellulitis, no associated medical condition or predisposing factor was detected.[9–11] Only one instance of pneumococcal cellulitis was associated with an abnormality of immunoglobulin, namely, macroglobulinemia

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caused by a plasma cell dyscrasia.[12] *S pneumoniae* was cultured from the blood in nearly all these patients; however, it was isolated from the skin in only eight.

The increased susceptibility to pneumococcus of individuals with hypogammaglobulinemia or dysgammaglobulinemia is well known. This increased susceptibility results from impaired antibody production.[2] Normally, anticapsular antibody is responsible for agglutination, immobilization, opsonization, and immune phagocytosis of pneumococcus.[13] In the absence of such antibody, subclinical infections can become clinical, and clinical infections can acquire a life-threatening potential. In one retrospective study,[2] patients with multiple myeloma had a 15 times higher incidence of infections, and those with CLL had a five times higher incidence of infections compared with a control population with atherosclerotic heart disease. In addition, the pneumococcus was the most frequent pathogen in patients with multiple myeloma and CLL.

The rarity of pneumococcal skin infection could either be attributed to low pathogenicity of pneumococcus for skin, or under-diagnosis of such infection. There are several possible causes of under-diagnosis. First, positive cultures, especially soft tissue cultures in the absence of pus, may be difficult to obtain. *S pneumoniae* is an autolytic organism that may be difficult to grow, especially if there is a delay in plating. In addition, pneumococcal cellulitis would, generally, respond to the same treatment as that for streptococcal or staphylococcal cellulitis, thus causing additional difficulty in the diagnosis of pneumococcal cellulitis, if cultures are inadequate or not done.

The severity of infection in this case is worthy of note. Both legs were involved in a cellulitic process, one more severely than the other. Although appropriate antibiotic treatment controlled fever and eradicated pneumococcal bacteremia, cellulitis of the legs continued to progress. The only other reported case of pneumococcal cellulitis associated with dysgammaglobulinemia also resulted in a fatal outcome in spite of appropriate antibiotic treatment. Although unsubstantiated, this could indicate that the development of adequate anticapsular antibody is more important in control of such infections than antibiotic therapy. Also of note is that the skin findings in this case mimicked fasciitis. In reviewing the literature, we found that such a differential diagnostic problem arose in at least one other case of pneumococcal cellulitis and was ruled out at autopsy.[4]

In summary, from the information that is available, pneumococcal cellulitis can be a severe infection but difficult to diagnose at times; may mimic fasciitis; and may be progressive in spite of correct antibiotic therapy, especially in patients with compromised humoral immunity. As the option of prophylaxis with pneumococcal vaccine is unavailable to immunocompromised patients,[14] a high index of suspicion and prompt treatment may be the only hope of saving patients with pneumococcal cellulitis. The role of gamma globulin therapy in the treatment of pneumococcal cellulitis is undefined and cannot be routinely recommended.

REFERENCES

1. Austrian R, Gold J: Pneumococcal bacteremia with especial reference to bacteremic pneumococcal pneumonia. *Ann Int Med* 1964; 60:759–776.
2. Twomey JJ: Infections complicating multiple myeloma and chronic lymphocytic leukemia. *Arch Intern Med* 1973; 132:562–565.
3. Burman LA, Norrby R, Trollfors B: Invasive pneumococcal infection: incidence, predisposing factors, and prognosis. *Rev Infect Dis* 1985; 7: 132–142.
4. Lewis RJ, Alma SR, McGrory JP: *Diplococcus pneumoniae* cellulitis in drug addicts. *JAMA* 1975; 232:54–55.
5. DiNubile MJ, Albornoz MA, Stumacher RJ, et al: Pneumococcal soft-tissue infections: Possible association with connective tissue diseases. *J Infec Dis* 1991; 163:897–900.
6. Mujais S, Uwaydah M: Pneumococcal cellulitis. *Infection* 1983; 11:173–174.
7. Varghese R, Melo JC, Chun C, et al: Erysipelas-like syndrome caused by *Streptococcus pneumoniae. South Med J* 1979; 72:757–758.
8. Peters NS, Eykyn SJ, Rudd AG: Pneumococcal cellulitis: A rare manifestation of pneumococcaemia in adults. *J Infect* 1989; 19:57–59.
9. Connio A: Erisipela diplococcica con setticemia. *Riforma Med* 1917; 33:709–712.
10. McGavin CR, Clancy LJ: Cellulitis in complicated pneumococcal pneumonia. *Br J Dis Ches* 1977; 71:213–214.
11. Hinnen RM, Trachtenberg DE, Miller MA, et al: *Streptococcus pneumoniae* cellulitis. *IMJ* 1986; 170:84–86.
12. Milstein P, Gleckman R: Pneumococcal erysipelas. *Am J Med* 1975; 59:293–296.
13. Davis BD, Dulbecco R, Eisen HN, et al (eds): *Microbiology*. New York, Evanston, London, Harper & Row 1973; p 651.
14. Shapiro ED, Clemens JD: A controlled evaluation of the protective efficacy of pneumococcal vaccine for patients at high risk of serious pneumococcal infections. *Ann Int Med* 1984; 101: 325–330.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Impaction of an ingested table fork in a patient with a surgically restricted stomach

TO THE EDITOR: Eighty percent of ingested foreign bodies occur in the pediatric age group.[1] Intentional ingestion of foreign bodies by an adult is typically associated with prison populations or psychiatric patients. Most ingested foreign bodies in the adult, however, are the result of an accident and involve edentulous patients. Dentures are thought to be frequently responsible for these mishaps, since they impair the ability of the palate to recognize small objects within the food bolus. The foreign bodies ingested are therefore often of small dimensions.

The case reported here is singular for two reasons: the size of the object accidentally ingested and its impaction within a gastric pouch created surgically to treat the patient's morbid obesity.

Case Report. The patient, a 43-year-old male, presented to the Emergency Department stating that he had accidentally swallowed a table fork while attempting to induce vomiting about one hour earlier. His past medical history was significant for gout and morbid obesity, for which he had undergone transverse gastric stapling with side-to-side gastrojejunostomy 11 months earlier. On physical examination the patient was in no distress, vital signs were stable, and the abdomen was soft to palpation. The results of routine laboratory tests were within normal limits. An abdominal film confirmed the presence of a table fork at the gastroesophageal junction with its prongs pointing craniad (Fig 1).

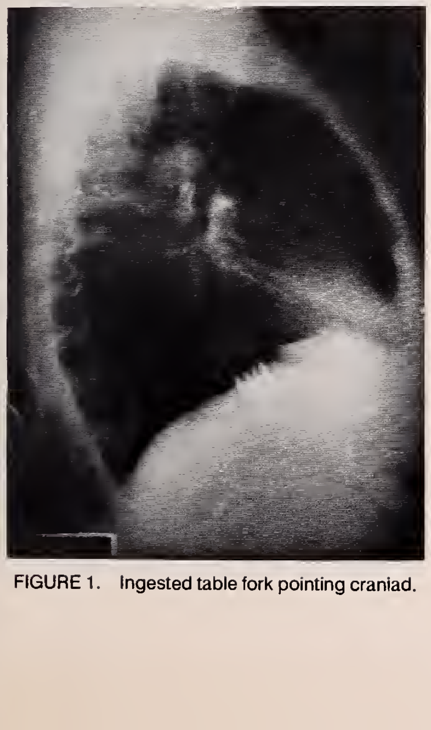

FIGURE 1. Ingested table fork pointing craniad.

Extraction by flexible endoscopy was immediately attempted by an experienced gastroenterologist. The fork was found to be lodged with the handle in the gastric pouch and the prongs at the cardia. The flexible snare did not provide an adequate grip on the tapered prongs and the fork could not be turned around in the stomach because of the previous gastric restrictive surgery. The patient was therefore taken to the operating room for a laparotomy. The gastric pouch was found to be packed with thick, pasty food residue that had prompted the emesis-inducing maneuver.

The fork, measuring approximately 18 cm in length, was extracted and the food particles evacuated through a small transverse anterior gastrotomy, which was then closed in two layers. The postoperative course was unremarkable, and the patient was discharged on the sixth postoperative day.

Discussion Foreign bodies ingested accidentally are typically of small dimensions. A search of the literature disclosed only one other case of a table fork swallowed unintentionally, which occurred under very similar circumstances.[2]

Whereas the great majority of ingested foreign bodies can be easily removed by flexible endoscopy, the management of sharp and pointed objects is more difficult. Since trailing points, in opposition to advancing points, cannot cause puncture, it is recommended that the object be turned around in the stomach.[3] This maneuver was not feasible in our patient because of the surgically created gastric restriction. Moreover, the craniad pointing prongs could easily have punctured the esophageal wall and their tapered ends did not offer an adequate grasp. The endoscopic removal of sharp or pointed ingested foreign bodies can be associated with significant morbidity, and in selected cases surgery is the treatment of choice.

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2. Katsas AG: Ingested table fork. *Arch Surg* 1968; 96:929–930.
3. Webb WA: Management of foreign bodies of the upper gastrointestinal tract. *Gastroenterology* 1988; 94:204–216.

Joseph Isaac Bluestone

TO THE EDITOR: I have a friend who is a physician, and every so often he gives me back issues of various medical journals he has read. Thus, I was privileged to read Dr Naomi Bluestone's wonderful article about Dr Joseph Bluestone in the June issue of the *Journal.*[1]

She is to be congratulated for having written such a beautiful article! The warmth and feeling she brings to the story of this fine and caring physician shine through the entire article. In addition, it is a piece of first-rate historical writing.

I am a historian also—a dental historian—and thus very interested in medical history. I authored a well-received book a few years ago, *Dentistry: An Illustrated History,*[2] and have also had about 80 papers published in various professional journals, both medical and dental.

I would be very interested in receiving any reprints of Dr Bluestone's previous papers since I am certain that they would be of interest.

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1. Bluestone NR: The memoir of Joseph Isaac Bluestone, MD. *NY State J Med* 1991; 91:254–261.
2. Ring ME: *Dentistry: An Illustrated History.* New York, Harry N. Abrams, 1985.

Project diabetes 2000

TO THE EDITOR: The American Academy of Ophthalmology has requested that health care professionals, institutions, and agencies across the nation lend a hand to eliminate preventable blindness from diabetic retinopathy by the year 2000. The project is entitled *Diabetes 2000.* The initial phase targets all health care professionals treating diabetics with the important message that the current rate of vision loss from diabetic retinopathy could be reduced 90% through preventative care. A Wisconsin study indicated that 55% of diagnosed diabetics who met treatment criteria recommended by the *Diabetic Retinopathy Study* did not receive it.[1] Preventive care means at least one dilated retinal examination each year, additional attention to the eyes by primary care physicians augmenting the retinal examinations, and closer collaboration between ophthalmologists and primary care physicians enforcing metabolic control issues. The second phase is public education, trying to reach the 12 million Americans who have diabetes, 6 million of whom are not yet diagnosed, as well as telling the general public of the seriousness of diabetes as a health issue.

State ophthalmological societies have been asked to organize programs enlisting agencies, such as the American Diabetes Association and the Lions Clubs, to communicate this message. Plans are currently being made for the New York State Ophthalmological Society to provide a speakers bureau for the American Diabetes Association Downstate Affiliate Club network as a means of reaching the public. In addition, ophthalmologists will participate in professional education symposia, translating research findings into clinical practice to achieve the goal of elimination of preventable blindness. The current economic climate mandates a grass roots approach, using existing programs for professional and patient education. A pilot hospital teaching day is being developed in Brooklyn, New York, which would provide simultaneous community and professional education. Similar interaction with the Upstate American Diabetes Association Affiliate is planned as well. The American Academy of Ophthalmology has prepared educational materials for patients and professionals, and their customer service number is (415) 561-8540.

Cotton recently summarized some of the cost savings issues.[2]

> In all, disability and rehabilitation for visually impaired diabetics cost $75 billion each year, according to the National Diabetics Data Group. Even if a year of sight costs only $2500, preventing vision loss is cost saving even if every penny of diagnosis and treatment cost is at federal expense. . . .Scheduling examinations less than every six months gets very expensive, with an aggregate of $37 million lost if one year passes between development of disease and treatment, and $80 million lost if two years pass. . . .We're talking about annual savings, even with suboptimal implementation, of over $200 million.

We are faced with an important humanitarian opportunity and a goal which is realistic. The difficult tasks of developing treatment modalities and following up with clinical trials have been accomplished. It is the earier task of translating this information into clinical practice, forging partnerships with professionals, agencies, institutions, and patients, which remains. If we cooperate, diabetes will no longer be the leading cause of blindness among working-age adults by the year 2000, a hopeful message for 12 million people. Kindly pass the word.

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1. Cotton, P: Advances in diabetic retinopathy could save sight, money with more frequent eye exams. *JAMA* 1990; 264:2608,2611.
2. Klein R, Klein BEK, Moss SE, et al: The Wisconsin epidemiologic study of diabetic retinopathy, VI. Retinal photocoagulation. *Ophthalmology* 1987; 94:747–753.

The human tail

TO THE EDITOR: I was rather surprised to read in the article, "The human tail," that only 24 cases had been reported in the literature in the past century.[1]

In 1935, I was engaged in rural practice in the Delaware Valley, centered at Cochecton NY, and adjacent Pennsylvania. I was summoned to deliver a farm girl already in labor. She had not engaged a doctor during pregnancy, and help was limited in the farm house. As I prepped my new patient, I noticed that she had an external tail, possibly 2 cm in length. The infant presented as a frank breech, and to my surprise, another human tail came into view. I remarked, "Two Generations." The newborn's grandmother was by my side, and she quickly said, "That's nothing. I have one, too."

Since the family was not interested in amputations, I dismissed the subject. I believe that many cases are unreported.

My 1935 records have been destroyed, but I vividly recall the experience.

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In reply. We were pleased to read Dr Standfast's letter concerning three generations of a family with a tail. Indeed, Dubrow[1] states authoritatively that there are only 24 cases reported since 1884. However, it is well known that many cases of medical curiosities are not reported in the literature. Had Dr Standfast reported his three cases, the number would now be 27.

Interestingly, finding a tail in three generations of females raised the strong possibility of a gene mutation expressing in a dominant fashion (autosomal or X-linked) causing the malformation. McKusick's *Mendelian Inheritance in Man*[2] does not make mention of a single gene transmission of a human tail (or caudal appendage), neither does the *Birth Defects Encyclopedia*[3] nor *Smith's Recognizable Patterns of Human Malformation*[4] discuss this defect indicating the rarity of a human tail.

We would like to add that since our article was written, three more cases were reported by Belzberg.[5]

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Westbury, NY 11590

1. Dubrow TAJ, Wackym PA, Lesavoy MA: Detailing the human tail. *Annals Plastic Surg* 1988; 20:340–344.
2. McKusick VA: *Mendelian Inheritance in Man.* 9th ed. Baltimore, Md, The Johns Hopkins University Press, 1990.
3. Boyse ML: *Birth Defects Encyclopedia*. London, England, Birth Defects Information Services, Inc, Blackwell Scientific Publications, 1990.
4. Jones KL: *Smith's Recognizable Patterns of Human Malformation*. 4th Ed. Philadelphia, WB Saunders Company, 1988.
5. Belzberg AJ, Muyles ST, Trevensen CL: The human tail and spinal dysraphism. *J Ped Surg* 1991 26:1243–1245.

The ultimate care givers

TO THE EDITOR: Physicians are identified as the care givers, but who takes care of them? I was reminded of that recently on attending the funeral of the wife of a colleague. Time after time, a minister, priest, or rabbi remind us of the selfless devotion of the wife-mother to their families. As physicians, we care for the world. To them, we give the best that we have to offer. What time and energy that is left to go to our families, friends, and finally to our wives. Ironically, it is often our wives who keep things going. Those who give the most get the least. They continually make excuses for us. Their favorite litany begins with, "You have to understand he is a doctor and his time is not his own." Children, relatives, and friends become accustomed to sitting down with the "doctor who is not there." As if that is not enough, they have to become "the doctor's personal therapist" as he unloads frustrations. One marvels at the men who take on this supportive role for women physicians. On top of everything else, these physicians have their unique problems.

These tenured generalists, these awesome jugglers, these unsung greats deserve our eternal thanks. Without their guidance and sensitive intervention, we would need a battery of specialists to keep us going.

With seamless ease, these keeper of the keys guard the gates, preserve the heritage, and burnish the image. When an unkind fate takes them from us, we are bereft and rudderless. They are the quintessential partners in a confusing and turbulent world. We raise an imaginary glass, in silent salute, to the ultimate care givers.

The term "spouse" once connoted the wife of a physician. This is no longer accurate. There has been a dramatic increase in the number of women physicians. Sensitive, supportive husbands have helped them to succeed. One can recall a recent president of a county medical society acknowledging the major role played by her husband. In addition to his professional career, he had often assumed both parental roles to allow her to devote more time to her practice and to organize medicine.

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Reprinted from the July/August 1991 issue of Epidemiology Notes (*Vol 6, No 7.*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Alcohol and breast cancer

Some epidemiologic studies have suggested that alcohol consumption may play a role in the development of breast cancer in women. Although results have not been completely consistent, the consensus is that an association of increased risk with alcohol consumption does exist. The Bureau of Cancer Epidemiology has completed a study of alcohol consumption and breast cancer risk among New York State residents. The complete results are reported in the *International Journal of Epidemiology.*[1] This article highlights some of the important findings from that analysis.

The study was conducted with 1,617 patients diagnosed with primary breast cancer between 1982 and 1984 in 18 New York State counties. A total of 2,050 primary breast cancer cases were eligible for the study. Physician clearance to conduct an interview was obtained for 1,786 (87%) eligible patients. Interviews were obtained for 91% of those cleared by physicians, for an overall response rate of 79%. The cooperation of physicians was instrumental in achieving an adequate response rate for this study.

Controls were selected from the drivers' license files of the New York State Department of Motor Vehicles. Controls were category matched on sex, age, and county of residence. Cases and controls were also limited to women who had listed telephone numbers, since the interviewing was to be conducted by telephone. Interviews were conducted with 1,617 controls, which represented 72% of those eligible. A standardized abstract form was used to collect sociodemographic, pathological, and clinical information from the hospital record of each patient. Epidemiologic data were collected with a structured telephone interview with each breast cancer patient and control. Several questions on alcohol consumption were included. Women were asked if they ever drank alcoholic beverages, the age they started and stopped drinking alcohol, and the amount and frequency of their usual beer, wine, and liquor consumption. Aside from alcohol, information was collected on a number of known and suspected breast cancer risk factors.

Higher breast cancer risks were observed for white women who had their first live birth at a later age, a late age at menopause, a history of benign breast disease, or first-degree relatives with breast cancer. Women who had undergone a bilateral oophorectomy were at reduced risk. Breast cancer risk increased as daily consumption of alcohol increased. Women who consumed 15 or more g of alcohol per day had a risk 1.37 times (95% CI = 1.07, 1.75) that of nondrinkers. Risk did not appear related to the total number of years a woman drank nor restricted to specific types of alcoholic beverages. Women who began drinking after the age of 21 tended to show higher risk of breast cancer at various levels of alcohol consumption. Other risk factors did not confound or modify the effect of alcohol.

Longnecker recently conducted a meta-analysis of the case-control and cohort studies of breast cancer and alcohol consumption published between 1966 and 1987.[2] Relative risk estimates for various levels of alcohol consumption in the meta-analysis were consistent with those found in the New York study. The study in New York failed to show increased breast cancer risk in women who began drinking at an early age. Increased risk was concentrated in women who began drinking later in life, suggesting that alcohol may act as a cancer promoter rather than as an initiator. Although the biologic mechanism for an alcohol effect is not known, animal studies have suggested that alcohol may act through lactation. Additional studies focusing on the patterns of alcohol consumption at different ages and exploring high-risk subgroups are needed.

REFERENCES

1. Nasca PC, Baptiste MS, Field NA, et al: An epidemiologic case-control study of breast cancer and alcohol consumption. *Int J Epidemiol* 1991; 19(3): 532–538.
2. Longnecker MP, Berlin JA, Ozra MJ, et al. A meta-analysis of alcohol consumption in relation to risk of breast cancer. *JAMA* 1988; 260:652–6.

BOOK REVIEWS

HOUSING RISKS AND HOMELESSNESS AMONG THE URBAN ELDERLY

Edited by Sharon M. Keigher. 156 pp. Binghamton, NY, Haworth Press, 1991. $24.95 (hardcover)

Libraries of information exist about homelessness and about aging, but interest in the combined problems of older persons is rare. This is perhaps to be expected, recognizing that homelessness and aging are two complex subjects difficult to define and frustrating to resolve. Therefore, any effort put forth to understand and meet the needs of older homeless persons is valuable and appreciated.

This slim volume documents important information about these individuals. Sharon Keigher and colleagues concentrate their analysis on housing problems associated with homelessness and near homelessness among very-low-income aged persons in Chicago. Their work, however, is easily understood as relevant to the general urban situation of the United States. Recent federal housing policy changes require Keigher's articulate interpretation. Among the many subjects covered are an analysis of the income and housing problems of the elderly; the eroding federal committment to affordable housing; an overview of single-room-occupancy (SRO) populations, policies, past history, and economic trends in the 1980s; the effects of shrinking housing resources on older communities, focused on Chicago; and racial differences regarding shelter and housing in Detroit. As Keigher points out:

> Homelessness among the elderly is a very dynamic state, typically short-term and not repeated, but not always so, and disturbingly prevalent among certain vulnerable older populations in certain neighborhoods having certain endogenous and exogenous resources. Seeing homelessness and near-homelessness as a continuum of conditions rather than as a category allows us to focus not only on its immediate causes, but also on the range of precarious situations that potentially lead to loss of a home and/or to institutionalization.

Forces that propel impoverished older persons into homelessness are wide-ranging and, as the author reveals, difficult to analyze comprehensively, but they include the growing shortage of low-income housing. In many cities, SRO housing stock, the last acceptable protected environment for the poor before they are forced to hit the streets, has almost disappeared because of gentrification.

The structure at 160 Bleecker Street in the Greenwich Village areas of Manhattan is a worthy example. This eight-story stone building was created as housing for working men in the 1890s, and called the Mills Hotel No 1. By the Depression, its residents were the poor and jobless. In the 1970s, by now called the Greenwich Hotel, the building underwent an internal reconstruction and became a site where 1,200 homeless men were placed in cubicles; and at last, as the value of the property grew, it has been transmuted into The Atrium, a high-cost condo.

Keigher's book has as a prime goal understanding how housing-related problems and substandard residences for the elderly lead to preventable death. This point is relevant to the lives of homeless persons in our country. In the United States, about 13% of the population is age 65 years and older. According to analyses prepared by demographer James Wright of Tulane University (*Homelessness and Health*, 1987), using information obtained from homeless health care programs across the country, about 4% of the homeless are in this age group. What happened? Where are the older homeless men and women? By age 65, some have tapped into social security and/or SSI benefits, have the funds to obtain housing on their own and thus are no longer homeless. Far more, however, have died early, victims of the homeless lifestyle. Professor Wright concludes that an average of 10–20 years in lifespan are lost by the chronically homeless. The homeless are old by age 45.

The essence of this book is contained in the chapter "Coping With Threats to and Loss of 'Home'." The focus here is on individual desires, older, homeless persons. Stereotyping is minimized and the urgent of these men and women as struggling viable human beings are brought forth. The pressing need of each for independence and to avoid the constraints of institutionalization is clear. The case histories reveal how a person establishes "a sense of 'home' on the streets" to avoid placement in a shelter or nursing home. Is this a misguided desire? Or are we imposing our values on others by trying to force the homeless into placement against their wills? Keigher describes a lifestyle of "hunting and gathering" on the streets, a reference to nomadic early human tribes that is apt and evocative when brought to the level of these individuals in the 1990s. The authors tell us about Dana, a woman now aged 62, who has been on the streets for 11 years. A map of her world is printed. It shows the 30 square blocks in which she lives her restricted life. They include a store entry where she sleeps, a thrift shop, a diner, a currency exchange, a meal program, the local social service office and a private social service agency, a bench at the bus turnaround and the police station. She copes.

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THE PURIFICATION PRESCRIPTION

By Sheldon Saul Hendler, MD, PhD. 300 pp. New York, William Morrow & Co, 1991. $22.95 (hardcover)

This book purports to prescribe five-week programs that are supposed to rid the body and brain of a broad spectrum of harmful chemicals. Part I, "The Purification Promise," describes the benefits of restoring a biochemical balance, imparting a feeling of wholeness. Part II, "The Elements of the Prescription," describes selected vitamins, minerals, lipids, herbs, pharmaceuticals, diet, light, exercise, relaxation, and their usefulness in cleansing the body and easing withdrawal from addictions. Part III, "The Purification Prescription," outlines the actual regimen for body detoxification from both environmental pollutants and food and drug addictions.

This book, like so many others in this area, attempts to offer easy solutions to multiple and complicated problems. It is true that for too many of us ". . . our bodies are being tainted not only by . . . (drugs) . . . but also by an ever-expanding spectrum of . . . (environmental) pollutants. . . ." However, the costs to public health are undeniably enormous.

Yet, there are problems of oversimplification in this book. The elements of the prescription (natural vitamins, minerals, amino acids), although necessary in trace amounts and sufficiently present in a balanced diet, have not been consistently shown to be of benefit in megadoses. Some have recently been found to be deadly in supplemental doses. The fish oils, the lecithins, and other natural additives may be beneficial, or may have side effects as yet unknown, but certainly have not been proven sufficiently to offer as a prescription.

Pharmaceuticals are also offered as possible prescriptions. There appears to be special emphasis on the antidepressant, fluoxetine, which may be useful ". . . in food, alcohol, and other obsessive-compulsive disorders. . . ." The book describes the well-known benefits and importance of a good high fiber, low fat diet, regular exercise, relaxation, and stress management.

The actual prescriptions present case studies as validation of the effectiveness of body detoxification from environmental pollutants and drugs (alcohol, nicotine, amphetamines, cocaine, barbiturates, opiods). Although some short discussion is given to the common sense strategies of healthy lifestyles, these prescriptions for drug detoxification rely heavily on supportive drugs and must be physician supervised. In addition, specific vitamins, minerals, and herbs, as suggested in supplemental doses "... *may* be of benefit...."

The book may be useful as an adjunct to supervised treatment in the understanding of a specific addiction and a specific supervised treatment. However, it offers no new proven prescriptions, no magic, purifying bullets for the lay person. As such, this book has limited usefulness as an addition to the medical literature.

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TEXTBOOK OF PERFORMING ARTS MEDICINE

Edited by Robert Thayer Sataloff, MD; Alice G. Brandfonbrener, MD; Richard J. Lederman, MD. 448 pp, illustrated. New York, Raven Press, 1991. $88.00 (hardcover)

According to the authors, the purpose of this book is to fill a compelling need for information among physicians, performers, teachers, and therapists about a new and exciting field, performing arts medicine. At this time, it is the only book that purports to deal with this topic. Two of the authors, Drs Brandfonbrener and Lederman, have published extensively on the topic of music medicine, and Dr Sataloff is very well known in the field of laryngology and has greatly contributed to our understanding of the problems of the professional voice. In spite of this, the book is generally somewhat of a disappointment, primarily because it lacks consistency, cohesiveness, and direction, leading the reader to ponder what this new specialty is and who should practice it.

The first chapter, about the history of performing arts medicine, emphasises the major progress made in music medicine. The areas of voice, dance, and psychology, where substantial progress has also been made and which have a long, well-documented bibliography, are treated in less detail. The chapter on epidemiology of medical problems of musicians lacks the depth necessary to be useful to the practitioner attempting to treat the ailing instrumentalist. Although there is interesting general information about the individual instruments, there is little specific information about occupational and physical therapy and the role of technique retraining and ergonomics in treatment and prevention. Dr Sataloff's chapter on the care of the professional voice is well written and useful and meets the criteria for a textbook. There are also well developed chapters on neurologic and psychiatric problems. The chapter on hand surgery makes little specific reference to instrumentalists and is essentially what one might find in a synopsis of surgery. It would certainly have been useful to have conveyed some information about which instruments and technique deficits produce specific hand and forearm problems, such as de Quervain's disease or carpal tunnel syndrome in pianists and guitarists. The chapter relating to the foot and ankle problems in the dancer is interesting and concise. However, further development of the role of physical therapy and other treatment and preventive concepts would have been helpful. In this reviewer's opinion, the information conveyed does not give enough detail to allow a physician to proceed with confidence in caring for some of these injuries.

The chapter on the temporomandibular joint syndrome, facial pain, and dental problems is well organized and filled with practical information. The chapter the legal aspects of medical treatment was tantalizingly short, and the subject matter was treated as an afterthought. Indexing is occasionally lacking in consistency. For example, focal dystonia is listed under "F," but dystonia is not listed at all.

To summarize, the book's main advantage is that it represents a first attempt to define a new area of medical interest and brings together some basic information about the field, albeit in a spotty fashion. The $88 price tag puts the book in the category of one that should probably only be purchased by those who think they need it because of a keen and specific interest in caring for performing artists or want a good reference resource.

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AZTEC MEDICINE, HEALTH, AND NUTRITION

By Bernard Ortiz de Montellano. 308 pp, illustrated. New Brunswick, NJ, Rutgers University Press, 1990. $40.00 (hardcover) $15.00 (softcover)

Is *Aztec Medicine, Health, and Nutrition* another collection of folksy medicine, superstitious belief, and herbal mumbo jumbo? What possible relevance could a long-dead medical system have to the modern practice of medicine? Weren't the Aztecs an impoverished and barbaric people, forced to cannibalism to survive, and so weak they were easily overthrown by the Spanish conquistadores?

Bernard R. Ortiz de Montellano, Professor of Anthropology of Wayne State University, in a compelling, rigorous, and myth-dispelling book, tackles these and other questions in his examination of the background and development of medicine as practiced centuries ago in Aztec society.

Ortiz depicts a fascinating story of Aztec culture before and after the Spanish conquest. Hernan Cortes arrived in 1519, with a force that should have been easily overwhelmed by the Aztecs, who vastly outnumbered the small band of conquistadores. Yet, in a short three years, the conquest was complete, and by 1521 the Spanish occupied and controlled Aztec Mesoamerica.

Were the Aztecs an overpopulated, undernourished, weak society, easy prey to the Europeans? Ortiz' analysis leads us to believe not, describing the richness of Aztec agriculture. The Aztec diet boasted a balanced vegetarian menu, including maize, amaranth, spirulina, and cacti. Ortiz further asserts "... the Aztecs ate practically every living thing that walked, swam, flew, or crawled, ..." including armadillos, pocket gophers, weasels, rattlesnakes, mice, iguanas, domesticated turkeys and dogs, fish, frogs, dragonfly larvae, grasshoppers, ants, and worms.

In fact, Ortiz painstakingly shows that there was plenty of food around, enough so the Aztecs did not have to eat each other to be adequately nourished. He supplants his well-substantiated argument against "cannibalism for food," however, with a less fully developed hypothesis that cannibalism served as human sacrifice, an integral part of Aztec religion. The author generally presents the Aztecs as an advanced civilization, and to this reviewer, the "eating people for religion" proposition might be easier to believe if the book provided greater detail of how human sacrifice fit in with Aztec society, family structure, and religious beliefs.

If the Aztecs were so well fed and strong, why did this small group of Spaniards easily defeat them? Ortiz points out that the Spaniards arrived in 1519, a year called "One Reed," a time Aztec myth proclaimed the mighty god Quetzalcoatl was to return from the east. However, from just this direction came Cortes and men. Ortiz believes "... the Aztecs at first considered the Spanish to be gods and were psychologically incapable of fighting them. When the less-than-divine behavior of the conquerors became apparent it was too late...."

Compounding the initial stunning psychological blow of the Spaniards appearing as returning Aztec deities was the presence of a killing disease. The illness, moreover, was not that of the Aztecs, but one imported by the conquerors. They brought with them smallpox, a lethal virus completely unknown to the Aztecs. The virus proceeded to wipe out large segments of the Aztec population, putting the final touches on the Spanish victory.

After the conquest, Aztec and Spanish society met and melded in a process called

Syncretism, a fusion of Mesoamerican and European worldview, religion, and medicine. Ortiz shows that the multiplicity of Catholic saints fitted very well with the Aztec pantheon. Catholic saints were believed to be specialists who cure specific diseases, such as St. Valentine for epilepsy and St. Fiacre for hemorrhoids. Aztec deities were thought to be cause and cure for certain diseases. Violations of the prescribed feast of Atamalqualiztli, for example, resulted in ringworm and skin diseases, and Atlatonan "... both afflicted and cured lepers and people born with physical defects and suffering from sores...."

In addition to religious and magical medical precepts, the Aztecs possessed considerable empiric knowledge of medicine. For example, the Aztecs followed a three step method of wound treatment, closely akin to modern principles: (1) wash the wound with warm, fresh urine (sterile in healthy humans); (2) treat the wound with matlalxihuit (*Commelina pallida*, a vasoconstrictor); and (3) dress the wound with hot, concentrated sap from agave leaves (having detergent and antibiotic properties).

Ortiz gives equally interesting examples of Aztec remedies and their contemporary explanations. The herb *cihuapatli* and the tail of the tlaquatl (opossum) were used to stimulate uterine contractions, both containing oxytoxic properties. Surgery was performed using hair to suture lacerations, and finely honed obsidian blades (thought to parallel today's surgical steel for precision) were used for making incisions. Salted bee honey (with antibiotic properties) was used as prophylaxis against wound infections.

These remedies are explained in a chapter, "Curing Illness," which should be of particular interest to practicing physicians as well as medical historians, since it describes the methods and modern explanations of these centuries old remedies. Indeed, *Aztec Medicine, Health, and Nutrition* is an informative book, stimulating enough to go beyond being a dry exposition of medical history to appeal to a broad medical and lay audience.

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SOCIAL WORK IN HEALTH CARE: A HANDBOOK FOR PRACTICE

Part I and Part II. Edited by Kay W. Davidson, DSW, and Sylvia S. Clarke, MSC. Part I, 517 pages, illustrated, Part II, 935 pp, illustrated. Binghamton, NY, The Haworth Press, 1990. $89.95 (set only) (hardcover) $49.95

This is a two-volume compendium of articles that represents leading, authoritative thinking on major relevant areas of social work practice in health care. It is a source book offering a wide scope of knowledge presented in sections, each introduced by the editors' commentaries. These commentaries capture the critical elements of each topic, providing the educational framework for the ensuing content. Both the articles and the discussions of each section are exciting, challenging, and compelling. Many of the articles are seminal and are considered classics in the field. Each was selected with careful thought and concern for comprehensiveness by Professor Davidson and Mrs Clarke who are esteemed and highly respected educators and editors. *Social Work in Health Care* reflects their assiduous work and professional stature.

The book contains seven sections, including education, practice, and research issues. Any number of articles would have value not only for social workers, but also for their colleagues in the health care field. Several articles relate particularly to medical student education, whereas others discuss psychosocial intervention in health care on behalf of specific populations and contexts. These include work with people with AIDS and patients with Alzheimer's Disease. Special contexts such as the health needs of Hispanic children, children in reverse isolation units, primary health care, trauma centers, and intensive care units is addressed. There is an entire section on interprofessional practices and team behaviors and another on research and evaluation. Innovative programs are described that would appeal to those in leadership and teaching roles in health care. One article "An Expanded Social Work Role in a University Hospital Based Group Practice: Service Provider, Physician Educator" is illustrative of a conceptual framework widely applicable to enhancing medical practice. The commentaries and references after each section offer further illumination.

This book is highly recommended for social workers, physicians, other health care professionals, and administrators. It should be made available in every health care library collection and would also make a welcome gift from a colleague invested in another's broad-based health care education.

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SYNOPSIS OF NEUROLOGY

Edited by Richard Lechtenberg, MD, 448 pp, illustrated. Malvern, Pa, Lea & Febiger, 1991. $19.75 (softcover)

Dr Lechtenberg has written an overview of clinical neurology for the non-neurologist. By organizing the chapters around specific symptoms, he has tried to meet a clinician's needs as he is caring for patients with particular problems.

The first two chapters review the neurological history, examination, and the standard imaging and diagnostic tests. Other chapters deal with the unresponsive patient, syncope, transient neurological signs, cognitive and mood disorders, sexual and urogenital problems, movement disorders, sleep disorders, weakness, sensory abnormalities, headache and facial pain, visual disturbances, vertigo, and related complaints. Four chapters at the end of the book deal with stroke, neoplastic disease, congenital malformations, and neurological complications of common medical problems.

The value of the book for a general clinician may be limited by its necessary brevity. A physician confronted with a patient with some of the diagnoses might be very frustrated by the limited information provided. The amount of information on therapeutic modalities is likewise limited in most cases. Although a bibliography accompanies each chapter, specific topics are not cited for individual references. At times, the author's organizational approach leads to unusual emphasis: eg, muscular dystrophy is listed as the first section of the chapter on weakness when stroke would be the most commonly encountered form of weakness.

Many of the chapters would have benefited from a more detailed description of the approach to the differential diagnoses of the particular symptom. For example, in the chapter on weakness, a more extensive discussion of the onset, distribution, and character of the weakness would have provided the reader with a more substantial understanding of the diagnoses and the management of the patient.

The writing is generally lucid and readable. The illustrations are well-chosen and contribute to the reader's understanding. The charts and tables are well organized, and the index is excellent.

In summary, this text partially meets its author's objective. For the price, it would be a possible choice for physicians or health professionals without extensive experience in neurology. However, one would also want to have available one of the more standard neurology textbooks for more detailed discussions of particular problems.

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NEOPLASMS WITH ECCRINE DIFFERENTIATION

Edited by Pascual Abenoza, MD, and A. Bernard Ackerman, MD, 536 pp, illustrated. Malvern, PA, Lea & Febiger, 1990. $120.00 (hardcover)

Abenoza and Ackerman have tried to provide a method for all specific and accurate diagnosis of skin tumors with eccrine differentiation. Scanning magnification with conventional microscopy is proposed to outline a "silhouette" of the tumor that could be used as a diagnostic technique for histologic classification. The authors carefully review the literature to produce the monograph, but actual data for each tumor classification from their own series are omitted with the exception of percentage graphs for the distribution of histologic neoplasms by age, sex, anatomic site, and/or clinical size at the time of biopsy. The correlation of histologic pattern, anatomic site, and age with actual differentiation of the eccrine neoplasm is also omitted from the discussion.

This broad review is commendable particularly with respect to the clinical problems, arising daily in the dermatopathology laboratory, including the discussion of non-neoplastic alterations of eccrine units. Certain principles are promoted for dermatopathologic diagnoses with documentation of factors influencing the algorithmic method for specific diagnosis of skin disease. For example, assessment of the anatomic site and/or the age of the patient inferred from histologic signs is promoted as an aid to the final diagnosis. However, the authors do not produce any data substantiating this conclusion from their own series of different eccrine tumors and histologic patters but simply report the information available from the clinician. In other words, the series of cases reported with each eccrine tumor are not objectively reviewed or subjected to "repeatable, reliable, and accurate criteria" or "histologic findings indispensable for diagnosis" of eccrine neoplasms. The conclusions apparently represent distillation of current knowledge or subjective analysis of their own material without peer review.

Certainly the authors' qualifications and the material available for review justify the catalog of neoplasms with eccrine differentiation. The monograph, as noted in the foreward, is based on the original contributions of multiple authors from the medical literature. The book will be used to the pathologist because of the excellent photographs and discussions. The contents include an introduction regarding principles of diagnoses used in the text with helpful chapters for understanding the embryology of skin and other histologic features of eccrine units. The proposition for "a least common denominator" required for diagnosis is helpful. However, the "silhouette" of each neoplasm could have been presented more forcefully since frequently the exceptions to the criteria cloud the silhouette. The histologic observations are too generalized for significant image analysis. The clear black-and-white photomicrographs from scanning to higher magnifications for cytologic detail are excellent. Accuracy of the contents is acceptable with the exception of the lesion identified as a regressing keratoacanthoma, on page 67 in figure 3.18, which was selected for demonstration of the acrosyringia. The following 11 photomicrographs abundantly illustrate acrosyringia in different clinical materials.

Hashimoto, Mehregan, and Kumakiri outline approximately 23 eccrine tumors in 64 small pages (Hashimoto K, Mehregan AH, and Kumakiri M: *Tumors of Skin Appendages*. Boston, Butterworths, 1987). These authors believe that morphological variations of eccrine tumors are so great from one variety of tumor to another that uniform criteria for definitive diagnoses were difficult. Histologic criteria at a higher magnification, however, demonstrated that the eccrine tumor cells tend to form lumina, and the clear cells have high glycogen content and a lesser propensity for keratinization. These observations are helpful guides in differentiating eccrine gland tumors from other adnexal neoplasms. These authors specify that ultrastructural observations would facilitate the identification of eccrine tumors but could only specify the portion of the eccrine gland or duct to which a tumor would differentiate. Histochemical and immunohistochemical stains were very helpful in their classification. These authors propose that new antibody studies would be progressively more useful.

The chapter "Neoplasms with Eccrine Differentiation" classifies this method of histologic diagnosis using a method of pattern analysis as an ambitious undertaking and acknowledges ". . . the limitations of the study and the limits of our knowledge." In my opinion the monograph would have been more authoritative if the authors had concentrated on their own material derived from 750,000 biopsy specimens (1979–1987). Objective analysis of their own histologic findings for the diagnosis of eccrine tumors by the proposed criteria would have been more helpful to me. Certainly, their work will stimulate review of previous controversial cases with a different enthusiasm, and therefore their labors were beneficial.

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COLOR ATLAS OF CLINICAL ORAL PATHOLOGY

Edited by Brad W. Neville, DDS, Douglas D. Damm, DDS, Dean K. White, DDS, Charles A. Waldron, DDS. Malvern, Pa, Lea & Febiger, 1991, $89.50 (hardcover)

In the preface, the four authors advise that the text is intended to assist ". . . the clinician in arriving at a differential diagnosis for lesions . . ." The book is divided into broad disease categories and is primarily an atlas. Therefore, text and references are kept to a minimum. The authors are all well known oral pathologists, clinicians, and teachers with fine reputations. The book is composed of excellent magnified color pictures of the close-up variety to accent the entity identified. Thus, only three photographs of X-rays fit on a page in vertical line. The accompanying text appears on the adjacent page.

Since the book is divided into broad disease categories, it is easy to locate and follow a disease entity in its various locations in the oro-facial and neck regions. As a color atlas, it is excellent and compares favorably with other atlases published over the years. The photographs are up-to-date in clarity, and the text is mostly clear in its description of the lesion portrayed in the radiographs. Although the text as a whole is excellent, various sections are ambiguous and provide incomplete information regarding therapy for certain diseases. Examples of this include the following: page nine, the torus palatinus is described as "... enlargement of the midline of the hard palate." The enlargement is not of the midline, although it appears at that juncture. On page 11, the radiograph of Eagle's Syndrome is lacking a marker for the side of the mandible involved.

In addition, an arrow pointing to the lesion would help the viewer considerably. Page 27 identifies a branchial cleft cyst in the neck of a middle-aged women with no mention of other entities located in this region (eg, metastatic disease) to aid in differential diagnosis. The term "branchial cyst" is older terminology. The newer terminology, mentioned in the text, is cervical lymphoepithelial cyst. Periapical abscess is identified on pages 74 and 75 via a radiograph with a radiolucent bony lesion. A periapical abscess is identified through its clinical presentation. Thus, a radiolucent bony lesion without evidence of a clinical picture of an abscess would not be called a "periapical abscess." Lacking from the text is the usual therapy for such an abscess, such as an incision and drainage and/or antibiotics. Under Proliferative Periostitis, on page 90, the text states "chronic dental infection . . . spreads toward the surface of the bone." A more technical term, such as the cortex rather than "surface of the bone" would be preferred. An extra-oral facial cellulitis is identified on page 92 and general therapy proposed consisting of "warm compresses . . .; if the infection localizes, . . . drained." Localization of an extra-oral cellulitis is not desired at times. Better oral surgical input regarding therapy would have prevented errors of this type.

Overall, the book achieves its objectives with one failure. There are almost no offerings of other lesions that could occur in the same locale to assist in differential diagnosis. Each lesion is presented without such comment.

The text is recommended with the limitations observed, as a valuable addition to the clinician's library alongside textbooks or oral pathology.

RAYMOND F. ZAMBITO, DDS, EdD
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Jamaica, NY 11432

Uniform requirements for manuscripts submitted to biomedical journals

International Committee of Medical Journal Editors

In the 12 years since it was first published, the "Uniform Requirements for Manuscripts Submitted to Biomedical Journals" (the Vancouver style) to which the *Journal* adheres, developed by the International Committee of Medical Journal Editors (ICMJE), has been widely accepted by both authors and editors; over 400 journals have stated that they will consider manuscripts that conform to its requirements. This is the fourth edition of the Uniform Requirements.

In January 1978, a group of editors from some major biomedical journals published in English met in Vancouver, British Columbia, and decided on uniform technical requirements for manuscripts to be submitted to their journals. The Vancouver group evolved into the International Committee of Medical Journal Editors. Over the years, the group has revised the requirements slightly; this is the fourth edition.

Over 400 journals have agreed to receive manuscripts prepared in accordance with the requirements. It is important to emphasize what these requirements imply and what they do not.

First, the requirements are instructions to authors on how to prepare manuscripts, not to editors on publication style. (But many journals have drawn on these requirements for elements of their publication styles.)

Second, if authors prepare their manuscripts in the style specified in these requirements, editors of the participating journals will not return manuscripts for changes in these details of style. Even so, manuscripts may be altered by journals to conform with details of their own publication styles.

Third, authors sending manuscripts to a participating journal should not try to prepare them in accordance with the publication style of that journal but should follow the "Uniform Requirements for Manuscripts Submitted to Biomedical Journals."

Nevertheless, authors must also follow the instructions to authors in the journal as to what topics are suitable for that journal and the types of papers that may be submitted—for example, original articles, reviews, or case reports. In addition, the journal's instructions are likely to contain other requirements unique to that journal, such as number of copies of manuscripts, acceptable languages, length of articles, and approved abbreviations.

Participating journals are expected to state in their instructions to authors that their requirements are in accordance with the "Uniform Requirements for Manuscripts Submitted to Biomedical Journals" and to cite a published version.

SUMMARY OF REQUIREMENTS

Type the manuscript double-spaced, including title page, abstract, text, acknowledgments, references, tables, and legends.

Each manuscript component should begin on a new page, in the following sequence: title page, abstract and key words, text, acknowledgments, references, tables (each table complete with title and footnotes on a separate page), and legends for illustrations.

Illustrations must be good-quality, unmounted glossy prints, usually 127 × 173 mm (5 × 7 in), but no larger than 203 × 254 mm (8 × 10 in).

Submit the required number of copies of manuscript and figures (see journal's instructions) in a heavy paper envelope. The submitted manuscript should be accompanied by a covering letter, as described under Submission of Manuscripts, and permissions to reproduce previously published material or to use illustrations that may identify human subjects.

Follow the journal's instructions for transfer of copyright. Authors should keep copies of everything submitted.

PRIOR AND DUPLICATE PUBLICATION

Most journals do not wish to consider for publication a paper on work that has already been reported in a published paper or is described in a paper submitted or accepted for publication elsewhere. This policy does not usually preclude consideration of a paper that has been rejected by another journal or of a complete report that follows publication of a preliminary report, usually in the form of an abstract. Nor does it prevent consideration of a paper that has been presented at a scientific meeting if not published in full in a proceedings or similar publication. Press reports of the meeting will not usually be considered as breaches of this rule, but such reports should not be amplified by additional data or copies of tables and illustrations. When submitting a paper an author should always make a full statement to the editor about all submissions and previous reports that might be regarded as prior or duplicate publication of the same or very similar work. Copies of such material should be included with the submitted paper to help the editor decide how to deal with the matter.

Multiple publication—that is, the publication more than once of the same study, irrespective of whether the wording is the same—is rarely justified. Secondary publication in another language is one possible justification, provided the following conditions are met.

The editors of both journals concerned are fully informed; the editor concerned with secondary publication should have a photocopy, reprint, or manuscript of the primary version.

The priority of the primary publication is respected by a publication interval of at least two weeks.

The paper for secondary publication is written for a different group of readers and is not simply a translated version of the primary paper; an abbreviated version will often be sufficient.

The secondary version reflects faithfully the data and interpretations of the primary version.

A footnote on the title page of the secondary version informs readers, peers, and documenting agencies that the paper was edited, and is being published, for a national audience in parallel with a primary version based on the same data and interpretations. A suitable footnote might read as follows: "This article is based on a study first reported in the [title of journal, with full reference]."

Multiple publication other than as defined above is not acceptable to editors. If authors violate this rule they may expect appropriate editorial action to be taken.

Preliminary release, usually to public media, of scientific information described in a paper that has been accepted but not yet published is a violation of the policies of many journals. In a few cases, and only by arrangement with the editor, preliminary release of data may be acceptable—for example, to warn the public of health hazards.

PREPARATION OF MANUSCRIPT

Type the manuscript on white bond paper, 216 × 279 mm (8 × 11 in) or ISO A4 (212 × 297 mm), with margins of at least 25 mm (1 in). Type only on one side of the paper. Use double-spacing throughout, including title page, abstract, text, acknowledgments, references, tables, and legends for illustrations. Begin each of the following sections on separate pages: title page, abstract and key words, text, acknowledgments, references, individual tables, and legends. Number pages consecutively, beginning with the title page. Type the page number in the upper or lower right-hand corner of each page.

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The title page should carry (a) the title of the article, which should be concise but informative; (b) first name, middle initial, and last name of each author, with highest academic degree(s) and institutional affiliation; (c) name of department(s) and institution(s) to which the work should be attributed; (d) disclaimers, if any; (e) name and address of author responsible for correspondence about the manuscript; (f) name and address of author to whom requests for reprints should be addressed or statement that reprints will not be available from the author; (g) source(s) of support in the form of grants, equipment, drugs, or all of these; and (h) a short running head or foot line of no more than 40 characters (count letters and spaces) placed at the foot of the title page and identified.

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All persons designated as authors should qualify for authorship. The order of authorship should be a joint decision of the coauthors. Each author should have participated sufficiently in the work to take public responsibility for the content.

Authorship credit should be based only on substantial contributions to (a) conception and design, or analysis and interpretation of data; and to (b) drafting the article or revising it critically for important intellectual content; and on (c) final approval of the version to be published. Conditions (a), (b), and (c) must all be met. Participation solely in the acquisition of funding or the collection of data does not justify authorship. General supervision of the research group is also not sufficient for authorship. Any part of an article critical to its main conclusions must be the responsibility of at least one author.

A paper with corporate (collective) authorship must specify the key persons responsible for the article; others contributing to the work should be recognized separately (see Acknowledgments).

Editors may require authors to justify the assignment of authorship.

Abstract

The second page should carry an abstract (of no more than 150 words for unstructured abstracts or 250 words for structured abstracts). The abstract should state the purposes of the study or investigation, basic procedures (selection of study subjects or laboratory animals, observational and analytical methods), main findings (give specific data and their statistical significance, if possible), and the principal conclusions. Emphasize new and important aspects of the study or observations.

Text

The text of observational and experimental articles is usually—but not necessarily—divided into sections with the headings Introduction, Methods, Results, and Discussion. Long articles may need subheadings within some sections to clarify their content, especially the Results and Discussion sections. Other types of articles such as case reports, reviews, and editorials are likely to need other formats. Authors should consult individual journals for further guidance.

Introduction

State the purpose of the article. Summarize the rationale for the study or observation. Give only strictly pertinent references, and do not review the subject extensively. Do not include data or conclusions from the work being reported.

Methods

Describe your selection of the observational or experimental subjects (patients or laboratory animals, including controls) clearly. Identify the methods, apparatus (manufacturer's name and address in parentheses), and procedures in sufficient detail to allow other workers to reproduce the results. Give references to established methods, including statistical methods (see below); provide references and brief descriptions for methods that have been published but are not well known; describe new or substantially modified methods, give reasons for using them, and evaluate their limitations. Identify precisely all drugs and chemicals used, including generic name(s), dose(s), and route(s) of administration.

Ethics

When reporting experiments on human subjects indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) or with the Helsinki Declaration of 1975, as revised in 1983. Do not use patients' names, initials, or hospital numbers, especially in any illustrative material. When reporting experiments on animals indicate whether the institution's or the National Research Council's guide for, or any national law on, the care and use of laboratory animals was followed.

Statistics

Describe statistical methods with enough detail to enable a knowledgeable reader with access to the original data to verify the reported results. When possible, quantify findings and present them with appropriate indicators of measurement error or uncertainty (such as confidence intervals). Avoid sole reliance on statistical hypothesis testing, such as the use of p values, which fails to convey important quantitative information. Discuss eligibility of experimental subjects. Give details about randomization. Describe the methods for and success of any blinding of observations. Report treatment complications. Give numbers of observations. Report losses to observation (such as dropouts from a clinical trial). References for study design and statistical methods should be to standard works (with pages stated) when possible rather than to papers in which the designs or methods were originally reported. Specify any general-use computer programs used.

Put general descriptions of methods in the Methods section. When data are summarized in the Results section specify the statistical methods used to analyze them. Restrict tables and figures to those needed to explain the argument of the paper and to assess its support. Use graphs as an alternative to tables with many entries; do not duplicate data in graphs and tables. Avoid nontechnical uses of technical terms in statistics, such as "random" (which implies a randomizing device), "normal," "significant," "correlations," and "sample." Define statistical terms, abbreviations, and most symbols.

Results

Present your results in logical sequence in the text, tables, and illustrations. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations.

Discussion

Emphasize the new and important aspects of the study and the conclusions that follow from them. Do not repeat in detail data or other material given in the Introduction or the Results section. Include in the Discussion section the implications of the findings and their limitations, including implications for future research. Relate the observations to other relevant studies. Link the conclusions with the goals of the study but avoid unqualified statements and conclusions not completely supported by your data. Avoid claiming priority and alluding to work that has not been completed. State new hypotheses when warranted, but clearly label them as such. Recommendations, when appropriate, may be included.

Acknowledgments

At an appropriate place in the article (title-page footnote or appendix to the text; see the journal's requirement) one or more statements should specify (a) contributions that need acknowledging but do not justify authorship, such as general support by a departmental chairman; (b) acknowledgments of technical help; (c) acknowledgments of financial and material support, specifying the nature of the support; (d) financial relationships that may pose a conflict of interest.

Persons who have contributed intellectually to the paper but whose contributions do not justify authorship may be named and their function or contribution described—for example, "scientific adviser," "critical review of study proposal," "data collection," or "participation in clinical trial." Such persons must have given their permission to be named. Authors are responsible for obtaining written permission from persons acknowledged by name, because readers may infer their endorsement of the data and conclusions.

Technical help should be acknowledged in a paragraph separate from those acknowledging other contributions.

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Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables, and legends by Arabic numerals in parentheses. References cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text of the particular table or illustration.

Use the style of the examples below, which are based with slight modifications on the formats used by the US National Library of Medicine in Index Medicus. The titles of journals should be abbreviated according to the style used in Index Medicus. Consult List of Journals Indexed in Index Medicus, published annually as a separate publication by the library and as a list in the January issue of Index Medicus.

Try to avoid using abstracts as references; "unpublished observations" and "personal communications" may not be used as references, although references to written, not oral, communications may be inserted (in parentheses) in the text. Include among the references papers accepted but not yet published; designate the journal and add "In press." Information from manuscripts submitted but not yet accepted should be cited in the text as "unpublished observations" (in parentheses).

The references must be verified by the author(s) against the original documents.

Examples of correct forms of references are given below.

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(1) Standard journal article (List all authors, but if the number exceeds three give three followed by et al.)

You CH, Lee KY, Chey RY, et al: Electrogastrographic study of patients with unexplained nausea, bloating, and vomiting. *Gastroenterology* 1980 Aug;79(2):311–4.

As an option, if a journal carries continuous pagination throughout a volume, the month and issue number may be omitted.

You CH, Lee KY, Chey RY, et al: Electrogastrographic study of patients with unexplained nausea, bloating and vomiting. *Gastroenterology* 1980;79:311–4.

Goate AM, Haynes AR, Owen MJ, et al: Predisposing locus for Alzheimer's disease on chromosome 21. *Lancet* 1989;1:352–5.

(2) Organization as author

The Royal Marsden Hospital Bone-Marrow Transplantation Team. Failure of syngeneic bone-marrow graft without preconditioning in post-hepatitis marrow aplasia. *Lancet* 1977;2:742–4.

(3) No author given

Coffee drinking and cancer of the pancreas [editorial]. *BMJ* 1981;283: 628.

(4) Article in a foreign language

Massone L, Borghi S, Pestarino A, et al: Localisations palmaires purpuriques de la dermatite herpetiforme. *Ann Dermatol Venereol* 1987;114:1545–1547.

(5) Volume with supplement

Magni F, Rossoni G, Berti F: BN-52021 protects guinea-pig from heart anaphylaxis. *Pharmacol Res Commun* 1988;20 Suppl 5:75–78.

(6) Issue with supplement

Gardos G, Cole JO, Haskell D, et al: The natural history of tardive dyskinesia. *J Clin Psychopharmacol* 1988;8(4 Suppl):31S–37S.

(7) Volume with part

Hanly C: Metaphysics and innateness: a psychoanalytic perspective. *Int J Psychoanal* 1988;69(Pt 3):389–399.

(8) Issue with part

Edwards L, Meyskens F, Levine N: Effect of oral isotretinoin on dysplastic nevi. *J Am Acad Dermatol* 1989;20(2 Pt 1):257–260.

(9) Issue with no volume

Baumeister AA: Origins and control of stereotyped movements. *Monogr Am Assoc Ment Defic* 1978;(3):353–384.

(10) No issue or volume

Danoek K: Skiing in and through the history of medicine. *Nord Medicinhist Arsb* 1982:86–100.

(11) Pagination in Roman numerals

Ronne Y. Ansvarsfall: Blodtransfusion till fel patient. *Vardfacket* 1989;13:XXVI–XXVII.

(12) Type of article indicated as needed

Spargo PM, Manners JM: DDAVP and open heart surgery [letter]. *Anaesthesia* 1989;44:363–364.

Fuhrman SA, Joiner KA: Binding of the third component of complement C3 by Toxoplasma gondii [abstract]. *Clin Res* 1987;35: 475A.

(13) Article containing retraction

Shishido A: Retraction notice: Effect of platinum compounds on murine lymphocyte mitogenesis [Retraction of Alsabti EA, Ghalib ON, Salem MH. In: *Jpn J Med Sci Biol* 1979;32:53–65] *Jpn J Med Sci Biol* 1980;33:235–237.

(14) Article retracted

Alsabti EA, Ghalib ON, Salem MH: Effect of platinum compounds on murine lymphocyte mitogenesis [Retracted by Shishido A. In: *Jpn J Med Sci Biol* 1980;33:235–237]. *Jpn J Med Sci Biol* 1979;32:53–65.

(15) Article containing comment

Piccoli A, Bossatti A: Early steroid therapy in IgA nephropathy: Still an open question [comment]. *Nephron* 1989;51:289–291. Comment on: *Nephron* 1988;48:12–17.

(16) Article commented on

Kobayashi Y, Fujii K, Hiki Y, et al: Steroid therapy in IgA nephropathy: A retrospective study in heavy proteinuric cases [see comments]. *Nephron* 1988;48:12–17. Comment in: *Nephron* 1989;51:289–291.

(17) Article with published erratum

Schofield A: The CAGE questionnaire and psychological health [published erratum appears in *Br J Addict* 1989;84:701]. *Br J Addict* 1988;83:761–764.

Books and Other Monographs

(18) Personal author(s)

Colson JH, Armour WJ: *Sports injuries and their treatment.* 2nd rev ed. London: S. Paul, 1986.

(19) Editor(s), compiler as author

Diener HC, Wilkinson M (eds): *Drug-induced headache.* New York: Springer-Verlag, 1988.

(20) Organization as author and publisher

Virginia Law Foundation. *The medical and legal implications of AIDS.* Charlottesville: The Foundation, 1987.

(21) Chapters in a book

Weinstein L, Swartz MN: Pathologic properties of invading microorganisms. In: Sodeman WA Jr, Sodeman WA, (eds). *Pathologic physiology: Mechanisms of disease.* Philadelphia: Saunders, 1974: 457–472.

(22) Conference proceedings

Vivian VL, (eds): Child abuse and neglect: A medical community response. Proceedings of the First AMA National Conference on Child Abuse and Neglect; 1984 Mar 30–31; Chicago. Chicago: American Medical Association, 1985.

(23) Conference paper

Harley NH: Comparing radon daughter dosimetric and risk models. In: Gammage RB, Kaye SV, (eds). *Indoor air and human health.* Proceedings of the Seventh Life Sciences Symposium; 1984 Oct 29-31; Knoxville (TN). Chelsea (MI): Lewis, 1985: 69–78.

(24) Scientific or technical report

Akutsu T: Total heart replacement device. Bethesda (MD): National Institutes of Health, National Heart and Lung Institute; 1974 Apr. Report No.: NIH-NHLI-69-2185-4.

(25) Dissertation

Youssef NM: School adjustment of children with congenital heart disease [dissertation]. Pittsburgh (PA): Univ of Pittsburgh, 1988.

(26) Patent Harred JF, Knight AR, McIntyre JS, inventors: Dow Chemical Company, assignee. Epoxidation process. US patent 3,654,317. 1972 Apr 4.

Other Published Material

(27) Newspaper article

Rensberger B, Specter B: CFCs may be destroyed by natural process. *The Washington Post* 1989 Aug 7;Sect A:2 (col 5).

(28) Audiovisual

AIDS epidemic: the physician's role [videorecording]. Cleveland (OH): Academy of Medicine of Cleveland, 1987.

(29) Computer file

Renal system [computer program]. MS-DOS version. Edwardsville (KS): Medi-Sim, 1988.

(30) Legal material

Toxic Substances Control Act: Hearing on S. 776 Before the Subcomm on the Environment of the Senate Comm on Commerce, 94th Cong, 1st Sess. 343 (1975).

(31) Map

Scotland [topographic map]. Washington: National Geographic Society (US), 1981.

(32) Book of the Bible

Ruth 3:1–18: *The Holy Bible.* Authorized King James version. New York: Oxford Univ. Press, 1972.

(33) Dictionary and similar references

Ectasia: *Dorland's illustrated medical dictionary.* 27th ed. Philadelphia: Saunders, 1988: 527.

(34) Classical material

The Winter's Tale: act 5, scene 1, lines 13–16. *The complete works of William Shakespeare.* London: Rex, 1973.

Unpublished Material

(35) In press

Lillywhite HB, Donald JA: Pulmonary blood flow regulation in an aquatic snake. *Science.* In press.

Tables

Type each table double-spaced on a separate sheet. Do not submit tables as photographs. Number tables consecutively in the order of their first citation in the text and supply a brief title for each. Give each column a short or abbreviated heading. Place explanatory matter in footnotes, not in the heading. Explain in footnotes all nonstandard abbreviations that are used in each table. For footnotes use the following symbols, in this sequence: *, †, ‡, §, ‖, #, ¶.

Identify statistical measures of variations such as standard deviation and standard error of the mean.

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The editor, on accepting a paper, may recommend that additional tables containing important backup data too extensive to publish be deposited with an archival service, such as the National Auxiliary Publication Service in the United States, or made available by the authors. In that event an appropriate statement will be added to the text. Submit such tables for consideration with the paper.

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Submit the required number of complete sets of figures. Figures should be professionally drawn and photographed; freehand or typewritten lettering is unacceptable. Instead of original drawings, roentgenograms, and other material send sharp, glossy black-and-white photographic prints, usually 127 × 173 mm (5 × 7 in), but no larger than 203 × 254 mm (8 × 10 in). Letters, numbers, and symbols should be clear and even throughout and of sufficient size that when reduced for publication each item will still be legible. Titles and detailed explanations belong in the legends for illustrations, not on the illustrations themselves.

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All hematologic and clinical-chemistry measurements should be reported in the metric system in terms of the International System of Units (SI). Editors may request that alternative or non-SI units be added by the authors before publication.

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Use only standard abbreviations. Avoid abbreviations in the title and abstract. The full term for which an abbreviation stands should precede its first use in the text unless it is a standard unit of measurement.

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Journals that have notified the International Committee of Medical Journal Editors of their willingness to consider for publication manuscripts prepared in accordance with earlier versions of the committee's uniform requirements identify themselves as such in their information for authors. A full list is available on request from the *New England Journal of Medicine* or the *British Medical Journal.* Citations of this document should be to one of the sources listed below.

International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *N Engl J Med* 1991; 324:424–8. International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *BMJ* 1991; 302:338–341.

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ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1992; 92:122–125). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

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An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

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FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Samples references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics*, ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

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150 mg b.i.d.

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[1] Data on file, Lilly Research Laboratories.

See accompanying page for prescribing information.

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nizatidine capsules

Brief Summary. Consult the package insert for complete prescribing information.

Indications and Usage: 1. *Active duodenal ulcer*—for up to 8 weeks of treatment at a dosage of 300 mg h.s. or 150 mg b.i.d. Most patients heal within 4 weeks.

2. *Maintenance therapy*—for healed duodenal ulcer patients at a dosage of 150 mg h.s. at bedtime. The consequences of therapy with Axid for longer than 1 year are not known.

3. *Gastroesophageal reflux disease (GERD)*—for up to 12 weeks of treatment of endoscopically diagnosed esophagitis, including erosive and ulcerative esophagitis, and associated heartburn at a dosage of 150 mg b.i.d.

Contraindication: Known hypersensitivity to the drug. Because cross sensitivity in this class of compounds has been observed, H_2-receptor antagonists, including Axid, should not be administered to patients with a history of hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*—1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests—False-positive tests for urobilinogen with Multistix® may occur during therapy.

Drug Interactions—No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility—A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups.The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of anti-androgenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

PV 2093 AMP [101591]

Additional information available to the profession on request.

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(continued from p 5A)

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Apr 10–11. **Lyme Disease 1992 State of the Art Conference.** Sheraton Stamford Hotel, Stamford. Contact: Lyme Borreliosis Foundation, Box 403, Tolland, CT 06084-0403. Tel: (203) 871-2900.

FLORIDA

Apr 3–5. **12th Annual National Pediatric Lung and Heart Diseases.** Ponte Vedra Beach, Florida. Contact: Paula Kehrli, Program Coordinator, Nemours Children's Clinic, PO Box 5485, Jacksonville, FL 32247. Tel: (904) 390-3638.

MARYLAND

Apr 6–10. **Perinatal and Pediatric Pathology.** Marriott Hotel, Bethesda. Contact: Education Division, Armed Forces Institute of Pathology, Washington, DC 20306. Tel: (301) 427-5231.

MISSOURI

Apr 2–3. **19th Annual Obstetrics & Gynecology Symposium.** Washington University Medical Center, St Louis. Contact: Cathy Caruso, Office of Continuing Medical Education, Washington University School of Medicine, Box 8063, 660 South Euclid, St Louis, MO 63110-1093. Tel: (800) 325-9863.

AROUND THE WORLD

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Apr 28–May 2. **Continuum of Care for Chronic Psychiatric Patients.** Caribe Hilton and Casino, San Juan. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650. □

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Action: Yohimbine blocks presynaptic alpha-2 adrenergic receptors. Its action on peripheral blood vessels resembles that of reserpine, though it is weaker and of short duration. Yohimbine's peripheral autonomic nervous system effect is to increase parasympathetic (cholinergic) and decrease sympathetic (adrenergic) activity. It is to be noted that in male sexual performance, erection is linked to cholinergic activity and to alpha-2 adrenergic blockade which may theoretically result in increased penile inflow, decreased penile outflow or both.

Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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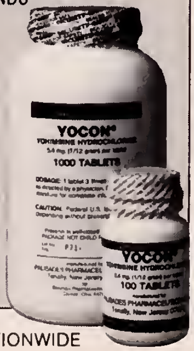

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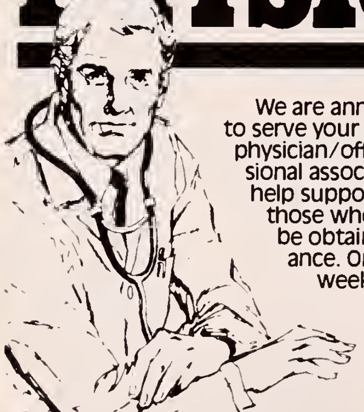

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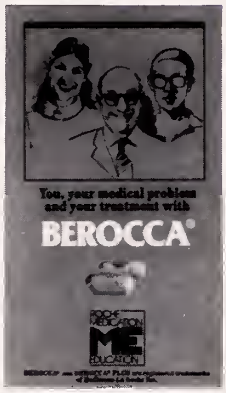
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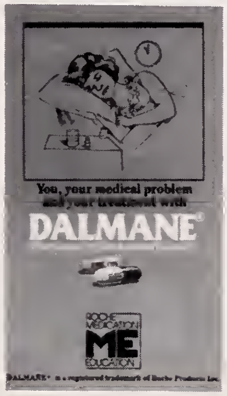
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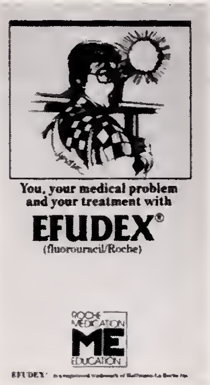
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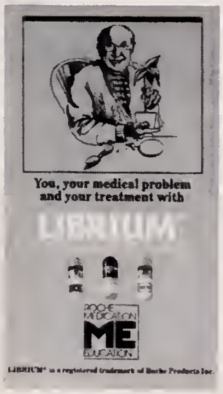
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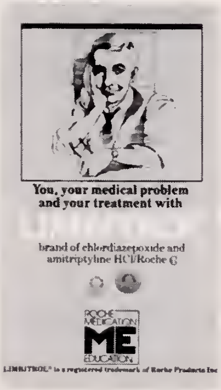
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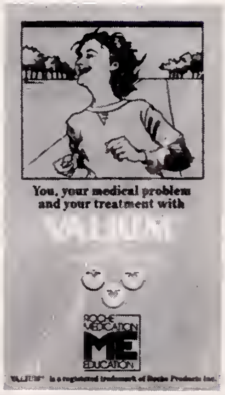
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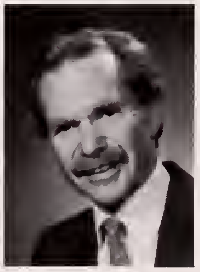
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VOL 92
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1992

EXPLORING POISONING DATA COLLECTION SYSTEMS

DO-NOT-RESUSCITATE AND THE ANESTHESIOLOGIST

DRUG POISONING PATTERNS: THE ALBANY EXPERIENCE, 1978 AND 1988

ADVERSE CUTANEOUS REACTIONS FROM CARDIOVASCULAR DRUG THERAPY

THE GOLDEN YEARS OR THE WASTELAND

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†Constipation, which is easily managed in most patients, is the most commonly reported side effect of Calan SR.

‡Verapamil should be administered cautiously to patients with impaired renal function.

References: 1. Data on file, Searle. 2. Edmonds D, Würth JP, Baumgart P, et al. Twenty-four-hour monitoring of blood pressure during calcium antagonist therapy. In: Fleckenstein A, Laragh SH, eds. *Hypertension—the Next Decade: Verapamil in Focus.* New York, NY: Churchill Livingstone; 1987:94-100. 3. Midtbø KA. Effects of long-term verapamil therapy on serum lipids and other metabolic parameters. *Am J Cardiol.* 1990;66:13I-15I. 4. Fagher B, Henningsen N, Hulthén L, et al. Antihypertensive and renal effects of enalapril and slow-release verapamil in essential hypertension. *Eur J Clin Pharmacol.* 1990;39(suppl 1):S41-S43. 5. Schmieder RE, Messerli FH, Garavaglia GE, et al. Cardiovascular effects of verapamil in patients with essential hypertension. *Circulation.* 1987;75:1030-1036. 6. Midtbø K, Lauve O, Hals O. No metabolic side effects of long-term treatment with verapamil in hypertension. *Angiology.* 1988;39:1025-1029.

BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°,2°,3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

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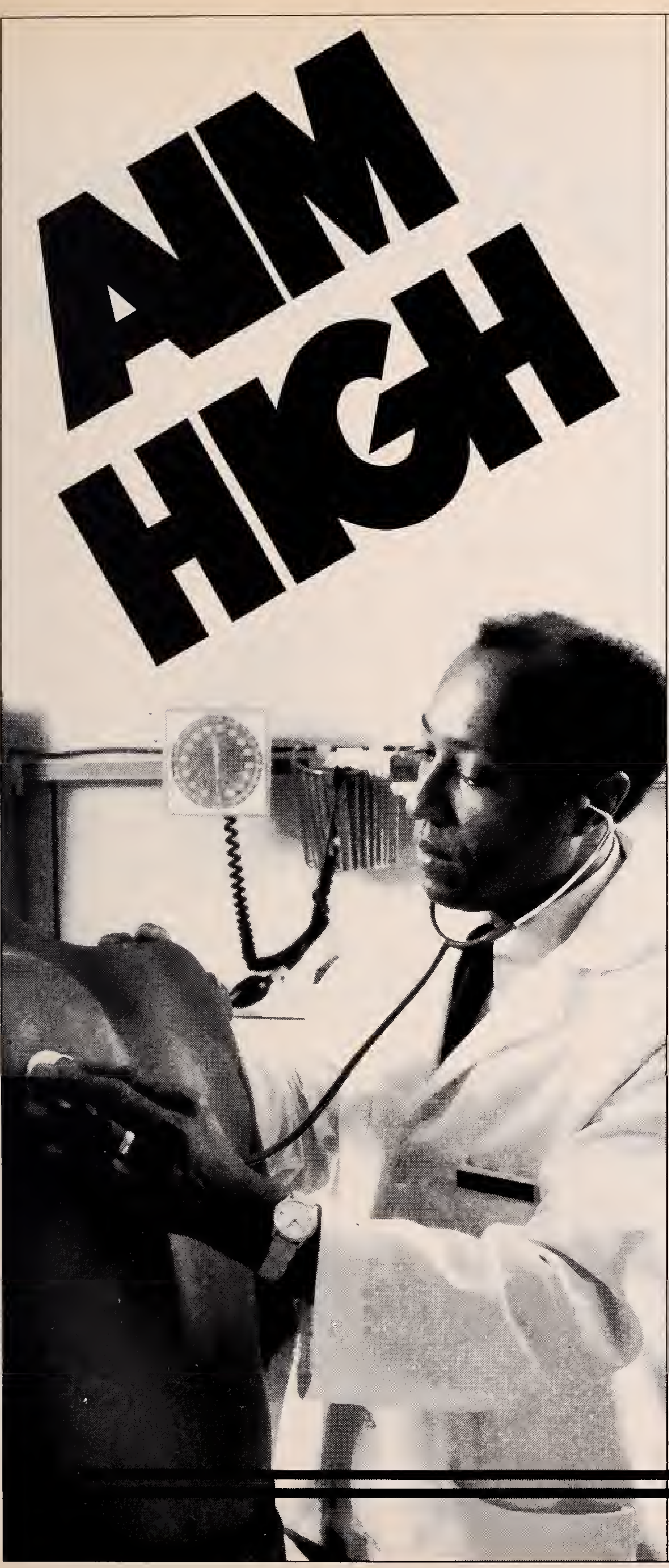

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New York State Journal of Medicine

APRIL 1992 / Volume 92, Number 4

Contents

COMMENTARIES

127 **Exploring poisoning data collection systems**
ROBERT S. HOFFMAN, MD

129 **Do-not-resuscitate and the anesthesiologist**
EARL GREENWALD, MD, CHARLES LOMANTO, MD

RESEARCH PAPERS

131 **The do-not-resuscitate order in a nursing home: Patient's choice or staff's decision**
GISELE P. WOLF-KLEIN, MD; CAROLYN S. WAGNER, RPAC; FELIX A. SILVERSTONE, MD

134 **Drug poisoning patterns: The Albany experience, 1978 and 1988**
M. DONALD McGOLDRICK, MB; RODRIGO URIZAR, MD; JORGE CERDA, MD

REVIEW ARTICLE

137 **Adverse cutaneous reactions from cardiovascular drug therapy**
DAVID M. REINER, MD; WILLIAM H. FRISHMAN, MD; STANLEY LUFTSCHEIN, MD; MARC GROSSMAN, MD

SPECIAL ARTICLE

148 **The Golden Years or The Wasteland**
IRVING A. BUNKIN, MD

COMMITTEE REPORT

151 **Ethical considerations concerning the HIV-positive physician**
FRED ROSNER, MD; PETER P. SORDILLO, MD; JONATHAN R. WOLPAW, MD; PETER B. FARNSWORTH, MD; ALLEN J. BENNETT, MD; ANTHONY BUSCAGLIA, MD; ERIC J. CASSELL, MD; ABRAHAM L. HALPERN, MD; JOHN B. HENRY, MD; PIETER R. KARK, MD; ALLISON B. LANDOLT, MD; LAURENCE LOEB, MD; ROBERT LOWENSTEIN, MD; PATRICIA J. NUMANN, MD; FERNANDO V. ONA, MD; HERMAN M. RISEMBERG, MD; PETER ROGATZ, MD; PHILIP H. SECHZER, MD

(continued on p 6A)

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2. July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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New York State
Journal of Medicine

CASE REPORTS

156 **Primary and secondary aortoenteric fistula and thoracic aortic aneurysm**
A. ADDISON BARMAN, MD, PAUL KERR, DO

158 **Metastatic hemangiopericytoma of the breast**
ARNOLD S. BREITBART, MD; MATTHEW N. HARRIS, MD; MADELINE VAZQUEZ, MD; JULIE R. MITNICK, MD

160 **Spontaneous rupture of the spleen as the initial manifestation of acute myeloid leukemia**
DAVID SERUR, MD, TERENIG TERJANIAN, MD

LETTERS TO THE EDITOR

162 **Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy**
HEIDI FLEISCHMAN, MD; ONYEKACHI IUFDU, MD; ELI A. FRIEDMAN, MD

162 **How we tell our doctors about pain**
MICHELE M. HOOPER, MD

162 **The physician and the poor**
HERBERT GERSHBERG, MD, L. P. HINTERBUCHNER, MD

164 BOOK REVIEWS

171 OBITUARY LISTINGS

174 AUTHOR GUIDELINES

8A MEDICAL MEETINGS AND LECTURES

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MAY 1992

AROUND THE STATE

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May 15. **Albert Einstein International Symposium in Urology.** Albert Einstein College of Medicine. Contact: Office of Continuing Medical Education, Montefiore Medical Center, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212) 920-6674.

BROOKLYN

May 14–17. **Jewish Medical Ethics–Third International Conference.** State University of New York Health Science Center at Brooklyn. Contact: Michael Akerman, MD, SUNY-HSCB, 450 Clarkson Ave, Box 19, Brooklyn, NY 11203. Tel: (718) 245-3849.

EAST ELMHURST

May 20. **A Clinical Day in Ophthalmology. Ocular Manifestations of Systemic Disease.** Holiday Inn Crowne Plaza. Contact: Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

LIVERPOOL

May 21. **Seventh Annual Symposium on Contemporary Topics in Clinical Micro-**

(continued on p13A)

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COMMENTARIES

Exploring poisoning data collection systems

In the current issue of the *Journal*, McGoldrick et al [1] present data on poisonings in Albany for the years 1978 and 1988. The data comprise 240 hospital admissions that resulted from a total of 1216 emergency department visits. Patient demographics, drugs ingested, mortality, and the need for extracorporeal drug removal are investigated. The authors suggest that "an apparent substantial change in the pattern of poisonings was observed."[1] This pattern consists of an increase in admissions, a closing of a "gender gap" noted in 1978, and a stabilization of the need for extracorporeal drug removal. To explore the implications of this study, one must first understand the nature of poison data collection systems.

Currently, there are three major poisoning databases in the United States: the American Association of Poison Control Centers National Data Collection System (AAPCC NDCS), the Drug Abuse Warning Network (DAWN), and the Centers for Disease Control's National Center for Health Statistics (NCHS). The AAPCC NDCS pools standardized data from 72 member poison control centers and reports on over 1.7 million annual "exposures."[2] Over 81% of these "exposures" initiate from the home and are largely unsubstantiated by either reliable history or confirmatory testing. Whereas "exposures" that originate from hospitals offer more reliable data, they are often recorded on the basis of the initial impression of the caller, and not modified on the basis of the final patient outcome or laboratory testing. The DAWN system collects data from hospitals and medical examiners' offices in a small number of metropolitan areas.[3] This system relies on "mentions" in hospital charts, which are collected and coded regardless of laboratory or clinical substantiation. Although data derived from medical examiners' reports are more reliable, the entire DAWN system has received severe criticism for a variety of issues.[4] These other potential errors in DAWN reporting stem from the use of nonmedical personnel for coding, the variable inclusion of ethanol, a changing database, and payment to centers on the basis of the number of "mentions" potentially resulting in data "padding."[4] Finally, the NCHS system focuses on death certificates and ICD-9 coding. Pollock et al recently discussed the lack of specificity of these ICD-9 codes, such as the inclusion of cocaine data with toxicity from other local anesthetics.[5]

Although these three data collection systems have the potential to overlap, they survey three very different aspects of poisoning: home events, hospital presentations, and fatalities, for the AAPCC, DAWN, and NCHS data, respectively. Conclusions about national patterns of poisoning based on data from any single source might be misleading. Large discrepancies have been noted when DAWN and NCHS data are compared.[5] In spite of the limitations of any one data collection system, rigorous data analysis may yield constructive results. These results, however, are only interpretable within the confines of the database. An excellent example of this limitation was recently presented by Weisman and Goldfrank in a discussion of a designer opioid epidemic in New York City.[6] A local epidemic of hospitalizations resulting from the substitution of a fentanyl derivative for heroin flooded the New York City Poison Control Center with inquiries. This surge produced a skew in local data, that would have produced catastrophic results if interpreted on a national scale.

The last consideration must focus on the issues of reporting and capture. The ideal database captures every case. For example, if every death must be certified and all death certificates are entered into a single database, a system that analyzes death certificates will capture 100% of poisoning deaths. This assumes, of course, that the cause of death is properly established and certified in every case. A database (such as DAWN) that relies on charting "mentions" relies on the ability of the person charting to recognize a poisoning. Rare poisonings and poisoning epiphenomena (such as trauma that occurs as result of intoxication) are easily missed. Voluntary reporting systems such as the AAPCC NDCS often rely on the experience of the reporter. Physicians are less likely to contact a poison control center for information on an ingestion of a common medication than for exposure to a rare and often fatal toxin. Thus, voluntary reporting systems based on accessing expert information (contacting a poison control center for assistance) are biased away from reporting common exposures and are determined by individual physician learning curves.[6]

Let us now apply these principles to the evaluation of the Albany experience. The first issue is population. McGoldrick et al focus on in-patients at Albany Medical Center Hospital. To compare in-patients in 1978 and 1988, a number of assumptions are required. Although the authors acknowledge that only a small percentage of poisonings require hospitalization, the implication is that hospitalized patients still represent the overall experience. Tracing the events that occur from poison exposure to hospital admission provides useful criteria for interpretation of in-patient data. The patient must be exposed (usually by ingestion). They must then gain access to the health care system, often by ambulance transport. In interpreting the data, it would be helpful to know whether or not hospitals have either opened or closed in the Albany area between 1978 and 1988 and whether or not admission rates for other common events increased or decreased. Given a stable population and community, direct comparison of events in these two years would be valid. After arriving at the hospital, poisoning must be recognized, assessed, and treated. Improved laboratory testing procedures might increase poison detection, allowing for previously undiagnosed admissions to be coded as poisoning cases. Changes in emergency department staffing or practices can also influence admission rates. For example, Albany Medical Center established an Emergency Medicine residency program during this time period. This change might have resulted in better physician training in the recognition of poisonings. In addition, it would be helpful to know whether acceptable emergency department observation times decreased as a result of the residency program, possibly accounting for the increased number of admissions of patients that would have previously been discharged after a prolonged emergency department stay. The authors do not comment on the fact that in 1988 less emergency department visits were required to produce a significant increase in-hospital admissions. Although this may reflect an increased severity of poisoning, it may also reflect a change in admission policy. Although the potential for confounding variables clearly exists, these issues do not intrinsically invalidate the database as changes in the practice of medicine always occur over time.

Capture was based on retrospective chart review and therefore suffers from the issues of recognition and charting discussed above. Severity was based on mortality and the need for extracorporeal drug removal. Conclusions based on 5 deaths and 16 extracorporeal procedures must be viewed with some degree of caution. Finally, the reader must ask what the comparison of 1978 and 1988 data represents. Although it would be helpful to know whether a trend has taken place over those 10 years, in reality only a direct comparison can be made. The number of hospital admissions for poisoning could have skyrocketed from 1978 on and is now on the decline, or could have plummeted and is now increasing. No trend analysis is possible with only two data points. Thus, only a limited interpretation of this data is permitted. In spite of these limitations, the authors present some stimulating issues that require further evaluation. Specifically, the concepts of a gender gap and age-specific mortality remind practitioners that poisoned patients (like all patients) must be approached in totality, rather than simply focusing on the ingested toxin.

Poisoning surveillance is a worthwhile endeavor. Changing patterns of both drug abuse and suicidal ingestion can alter diagnostic and management strategies and improve the focus of public education campaigns. America is in need of a reliable poisoning database. Until such a system exists, individual efforts to define local problems such as the work by McGoldrick et al[1] must continue. We must be cautious, however, about applying the results of these studies to larger populations.

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1. McGoldrick MD, Urizar R, Cereda J: In-patient drug poisoning patterns, the Albany experience (1978 and 1988). *NY State J Med* 1992; 92:134–136.
2. Litovitz TL, Bailey KM, Schmitz BF, et al: 1990 Report of the American Association of Poison Control Centers National Data Collection System. *Am J Emerg Med* 1991; 9:461–509.
3. National Institute on Drug Abuse: Data from the drug abuse warning network (DAWN): Annual data 1988. Rockville, Md, Pub Heal Ser, US Dept of Heal Hum Ser, 1989.
4. Ungerleider JT, Lundberg GD, Sunshine I, et al: DAWN: Drug abuse warning network or data about worthless numbers? *J Anal Toxicol* 1980 ;4:269–271.
5. Pollock DA, Holmgreen P, Lui KJ, et al: Discrepancies in reported frequency of cocaine-related deaths, United States, 1983 through 1988. *JAMA* 1991; 266:2233–2237.
6. Weisman RS, Goldfrank LR: Poison center numbers. *Clin Toxicol* 1991; 4:553–557.

Do-not-resuscitate and the anesthesiologist

Do-not-resuscitate (DNR) orders have become widely accepted and instituted in health care facilities in the United States. For many patients, families, and health care professionals, the arrival of regulations permiting non-interference with certain aspects of the normal dying process was welcomed. Previously, all involved in the dying process experienced prolongation of suffering because of perceived legal, ethical, and moral mandates to preserve life as long as possible and at all costs.

Two achievements have provided the long-desired means to deal with dying in a more humane and reasonable manner: recognition of the moral, ethical, and legal propriety of withholding attempts to resuscitate a patient under certain conditions, and delineation and implementation of policies and procedures overseeing this process. Methods of providing appropriate information to assist patients, their proxies, and/or family in DNR decision-making are defined, and systems of medical record documentation and requirements for issuance of DNR orders, and for their periodic review, have been established.

The most familiar process for obtaining consent for a DNR order occurs between the primary physician and the patient, the patient's proxy, and/or family. There is a broad spectrum of clinical situations in which patients request DNR orders. The most common arise among patients in or near terminal phases of fatal illnesses. All persons involved frequently agree at this point that resuscitation may impose burdens in excess of benefits.

The hospital environment, however, is a setting in which there is a broad range of relationships. Variations in the context of these relationships have begun to reveal previously unanticipated problems. The relationship between patients and anesthesiologists is unique. Under general anesthesia, patients may experience iatrogenic respiratory arrest, in which case anesthesiologists essentially initiate resuscitation by intubating and placing them on a respirator.

Can the anesthesiologists allow death when faced with a readily reversible cardiac arrest during the administration of general anesthesia to a patient in the terminal phase of a fatal illness? Many would be able to deal with withholding cardiac resuscitative efforts in this situation.

What of the patient who is experiencing a fatal disease that is not in a terminal phase, and is not, at the moment, causing suffering? More perplexing, what of the patient in generally good health who would prefer not to experience resuscitation if cardiac or respiratory arrest were to occur coincidentally during general anesthesia for treatment of a curable disease? Can and should the anesthesiologist comply with a DNR order in these situations?

In New York State, DNR regulations permit patients to request orders not to resuscitate in the event cardiopulmonary arrest without regard to their medical conditions.[1] Indeed, the issue is the right of people with the capacity to make health care decisions or, lacking that capacity, for appropriately appointed agents to decide what may be done. Regulations and medical ethics require that providers, and here we are speaking about anesthesiologists, comply with a DNR order or, if they cannot or will not do this because of moral, ethical, or other considerations, facilitate transfer of the patient to a provider who will comply.

Confronted with a DNR order written for a patient who is most likely to survive resuscitative efforts, an anesthesiologist must decide whether or not it is acceptable to withhold minimally or totally non-invasive treatments with a likelihood of success with minimal or no sequelae. What would be the ethical, moral, and legal implications and consequences of withholding treatments in these situations?

The writing of a DNR order requires careful counseling by the physician and thorough consideration by the patient, proxy, or family, of all existing and reasonably predictable factors. A patient's medical circumstances may be quite different when consent for a DNR order is initially obtained compared with those prevailing when consent for anesthesia is requested. The spectra of anesthesia, loss of self-control, and self-determination, bodily disfigurement by surgery, and the physiologic impact of both anesthesia and surgery are important factors. None of them may have been operative or even known by the patient, proxy, or family at the time of initial consent to the DNR order. The request not to resuscitate may no longer be acceptable conceptually to the patient, proxy, or family when the events that may occur during general anesthesia and the possibility of reversing some of these events are discussed. Many patients, proxies, or families may feel that it would be reasonable to provide resuscitation under these circumstances even if they previously consented to a DNR order.

It is incumbent on anesthesiologists to revisit the issue of DNR orders with patients, proxies, or families during pre-anesthesia counseling. The reaffirmation process should be recorded by the anesthesiologists in the medical record. If the patient, proxy, or family requests, an order should be written to suspend the DNR order during anesthesia. If the desire for withholding resuscitation is reaffirmed by the patient, proxy, or family, it should be honored during anesthesia.

It is essential that departments of anesthesia be notified at the earliest possible moment of discussions to perform surgery requiring general anesthesia on patients with DNR orders. This allows time for anesthesiologists to locate and convene patients, proxies, or families in order to counsel them. These encounters should include open discussion of the impact of surgery and anesthesia, so that the patient, proxy, or family can incorporate these new considerations into their decision with respect to an existing DNR order. Reaffirmation of the DNR order should be documented in the medical record by the anesthesiologist.

If pre-anesthesia counseling includes reaffirmation, the anesthesiologists has a reasonable opportunity to consider options. As with all physicians, anesthesiologists have the right not to provide care if they believe that they cannot comply with a DNR order. Through mechanisms defined by policy, they can implement an orderly transfer of the patient to the care of another anesthesiologist willing to honor the DNR order.

Acknowledgments. We wish to acknowledge significant contributions to this document by the Ethics Committee of Staten Island University Hospital and by Kathleen Powderly, Assistant Director, Division of Humanities in Medicine, State University of New York, Health Science Center at Brooklyn.

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1. NYS Pub Heal Law Art 29-B, §2960-78.

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SUDDEN ASPHYXIAL DEATH

Asphyxial death is usually sudden. A boy goes out to swim too soon after a big meal, is seized with "cramps," drowns alone, or if pulled ashore fails to rally. Firemen or police come with a "resuscitator," but resuscitation is not accomplished. One sometimes wonders why a physician is not called. Has the public lost confidence in our ability to revive an asphyxiated person?

In 1933 an article was published in the *American Journal of Surgery* entitled "Asphyxial Death a Professional Disgrace." Out of the interest it aroused the Society for the Prevention of Asphyxial Death was incorporated, with its founder, Dr. Paluel J. Flagg, as president-director. Dr. Flagg is asking some pertinent questions. Of the general practitioner he is inquiring: What routine is followed in an ear and throat postoperative patient who shows signs of asphyxia after being put back to bed? Of medical school officers he would like to know what instruction is given to students if they were called on to revive an asphyxiated person, other than the Schafer or the Silvester method? How many can use an electrically lighted laryngoscope or remove a foreign body by direct vision? Of firemen and policemen: What stages or degrees of asphyxia can you men recognize? Give the treatment you would apply other than mechanical. Impending death from asphyxia is of immense importance. Why then is so little known about it among "general men?" Answer: They have never been taught.

In order to raise money to do just this, Dr. Flagg has a plan whereby anybody who lost a son in this War may buy a twenty-five dollar bond, and offer it or its equivalent to the Society for the Prevention of Asphyxial Death to develop a fund, the proceeds from which may be used to instruct doctors, nurses and other medical personnel in the art of resuscitation.

(*NY State J Med* 1945; 45:1764)

The do-not-resuscitate order in a nursing home: Patient's choice or staff's decision

GISELE P. WOLF-KLEIN, MD; CAROLYN S. WAGNER, RPAC; FELIX A. SILVERSTONE, MD

ABSTRACT. An 86-year-old women in a residential health care facility suffered a massive stroke; immediate intubation was performed, she was transferred to a nearby hospital where she died two months later.

Within 48 hours of this event, three mentally competent residents and one family member on the unit requested a do-not-resuscitate order. They, along with the seven other mentally competent and non-terminal residents on the unit, participated in a questionnaire survey, the purpose of which was to elicit information on DNR attitudes. Only one respondent requested cardiopulmonary resuscitation. All insisted that do-not-resuscitate decisions were theirs alone to make. A staff questionnaire was given to 81 employees having many years of health care experience. Most had witnessed death and cardiopulmonary resuscitation and knew of its poor outcome. However, 51% were very willing to participate in cardiopulonary resuscitation, and 65% thought cardiopulmonary resusciation was worthwhile in residential health care facilities. Furthermore, 56.8% thought that families should participate in the decision. The dichotomy between residents' wishes and staff perceptions merits recognition and further study.

(NY State J Med 1992; 92:131–134)

A new provision of the NYS Public Health Law, titled Orders Not to Resuscitate (article 29-B as added by chapter 818 of the laws of 1987), became effective on April 1, 1988. This provision permits patients or a surrogate, if need be, to consent to a do-not-resuscitate (DNR) order, an advance directive not to resuscitate in the event of cardiopulmonary arrest.[1]

Along with other health care facilities, the Parker Jewish Geriatric Institute, a 527-bed residential health care facility (RHCF) located in New Hyde Park, developed a DNR protocol. Competent residents were asked to express their wishes regarding resuscitation should they sustain a cardiorespiratory arrest. In the case of incompetent residents, the next of kin was contacted by the physician. However, many residents and their families did not come to a final decision and remained without a documented DNR choice.

In September, an alert and active elderly woman with rheumatoid arthritis and atherosclerotic heart disease developed sudden and unexpected cardiovascular decompensation and experienced respiratory arrest. This event occurred in her residential unit, at a time when other residents were returning from the dining room and families were visiting. The resident had returned to her room and called the nurse because she felt ill. The nurse, on entering the room, realized that the resident was in acute distress and called a code. A physician and a physician assistant were present on the floor at the time and instituted cardiopulmonary resuscitation (CPR) within one minute. Intubation was successful at the first attempt. An intravenous line was placed and cardiac monitoring was initiated. She was immediately transferred with ventilatory assistance to the adjoining hospital, where she died two months later of a myocardial infarction and a cerebrovascular accident.

Within two days of this resident's resuscitation, three competent residents and one daughter of a mentally incompetent resident in the same unit approached the medical team and requested that no such efforts, namely resuscitation, be undertaken should they have a sudden cardiorespiratory arrest.

Within two to three months later, all of the other competent residents in the unit were individually interviewed by the unit physician and social worker. A resident-oriented questionnaire was used to elicit data, to encourage verbalizing concerns, to alleviate anxieties by education and understanding, and to assist the residents in the appropriate considerations for making a DNR decision. After this survey, a second questionnaire was designed and administered to staff members to explore their understanding, concerns, and attitudes about the DNR order for residents.

METHODS

Resident Questionnaire. The resident questionnaire consisted of 14 questions designed to determine attitudes toward and understanding of CPR and the DNR order. Of the 21 residents occupying the unit, 9 were male, and 12 were female. Four of the males and seven of the females were alert and competent and did not have terminal illnesses or depression. Their average ages were 71 and 81 years, respectively. One of these 11 residents was admitted to a hospital shortly after the earlier event, and therefore questionnaires were obtained from the remaining ten.

Staff Questionnaire. A weekly, routine team meeting provided a sample of 81 staff members, who were asked to complete a 47-item questionnaire. The staff consisted of 20 (24.7%) physicians, 8 (9.9%) recreational therapists, 15 (18.5%) nurses, 9 (11.1%) nurse's aides, 7 (8.6%) social workers, and 22 (27.2%) physical and occupational therapists. The average age of the staff was 34 years, and their average length of health care experience was 13.5 years. They had been employed at The Parker Jewish Geriatric Institute for 5.5 years on average, which attested to their gerontologic experience. The majority of the staff were married (69.2%), white (60.5%), and college educated (75.3%); 24% considered themselves very religious,

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Address correspondence to Dr Wolf-Klein, Physician-in-Charge, Geriatric Community Health Center, The Parker Jewish Geriatric Institute, 271 11th Ave, New Hyde Park, NY 11042.

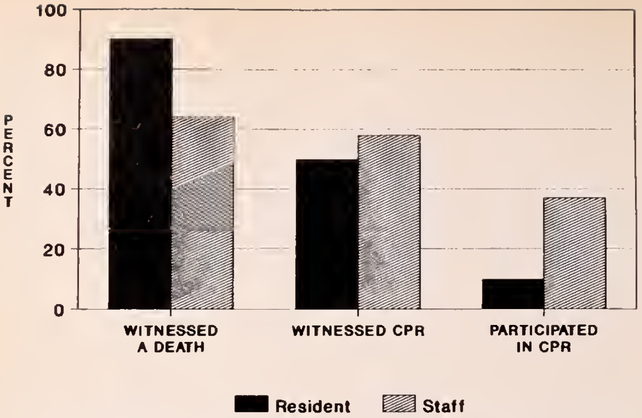

FIGURE 1. Resident vs staff personal experience with death and DNR/CPR.

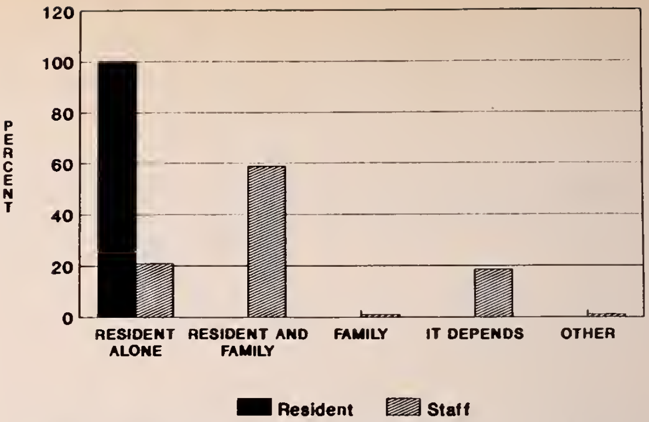

FIGURE 3. Resident vs staff opinions regarding who should make DNR decisions.

and 42% considered themselves somewhat religious. The staff questionnaire dealt with their personal experience with death and CPR, their reaction to five case vignettes, and their general beliefs regarding DNR guidelines for nursing home residents.

Results

Resident Questionnaire. Nine participants reported having thought about their own death. They had witnessed the death of parents or spouses and wanted a "quick and quiet" death, preferably in their sleep. Four of these residents knew what a DNR order meant, and six residents were familiar with CPR. One half of the group had actually witnessed a resuscitation attempt, and one had participated (Fig 1), yet no one had made a personal decision regarding the DNR/CPR issue.

The resuscitated resident was known to seven of the ten participants as a peer on the unit. They had learned that she was minimally responsive in the intensive care unit, was on a respirator, and had had multiple "tubes" for feeding, fluid intake, and medications.

When questioned whether their thoughts about CPR and DNR had changed since this incident, three of the ten participants reported that they would now want a DNR order. Three of the remaining seven participants said that, although they had previously decided against CPR, they had not made this decision known to any staff member until approached with the questionnaire. Three residents were still undecided; although not yet willing to sign the DNR form, they were opting against CPR. The remaining resident chose resuscitation (Fig 2). Even though he had actually witnessed his wife's death after an unsuccessful resuscitation attempt, he stated: "As long as there is a spark of life, there is hope."

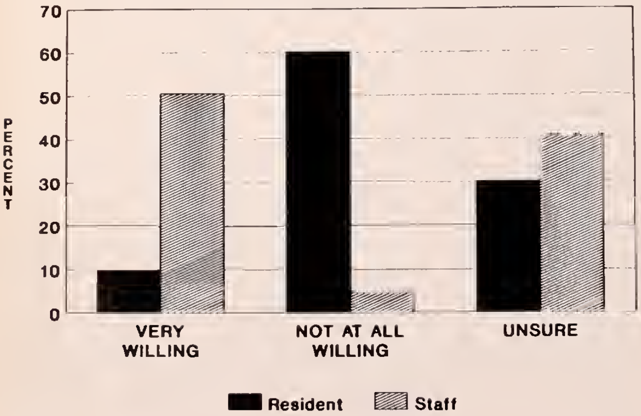

FIGURE 2. Staff willing to perform CPR vs resident willing to be resuscitated.

Although nine of the ten residents had discussions with family members before making a decision, all residents responded that the DNR decision was solely their own. Furthermore, only one of the ten residents wanted discussions with clergymen or lawyers either before or after making their decision.

Five residents thought that the best time to be approached about the DNR/CPR decision was within the first week of admission. Four residents suggested an approach within the first month of admission. All residents found helpful a structured interview presenting, in a stepwise fashion, the issues relating to DNR.

Staff Questionnaire. As with the residents, the majority of the staff members had had personal experiences with critical illness and death in their own families (63%), and half of these deaths had involved CPR. Fifty-two staff members had actually been at the side of a dying patient, and the majority of them had witnessed a resuscitation attempt. In fact, more than a third of the staff had actually participated in a resuscitation attempt (Fig 1). The outcome of these CPR attempts was death in 54.1%, and initial survival was soon followed by death in 16.2%. Twenty-seven percent of the patients were thought to have survived, although the length of survival and functional status remained unknown. Forty-three percent of staff participants believed CPR is sometimes successful for older people in nursing homes, 39.5% regarded it as only rarely successful, and 2.5% responded that it is never successful. The remaining staff members were unable to offer an opinion. However, the majority of the staff (50.6%) were very willing to be involved in a resuscitation attempt, 28.4% were somewhat willing, 12.3% were not very willing, and 4.9% were not at all willing (3.8% did not respond) (Fig 2).

The majority of the staff (89.7%) had discussed DNR or CPR in general terms with their own family members (75.3%), friends (38.3%), health care professionals (50.6%), clergy (7.4%), and with lawyers (4.9%). However, staff usually had not discussed DNR or CPR with a resident or the family member of a resident (54.3%). The staff was equally divided in deciding whether or not to attempt CPR in every resident when no prior formal decision regarding DNR had been made. They favored the recommendation (53.1%) that all residents admitted to the nursing facility be approached about DNR or CPR. A third of the staff (34.6%) stated that only those patients who were alert, that is, not confused, be consulted. For most residents, the staff thought that residents and families should be involved in the discussion about DNR and CPR (74%) and in the decision (56.8%) (Fig 3). In cases of incompetent or confused residents, they would rely on the spouse to make the decision. In the absence of a spouse, the child visiting most frequent would be selected.

When family members disagreed, the staff thought the spouse's opinion should prevail (65.4%) or the opinion of the courts should prevail (13.6%). Four percent indicated that the nursing home should make the final decision. The staff was remarkably divided regarding what should be the main determinant of the DNR status, with 29.6% agreeing that the medical status was the key to the decision and 24.7% disagreeing with this statement; similarly, 16% stated that the mental status was the key to the decision, and 39.1% contradicted this statement. Furthermore, 32% felt that CPR should be attempted on patients who do not have a DNR order, and 25.9% disagreed.

Confronted with five typical case presentations, the general consensus was to resuscitate a young car accident victim (76%) and an active 55-year-old businessman found at the point of death in bed (62%). However, in the case of a 50-year-old woman with a terminal brain tumor who suffers respiratory arrest, only 25% of the staff strongly felt that CPR should be attempted, with 40% clearly expressing no need for resuscitative measures, and a last third (34%) remaining unsure. In the case of an alert 84-year-old resident of a nursing facility with arthritis and functional disability who collapses suddenly, 80% of the staff would definitely attempt resuscitation, with only 7% not instituting CPR, and 12% unsure. The last case was a 72-year-old, long-standing Alzheimer's patient, institutionalized for seven years, found by the nurse in his bed not breathing. Thirty-six percent of the staff would definitely provide CPR, 40% would definitely not provide CPR, and 25% remained unsure.

DISCUSSION

The majority of the ten residents had experienced the death of close relatives and had even witnessed CPR. Of the 10, 9 did not want DNR and felt this decision was theirs alone to make. The staff participants believed CPR was generally worthwhile in elderly people and were quite willing to participate in a resuscitation attempt. Furthermore, they generally thought the DNR decision should be made by the resident and the family. The reasons for the preference for family involvement by the staff compared with personal decision making by the resident are not clear, since both groups had witnessed death and even CPR. The staff may have believed that the resident is less able to make a correct choice than the more healthy and perhaps younger relatives. On the other hand, residents may want to retain complete control over a decision between themselves and the doctor and not be subject to family opinion. Alternatively, residents may seek not to inflict the pain of this decision on the family. Elderly residents tend to have a different outlook on life and death and may not feel that they can communicate or share this perspective with younger relatives.

The sharp difference in the staff's willingness to resuscitate in contrast with the residents' choices again is unclear. Perhaps the staff, trained in preserving life and reversing the pathological process, are motivated unidirectionally to apply their skills and hope for success. Residents do not share this motivation. The staff's perspective, professionally and ethically, is inclined to value and preserve life in a general sense, whereas residents are concerned about their subjective personal life situation. Additionally, residents may be driven by a fear of suffering and a depressed outlook, whereas the staff may be focusing on an optimistic degree of success.

Perhaps our most important finding is the enthusiastic response of residents with regard to the usefulness of the questionnaire in making their choices. At a time of increasing pressure on the health care system, aggravated by the extensive use of costly intensive care units, we suggest that reasonable and rational efforts should and can be made to avoid inappropriate aggressive measures. Several studies have addressed the role of clinical decision-making in ordering DNR and the initiation and withdrawal of life support.[2–12] The physician's role in avoiding inappropriate resuscitation seems better practiced in patients with terminal disease than in the chronically ill elderly.[13–15] A reassessment of appropriate policies and practice, particularly in nursing homes and long-term institutions, is generally needed, and this need is highlighted by our findings.[16–21]

The education and training of physicians and other health care providers with regard to the role of the patient, the patient's surrogate for DNR decisions, and the patient's proxy for general decisions including DNR must encompass the concept of patient autonomy and the legalization of advance health care directives.[16–21] Under the Nursing Home Minimum Standards of the New York State Public Health Law, Part 415, residents of nursing homes have the right not only to be informed of their diagnosis and treatment, but also the right to refuse treatment. These rights are now in effect on a nationwide scale by the Federal Patient Self-Determination Act, which became effective toward the end of 1991. The DNR and the Health Care Proxy Laws[1,22] in New York delineate the process of refusal of CPR and the right to appointment of a proxy for health care decisions, the latter in the event of the resident's incapacity.

Widespread education of the public, particularly of the elderly and their families, needs to be encouraged and initiated. As patient advocates, physicians will be expected to and should play a major role in education pertaining to health care directives and patients' rights. Patients and their families will expect their physicians to provide such information and guidance. Such education should not be postponed to a time of crisis when medical action must occur without delay. Preferably, patients and next-of-kin should be informed during routine contacts regarding the existence of CPR procedures and the options and actions available to them. It is essential that all patients clearly understand the difference between CPR and other types of aggressive medical care. Many patients and many physicians still believe that a DNR order precludes any other form of intensive medical treatment such as intravenous therapy, artificial feeding, diagnostic investigations, and other therapeutic procedures.[23] Descriptive material for patients, including pictorial representations of intubation and resuscitative technologies, and the use of a DNR questionnaire similar to the one described in this report may prove helpful. Informing residents and families about DNR regulations and health care proxies and supporting them in their decision is becoming an essential part of today's geriatric practice.

Acknowledgments. The authors thank Tom Donlon, RN, Lorraine McCrystal, RN, and A. Belanoff, CSW, for their assistance with the questionnaire, and the patients, their families, and The Parker Jewish Geriatric Institute staff who graciously participated in this study.

REFERENCES

1. NYS Do Not Resus Law Art 29-B tit 10 §405.43
2. Fox M, Lipton HL: The decision to perform cardiopulmonary resuscitation. *N Engl J Med* 1983; 309:607–608.

3. *Guidelines on the Termination of Life-Sustaining Treatment and the Care of the Dying*. Bloomington, In, Indiana University Press, 1987.
4. Levinson W, Shepard MA, Dunn PM, et al: Cardiopulmonary resuscitation in long-term care facilities: A survey of do-not-resuscitate orders in nursing homes. *J Am Geriatr Soc* 1987; 35:1059–1062.
5. Lo B, Johnsen AR: Clinical decisions to limit treatment. *Ann Intern Med* 1980; 93:764–788.
6. Miles SH, Gomex CF, Zweibel NR, et al: Nursing home policies addressing the use or withdrawal of life-sustaining medical treatments. *Clin Geriatr Med* 1988; 4:681–690.
7. Miles SH, Ryden MB: Limited-treatment policies in long-term care facilities. *J Am Geriatr Soc* 1985; 33:707–711.
8. Murphy DJ: Do-not-resuscitate orders: Time for reappraisal in long-term care institutions. *JAMA* 1988; 260:2098–2101.
9. Ruark JE, Raffin TA: Initiating and withdrawing life support. *N Engl J Med* 1988; 318:25–30.
10. Schiedermayer DL: The decision to forgo CPR in the elderly patient. *JAMA* 1988; 260:2096–2097.
11. Tomlinson T, Brody H: Ethics and communication in do-not-resuscitate orders. *N Engl J Med* 1988; 318:43–46.
12. Wanzer S, Adelstein J, Cranford R, et al: The physician's responsibility toward hopelessly ill patients, a second look. *N Engl J Med* 1984; 310:955–959.
13. Wanzer SH, Federman DD, Adelstein SJ, et al: The physician's responsibility toward hopeless ill patients, a second look. *N Engl J Med* 1989; 320:844–849.
14. Bedell SE, Pel Le D, Maher PL, et al: Do-not-resuscitate orders for critically ill patients in the hospital: How are they used and what is their impact? *JAMA* 1986; 256:233–237.
15. Lipsky MS, Hickey DP, Browning G: Treatment limitations in nursing homes in Northwest Ohio. *Arch Intern Med* 1988; 148:1539–1541.
16. Kapp MB: Decision making by and for nursing home residents. *Clin Geriatr Med* 1988; 4:687–679.
17. Areen J: The legal status of consent obtained from families of adult patients to withhold or withdraw treatment. *JAMA* 1987; 258:229—335.
18. Glasser G, Zweibel NR, Cassel CK: The ethics committee in the nursing home: Results of a national survey. *J Am Geriatr Soc* 1988; 36:150–156.
19. Uhlman RF, Clark H, Pearlman RA, et al: Medical management decisions in nursing home patients: Principles and policy recommendations. *Ann Intern Med* 1987; 106:879–885.
20. President's commission for the study of ethical problems in medicine and biomedical and behavioral research: Deciding to forego life-sustaining treatment: Ethical, medical, and legal issues in treatment decisions. Washington DC, US Government Printing Office, 1983, pp 440–456.
21. NYS Heal Care Prox Law Art 29-C tit 10 §405.43
22. Blackhall LJ: Must we always use CPR? *N Engl J Med* 1987; 317:1281–1285.

Drug poisoning patterns: The Albany experience, 1978 and 1988

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ABSTRACT. This study retrospectively compares the demography and patterns of poisonings and their local variations among patients admitted to Albany Medical Center Hospital in the years 1978 and 1988. An increase in the number of admissions during this period of time, and an apparent substantial change in the pattern of poisonings was observed; in addition, the gender difference observed in 1978 disappeared. Although street drugs were a common source of poisoning, the resulting morbidity and mortality was low. The use of extracorporeal drug removal procedures appears to have declined in this institution in the two years evaluated.

(NY State J Med 1992; 92:134–136)

The incidence of poisonings by medicinal and non-medicinal agents in the United States is 20.2 per 100,000 population per year, affecting nearly five million people nationwide. Three million of the five million poisonings are related to smoke inhalation.[1] Poisoning fatalities by all substances total 11,000 per year, with 6,094 (55.5%) related to pharmaceutical products, 3.660 (33%) to carbon monoxide, and the remainder to other solid and liquid household products and pesticides.[2,3]

Although the mortality rate may be as high as 34% in comatose (stage IV) poisoned patients, overall it is usually less than 1%.[4–7] Only 13 of 100 persons described in phone calls to a poison control center will lead to treatment in an emergency department, and three of these patients will be admitted to the hospital.[1,8] This paper addresses the 3% of poisoned patients who were admitted to the Albany Medical Center Hospital (AMCH) in the year 1978, and compares them with those entering the same institution in 1988. We emphasize the poisoning patterns of this decade in the Albany area and their local clinical implications.

PATIENTS AND METHODS

The data presented here have been drawn from a poison treatment center and from national statistics that include information from routine Emergency Department intake.[9] These data were contrasted with those of the New York State Hospital Discharge Information (SPARCS, 1988) statewide statistics, kindly provided in a personal communication by Candace Noonan, research scientist, of the Bureau of Communicable Disease Control, NYS Department of Health (1990). Poisoning has been defined as the conglomerate of clinical symptoms, signs, and laboratory alterations resulting from self-imposed, accidental, or iatrogenic inhalation, ingestion, or parenteral administration of medicinal or non-medicinal substances.

AMCH is a designated poison control treatment center (recognized by the Regional Emergency Medical Organization, Albany, NY) serving a stable population of approximately 250,000 inhabitants. It includes Albany County and parts of Rensselear and Schenectady counties. At AMCH, radioimmunoassay is used to test urine for barbiturates, cocaine, opiates, amphetamines, benzodiazepines, and phenothiazines.[10] A separate assay screens for methadone.[11] The serum is examined by gas chromatography to identify charcoal absorbable drugs such as sedatives-hypnotics, anti-convulsants, theophylline, acetaminophen, and antidepressants.[12] Serum levels of other substances such as aspirin, lithium, digitalis, and alcohol-glycol preparations are also routinely measured.[13]

The poisoning diagnosis obtained from charts reviewed in 1978 and 1988 and their statistical significance are listed in Table I. The population demographics from 1978 and 1988, the mortality rates, and the number of poisoning necessitating

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TABLE I. Most Frequent Causes of Poisonings Among Patients Admitted to the Albany Medical Center

	N (%) 1978	N (%) 1988	p
Acetaminophen	4 (4)	9 (7)	NS*
Barbiturates	15 (16)	7 (5)	0.0036
Benzodiazepines	7 (7)	11 (9)	NS
Phenothiazines	7 (7)	7 (5)	NS
Theophylline	1 (1)	7 (5)	NS
Anticonvulsants	1 (1)	7 (5)	NS
Cocaine	0 (0)	6 (4)	0.0466
Digitalis	1 (1)	6 (4)	NS
Lead	1 (1)	6 (4)	NS
Lithium	1 (1)	5 (3)	NS
Antineoplastics	0 (0)	5 (3)	0.0762
Miscellaneous	56 (56)	70 (70)	NS
Total	94	146	<0.0001

*NS = not significant.

extracorporeal drug removal (ECDR) procedures are presented in Table II. The statistical comparisons of 1978 and 1988 data are based on the Z test for equality of proportions.[14] The Z test was also used for the comparisons of AMCH with New York State reference data.

RESULTS

In contradistinction to 1978, the 1988 statistics on poisonings showed a 55% increase in the number of hospitalized patients (94 vs 146, $p < 0.0001$), and a concurrent change in the most frequently used poisoning agents (Table I). Although the severity of clinical manifestations observed in both instances was comparable, only eight patients required ECDR procedures, 8.5% (n = 94) 1978 and 5.4% (n = 146) 1988 ($p = 0.358$).

Interestingly, when the 1988 patients were contrasted with those in 1978, the gender difference was eliminated (Table II): the 73% of female patients observed in 1978 dropped to 52% in 1988 ($p = 0.0001$). Of the 94 hospitalized patients in 1978, one with acetaminophen poisoning (1.06%) died, whereas in 1988 four fatalities were observed in the 146 hospital admissions (2.74%, $p = 0.375$). A similar number of poisonings were considered intentional or accidental in 1988 (72 and 74, respectively), with all four deaths occurring in the latter group. This series does not include post-mortem data on patients who did not make it to the hospital alive. In 1988, only six patients required hospitalization out of 146 treated in the AMCH Emergency Department. This study showed an unexpectedly low morbidity/mortality in patients who were admitted to the hospital with "street drug" poisonings.

Age as a risk factor in poisonings is shown in Table III. Whereas only one death was recorded in patients under 65 years of age, 18.75% of patients 65 years or older died ($p = 0.0001$). It is of general interest to note that of seven cases of theophylline poisoning, two patients died; they had a mean age of 78 years and a mean blood level of 31.1 mg/dL. The patients who survived in spite of a mean blood level of 53.7 mg/dL had a mean age of 21 years.

TABLE II. Demographics and Extracorporeal Drug Removal Procedures (ECDR) in Patients with Poisonings Admitted to the Albany Medical Center

	1978	1988	p
Patient Population			
Emergency dept visits	628	588	
Hospitalizations	94	146	0.0001
Female (%)	69 (73)	76 (52)	0.001
Male (%)	25 (27)	70 (48)	
Age (years)	34.1	35.9	
Mortality (%)	1 (1.06)	4 (2.7)	0.375
ECDR (%)	8 (8.5)	8 (5.4)	0.358

TABLE III. Deaths According to Age in 1988 (All Poisonings, with Special Reference to Theophylline)

Age (years)	Deaths (N/total)	p	Theophylline (N/total)	p
≤ 65	1/130	<0.0001	0/4	0.0534
>65	3/16		2/3	
Total	4/146		2/7	

DISCUSSION

The increase in the number of hospitalized poisoned patients in 1988 (n = 146) compared with 1978 (n = 94) patients out of 628 Emergency Department visits is considerable. According to the US Census Bureau, the population served by AMCH did not change significantly over this period.[15] Changed in societal use of drugs may have contributed to the larger number of reported poisonings in the 1988 series, in addition to the routine improved methodology to screen, confirm, and monitor substances such as theophylline, lithium, and digoxin.

Treatment methods changed significantly from 1978 to 1988. For example, digoxin antibodies and *N*-acetyl cysteine have been introduced to control digoxin and acetaminophen poisonings, respectively, in addition to the more targeted use of ECDR procedures.

The reason for the change in gender proportions between 1988 (52% female) and 1978 (73% female, $p = 0.0001$) remains unclear. This trend also reported by McMurray et al in 1987[16] may reflect change in societal attitudes and habits and deserves further scrutiny.

In sharp contrast with a national study[9] that showed street drugs as the third leading cause of poisoning fatalities and the second highest fatality risk category, this Albany series showed an apparent low morbidity and no mortality from such drugs. This disparity may be explained by the report of Chafee-Bahamon,[17] which noted that up to 80% of poisoned patients may succumb before arrival at a medical facility. Similarly, the national study conducted in 1988 showed that 28.6% of all drug fatalities had had a cardiorespiratory arrest[9] before reaching the hospital. As the study reported on here deals only with hospitalized poisoned patients; street drug fatalities outside of this setting are not addressed. Of the seven digoxin poisonings observed in this series, no one died, perhaps reflecting earlier diagnosis and therapy.[18,19] It is noteworthy that in spite of an increased number of acetaminophen poisonings recorded in 1988, no fatalities were observed. It is likely this may reflect the almost universal use of *N*-acetyl cysteine in recent years.[20–24]

An almost equal number of intentional and accidental poisonings were documented in 1988. All four deaths that occurred in the latter group are consistent with a high percentage of accidental poisonings in elderly individuals who, in general, have a greater risk of dying.[25–27] In the 1988, AMCH series of patients, the elderly, the accidentally poisoned, and those with theophylline poisoning had higher mortality rates. This influence of aging on mortality caused by poisoning was alluded to by Klein-Schwartz et al[26] and highlighted by Anderson et al[28] among cases of salicylate intoxication; the younger patients with higher salicylate levels had a mortality rate of 2%, compared with a 25% mortality in elderly patients.[29,30] Brancata et al[3] concluded that the greater the age (>65 years) the higher the mortality rate of drug poisonings. Two of three patients older than 65 years died, whereas younger individuals with higher theophylline levels survived. However, there is no evidence that theophylline was the responsible agent causing the demise of these patients. This may have relevance to our clinical observations, since national data for 1988 showed that theophylline preparations were the eighth most common cause of lethal drug poisoning accounting for 26 (4.7%) deaths.[9]

It appears that the aged are at much higher risk of dying of accidental theophylline poisoning. Theophylline poisoning, as observed in the 1988 Albany series, may reflect recent increments in marketing and sales, particularly of slow-release varieties of this drug.[31,32] The frequently cited narrow therapeutic index of theophylline has considerably limited its use in the treatment of bronchospastic disease.[31–37] The delayed gastrointestinal absorption of theophylline may result in peak concentrations 12–24 hours after a given therapeutic dose.[38,39] It is beyond the scope of this paper to delve into the toxicity of theophylline; these issues have been thoroughly reviewed elsewhere.[40–47] Routine supportive care, and ECDR using charcoal hemoperfusion, are considered the therapy of choice in these high-risk patients.[46,47]

Acknowledgments. The authors thank Nancy Abrams Sutter and John Meola, PHD, for their support and help.

REFERENCES

1. Litovitz TL, Normann SA, Veltri JC: 1985 annual report of the American Association of Poison Control Centers National Data Collection System. *Am J Emerg Med* 1986; 4:427–458.
2. Centers for Disease Control: Update: Childhood poisoning—United States. *JAMA* 1985; 253:1957.
3. Brancata DJ, Nelson RC: Poisoning mortality in the United States 1980. *Vet Hum Toxicol* 1984; 26:273–275.
4. Clemmesen C: Treatment of narcotic intoxication. Results and principles of the "Scandinavian" method especially concerning stimulation. *Dan Med Bull* 1963; 10:97–99.
5. Etthinger RW, Flord LP: Attempted suicide: Exposure of 500 cases at a general hospital. *Acta Psychiatr Scand Suppl* 1955; 103:1–45.
6. Arieff AJ, Friedman E: Coma following non-narcotic drug overdosage: Management of 208 adult patients. *Am J Med Sci* 1973; 266:405–426.
7. Litovitz TL, Schmitz BF, Matyunas N, et al: The 1987 annual report of the American Association of Poison Control Centers National Data Collection System. *Am J Emerg Med* 1988; 6:479–515.
8. Schaeffer JH, Lavengood SJ: 1980 survey of poison control centers. *Natl Poison Control Cent Bull* 1982; 26:2–210.
9. Litovitz TL, Schmitz BF, Holm KC: 1988 annual report of the American Assocation of Poison Control Central Centers National Data Collection System. *Am J Emerg Med* 1989; 7:495–545.
10. Baselt RC: Urine drug screening by immunoassay: Interpretation of results, in Baselt RC (ed): *Advances in Analytical Toxicology*, Vol 1, Foster City, CA, Biomedical Publications, 1984 pp 81–123.
11. Meola J, Vanko M: Use of charcoal to concentrate drugs from urine before drug analysis. *Clin Chem* 1974; 20:184–187.
12. Meola J, Vanko M: Serum drug screen using the double detector gas chromatograph. *Clin Chem* 1972; 18:713.
13. Sunshine I: Methodology for analytical toxicology. Boca Raton FL, CRC Press, Inc, 1975 pp 393.
14. Daniel WW: *Biostatistics: A foundation for Analysis in the Health Sciences*, 4th ed, New York, Wiley 1987.
15. US Census Bureau, 1990.
16. McMurray JS, Northridge DB, Abernathy V, et al: Trends in analgesic self-poisoning in West Fife. *Quart J Med* 1987; 65:835–843.
17. Chafee-Bahamon C: Epidemiology of serious poisonings. *Clin Toxicol Rev* 1983; 5:1–2.
18. Stolshek BS, Osterhout SK, Dunham G: The role of digoxin-specific antibodies in the treatment of digitalis poisoning. *Med Toxicol* 1988; 3:167–171.
19. Smith TW, Haver E, Yeatman L, et al: Reversal of advanced digoxin intoxication with Fab fragments of digoxin-specific antibodies. *J Am Coll Cardio* 1985; Suppl:118A–123A.
20. Smilkstein MJ, Knapp GL, Kulig KM, et al: Efficacy of oral N-acetylcysteine in the treatment of acetaminophen overdose. *N Eng J Med* 1988; 319: 1557–1562.
21. Prescott LF: Paracetamol overdosage: Pharmacologic considerations and clinical management. *Drugs* 1983; 25:290–314.
22. Mitchell JR: Acetaminophen toxicity. [editorial] *N Engl J Med* 1988; 319: 1601–1603.
23. Read RB, Tredger JM, Williams R: Analysis of factors responsible for continuing mortality after paracetamol overdose. *Hum Toxicol* 1986; 5:201–206.
24. Flanagan RJ: The role of acetylcysteine in clinical toxicology. *Med Toxicol* 1987; 2:93–104.
25. Garriott JC, DiMaio VM, Petty CS: Death by poisonings. A ten-year survey of Dallas County. *J Forensic Sci* 1982; 27:868–879.
26. Klein-Schwartz W, Oderda GM, Booze L: Poisoning in the elderly. *J Am Geriatr Soc* 1983; 31:195–199.
27. Accident Facts. National Safety Council, Chicago, IL, 1980.
28. Anderson RJ, Potts DE, Gabon PA, et al: Unrecognized adult salicylate intoxication. *Ann Intern Med* 1976; 85:745–748.
29. Hrnicek G, Skelton J, Miller WC: Pulmonary edema and salicylate intoxication. *JAMA* 1974; 230:866–867.
30. Hormaechea E, Carlson RW, Rocove H, et al: Hypovolemia, pulmonary edema and protein changes in severe salicylate poisoning. *Am J Med* 1979; 66: 1046–1050.
31. Bukowsky M, Nakatsu K, Munt PW: Theophylline reassessed. *Ann Intern Med* 1984; 101:63–73.
32. Sessler CN: Theophylline toxicity: Clinical features of 116 consecutive cases. *Am J Med* 1990; 88:567–576.
33. Hensdeles L, Weinberger M: Theophylline—a "state of the art" review. *Pharmacotherapy* 1983; 3:2–44.
34. Jenne JW: Theophylline use in asthma: Some current issues. *Clin Chest Med* 1984; 5:645–658.
35. Marks MB: Theophylline primary or tertiary drug? A brief review. *Ann Allergy* 1987; 59:85–87.
36. Hiu NS: The use of theophylline in "irreversible" chronic obstructive pulmonary disease: An update. *Arch Intern Med* 1988; 148:2579–2584.
37. Rogers RM, Owens GR, Pennock BE: The pendulum swings again toward a rational use of theophylline. *Chest* 1985; 87:280–282.
38. Meustrand T, Svedmyr N, Fagerstrom PO: Absorption of theophylline from conventional and sustained released tablets. *Eur J Respir Dis* 1980; 61:54–61.
39. Coaser BC, Youngs C, Baughman RP: Prolonged toxicity following massive ingestion of sustained release theophylline preparation. *Chest* 1985; 88: 749–750.
40. Hendeles L, Massangari M, Weinberger M: Update on pharmacodynamics and pharmacokinetics of theophylline. *Chest* 1985; 88:103S–111S.
41. VanKesteren RG, VanDijk A, Klein SW, et al: Massive theophylline intoxication: Effects of charcoal hemoperfusion on plasma erythrocyte and theophylline concentrations. *Hum Toxicol* 1985; 4:127–134.
42. Ogilvie RI: Clinical pharmacokinetics of theophylline. *Clin Pharmacokinet* 1978; 3:267–293.
43. Barnhart JL, Combes B: Effect of theophylline on hepatic excretory function. *Am J Physiol* 1974; 227:194–199.
44. Baself RC, Albertson TE: Markedly prolonged theophylline half-life in liver failure. *J Analyt Toxicol* 1982; 6:62–63.
45. Campbell MA, Plachetka JR, Jackson JE, et al: Cimetidine decreases theophylline clearance. *Ann Intern Med* 1981; 95:68–69.
46. Conrad KA, Nyman DW: Effects of metoprolol and propranolol on theophylline elimination. *Clin Pharmacol Ther* 1980; 28:463–467.
47. Jacobs MH, Senior RM, Kessler G: Clinical experience with theophylline—relationships between dosage, serum concentration, and toxicity. *JAMA* 1976; 235:1983–1986.

Adverse cutaneous reactions from cardiovascular drug therapy

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The past 20 years have brought remarkable advances in medical therapeutics, particularly in the area of cardiovascular pharmacology. Because of the high prevalence of coronary vascular disease and hypertension in the United States, there are millions of patients currently receiving drug therapy for these conditions. With the widespread use of such agents, reports of undesirable cutaneous drug reactions have increased enormously. Some of the major skin abnormalities described and the cardiovascular drugs causing them are listed in Table I. A need exists for a compendium of reported side effects of cardiovascular drugs.

Undesirable cutaneous reactions to drugs may occur early on or late in the course of treatment. Symptoms may regress with continuation of the drug but frequently require discontinuation for resolution. The ingestion of drugs may also aggravate existing dermatologic conditions. Cutaneous manifestations of drug reactions vary widely in severity. Generally, they are mild and transient; rarely, they can lead to severe systemic illness that can become life threatening and may, on occasion, be fatal. This is particularly true for toxic epidermal necrolysis, anaphylactic reactions, erythema multiforme, bullous eruptions, exfoliative dermatitis, severe thrombocytopenia, and angioneurotic edema. Adverse reactions may be caused not only by the active agent, but also by additives that may not be listed on the package or insert. Fortunately, reactions to these additives are extremely rare compared with reactions from the active agents.

The exact incidence of dermatologic side effects from cardiovascular drugs has been difficult to estimate because of sporadic reporting. In this review, the cutaneous side effects will be discussed according to drug class and incidences described.

β-Adrenergic Blockers

Practolol. Many of the original reports describing side effects from β-adrenergic blockers mention practolol, a B_1-selective agent. The initial studies with this drug revealed an extremely low incidence of adverse reactions. However, subsequent case series found this not to be accurate.[1] One study reported a 12% incidence of skin reactions.[1] The most commonly reported rash appeared to be of the psoriasiform type, which started with scaling and thickening of the skin of the palms and soles, associated with psoriasis-like plaques over the knees and elbows and other bony prominences. Typically, it also spread to other areas of the limbs and trunk as pruritic erythematous macules with marginated scaling. In spite of the psoriasiform appearance of the eruption, the histology was more suggestive of lichen planus.[1] Typical histologic findings included atrophy of the epidermis with the production of large numbers of colloid bodies and patchy swelling with disruption of the basement membrane.

Felix et al[2] described 21 patients with practolol-induced rashes, including three who had ocular reactions as well. This entity became known as the "oculomucocutaneous syndrome"[3] comprising ketatoconjunctivitis sicca, conjunctival scarring, nasal and mucosal ulcerations, and the aforementioned rashes. Not infrequently, the ocular damage was irreversible.

Practolol has also been implicated as a cause of drug-induced systemic lupus erythematosus (SLE), manifested by arthralgias, rash, a raised erythrocyte sedimentation rate, and positive tests for lupus erythematosus cells and antinuclear antibody.[4] The rash described is erythematous, involving the trunk, arms, and legs, particularly sun-exposed surfaces.

Propranolol. Propranolol is a non-selective β-antagonist, which has been in wide use for many years. Experience with it has been very favorable, and in contrast to practolol, the incidence of reported skin reactions is rather low, observed in only six of approximately 2,000 patients in one series.[5] The most common dermatologic reaction seems to be a psoriasiform eruption, closely resembling that seen with practolol. Erythematous, scaly, nummular lesions on the limbs and trunk are the usual appearance, and they do not respond to conventional psoriatic therapy.[6] Skin biopsies of these lesions, in general, are consistent with a lichenoid type of drug reaction and lack the usual characteristics of psoriasis.[7] Rare reports do exist describing true plaque-like psoriasis and pustular psoriasis with propranolol.[8] However, more commonly, the drug exacerbates preexisting psoriasis.[8]

The most common adverse cutaneous effect of this drug is probably a nonspecific erythematous or maculopapular rash.[9] A case of generalized giant urticaria associated with pruritus has also been described. Other cutaneous reactions with propranolol include erythema multiforme[10] with the Stevens-Johnson syndrome,[11] exfoliative dermatitis, lichen planus, hyperkeratosis of the palms and soles, nail

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TABLE I. Dermatologic Reactions Observed with Various Cardiovascular Drugs

Macular/Maculopapular	
Amiodarone	Methyldopa
Aspirin	Minoxidil
Captopril (>5%)	Nifedipine
Clonidine	Prazosin
Diazoxide	Procainamide
Digoxin	Propranolol
Dilantin (>5%)	Quinidine
Diltiazem	Tocainide
Enalapril	Triamterene
Hydralazine	Verapamil
Hydrochlorothiazide (HCTZ)	Warfarin
Pruritus	
Amiodarone	
Aspirin	Labetalol
Captopril	Lovastatin
Clonidine	Nicotinic acid
Diazoxide	Nifedipine
Digoxin	Prazosin
Diltiazem	Procainamide
Dobutamine	Propranolol
Furosemide	Quinidine
HCTZ	Tocainide
Lichenoid	
Aspirin	Methyldopa
Captopril	Nifedipine
Diazoxide	Practolol
Dilantin	Prazosin
Diltiazem	Propranolol
Furosemide	Quinidine
HCTZ	Spironolactone
Labetalol	Triamterene
Eczematous	
Aspirin	Metoprolol
Captopril	Methyldopa
Clonidine	Nitroglycerin (NTG)
Digoxin	Practolol
Furosemide	Propranolol
Heparin	Quinidine
Hydralazine	Spironolactone
HCTZ	Warfarin
Bullous/Vesicular	
Acetazolamide	Heparin
Amiodarone	HCTZ
Aspirin	Labetalol
Clonidine	Minoxidil
Digoxin	Propranolol
Dilantin	Quinidine
Furosemide	Warfarin
Urticaria	
Acebutolol	Methyldopa
Amiodarone	Nifedipine
Aspirin	Procainamide
Captopril	Propranolol
Digoxin	Quinidine
Dilantin	Tocainide
Diltiazem	Triamterene
Heparin	Verapamil
Hydralazine	Warfarin
Lovastatin	
Vasculitis	
Acebutolol	Furosemide
Amiodarone	Hydralazine
Captopril	HCTZ
Chlorthalidone	Nifedipine
Dilantin	Procainamide
Diltiazem	Propranolol
Ethacrynic acid	Quinidine
Angioneurotic edema	
Aspirin	Heparin
Captopril	Hydralazine
Clonidine	Lovastatin
Digoxin	Procainamide
Dilantin	Verapamil
Diltiazem	Warfarin
Enalapril	
Erythema multiforme	
Acetazolamide	Enalapril
Aspirin	Furosemide
Atropine	HCTZ
Bumetanide	Methyldopa
Captopril	Propranolol
Dilantin	Quinidine
Diltiazem	Tocainide
Dipyramidole	
Stevens-Johnson syndrome	
Dilantin	Propranolol
Diltiazem	Tocainide
Furosemide	Verapamil
Minoxidil	
Drug-induced systemic lupus erythematosus	
Acebutolol	Methyldopa
Chlorthalidone	Pindolol
Clofibrate	Practolol
Clonidine	Procainamide
Dilantin	Propafenone
Furosemide	Propranolol
Hydralazine	Quinidine
HCTZ	Reserpine
Labetalol	Triamterene
Lovastatin	
Fixed drug eruption	
Aspirin	Hydralazine
Digoxin	Nicotinic acid
Dilantin	Nifedipine
Epinephrine	Quinidine
Photosensitivity	
Amiodarone	Methyldopa
Chlorthalidone	Nifedipine
Diltiazem	Quinidine
HCTZ	Triamterene
Psoriasis	
Amiodarone	Pindolol
Aspirin	Practolol
Clonidine	Propranolol
Digoxin	Quinidine
Metoprolol	
Exfoliative dermatitis	
Amiodarone	Nifedipine
Aspirin	Practolol
Captopril	Propranolol
Dilantin	Quinidine
Diltiazem	Tocainide
Hydralazine	
Toxic epidermal necrolysis	
Acetazolamide	Diltiazem
Amiodarone	Propranolol
Dilantin	

changes,[7] generalized pruritus,[12] and toxic epidermal necrolysis.[13] Rare reports also exist documenting propranolol-induced lupus erythematosis syndrome. Harrison et al[14] described a patient with myalgias, fever, arthralgias, pleuritic chest pain, and a skin rash. This syndrome was accompanied by a positive lupus erythematosis cell preparation and an elevated erythrocyte sedimentation rate and was confirmed by rechallenge of the drug.

Propranolol is also included in the list of drugs that can cause pemphigus.[15] Drug-induced pemphigus manifests itself as pemphigus foliaceous or erythematosis, presenting with blister formation, desquamation, and crusting. Peripheral skin necrosis is another complication reported with use of propranolol. Multiple painful areas of skin necrosis on the toes and soles of both feet have been reported in patients whose peripheral pulses remained intact.[16]

Propranolol-induced alopecia is another well-described phenomenon.[17] A typical presentation is patchy hair loss of the scalp, chest, and arms, progressing if the drug is continued. The underlying skin remains normal. Hair growth returns with discontinuation of the drug.

Labetalol. The most frequently described cutaneous reaction to labetalol, an α-β-adrenergic blocker, is a lichenoid eruption, presents as confluent erythema and scaling typically on the face, scalp, neck, or torso with associated pruritus.[18] Bullous lichen planus has also been described, consisting of reddish purple macules and papules, with some lesions being bullous or ulcerated.[19] The mechanism of this lichenoid reaction is thought to be immune related of the cell-mediated type rather than the antibody type. Two case reports document a lupus-like syndrome associated with labetalol, presenting with a vasculitic edema.[20,21]

Metoprolol. Metoprolol is a β_1-selective antagonist, to cause a psoriasiform and an eczematous rash.[22] After oral provocation with metoprolol in one patient, not only did the lesions reappear, but lesions similar to that in pustular psoriasis appeared on the soles. Similar to propranolol, metoprolol can also exacerbate pre-existing psoriasis and can cause diffuse alopecia.[23] This drug has also been reported to cause multiple areas of skin necrosis on the soles of the feet, similar to that described with propranolol.[16]

Pindolol. Pindolol, a β-blocker with partial agonist activity, can cause a flare-up of an existing but latent psoriasis, as can most other β-blockers. Pindolol has been known to cause hyperpigmentation,[24] and a drug-induced SLE syndrome also has been described.[25]

Acebutolol. Acebutolol, a long-acting β-antagonist, has been shown to cause a generalized urticarial rash that is associated with arthralgias and fever.[26] A skin biopsy in a patient revealed changes consistent with a leukoclastic vasculitis. Prick testing with a 5% aqueous solution of acebutolol was positive in the patient and negative in ten controls. This drug has also been documented as causing a lupus-like syndrome.[27]

Nadolol. Nadolol is another β-blocker documented to cause diffuse alopecia.[23]

Atenolol. Atenolol, like propranolol and metoprolol, has caused the adverse reaction of skin necrosis. In one case, an elderly man with painful feet was found to have necrotic patches on the toes and sole of his right foot.[16]

Calcium-Channel Blockers

Diltiazem. Cutaneous reactions to this drug occur rather infrequently, being reported in less than 1% of patients treated.[28] The most commonly reported skin reactions are maculopapular eruptions, photosensitivity, pruritus, petechiae, and urticaria. These reactions are minor, reversible, and require nothing more than merely stopping the drug. Rarer cases of photosensitive lichen planus, toxic epidermal necrolysis, and widespread cutaneous vasculitis have also been reported.[29]

Another rare adverse dermatologic side effect observed with diltiazem is exfoliative dermatitis.[30] This is a more common reaction in patients with a history of psoriasis. Another exanthem shown to be caused by diltiazem is a pustular drug eruption.[31] Skin biopsy reveals subcorneal neutrophilic pustules associated with a perivascular lymphocytic infiltrate in the superficial and mid-dermis. Another severe reaction, including the Stevens-Johnson syndrome,[32] is erythema multiforme. An interesting dermatologic side effect reported with the drug is severe periorbital angioedema, associated with significant edema of the upper lip.[33] Described with diltiazem, also, is a case of vasculitic leg ulcers in a male patient with hypertension and angina. A nonpruritic, palpable, purpuric rash developed symmetrically on the legs, evolving into erosions and multiple painful, punched-out ulcers, necessitating admission to the hospital.[34]

Nifedipine. Nifedipine is a widely used antianginal and antihypertensive drug with potent vasodilatory action. The majority of reported side effects with this drug are due to vasodilation. Cutaneous reactions, although, are rare. Of these, the most common cutaneous reactions are maculopapular eruptions, generalized pruritus, and urticaria.[32] These tend to be mild and rapidly reversible with discontinuation of the drug. Photosensitivity reactions have been reported, presenting as severe blistering eruptions after sun exposure.[35] Erythromyalgia has been described, presenting as intense red discoloration, elevated skin temperature, and burning pain involving most commonly the hands, feet, or lower legs.[36] Edema of the legs, associated with erythema, has also been described.[37]

Exfoliative dermatitis was discovered in a 63-year-old patient two months after starting nifedipine.[38] The patient was rechallenged with nifedipine after resolution of the dermatitis, and exfoliative erythroderma developed associated with diffuse alopecia. Described with nifedipine, also, is bullous fixed drug eruption.[39] This manifested as circumscribed bullous eruptions that occurred at the same sites on repeated exposure to the drug. Finally, leukocytoclastic vasculitis associated with nifedipine use has been reported.[40]

Verapamil. Little documentation of cutaneous reactions to verapamil exists. One report mentions the following adverse reactions: alopecia, maculopapular eruptions, ecchymoses, purpuric vasculitic lesions, urticaria, and hyperkeratoses.[41] A case of angioedema associated with verapamil has been described in which a pruritic periorbital eruption started two weeks after medication was begun.[27] A transient flush reaction, with redness of the face, chest, or back, has also been described with verapamil, and with nifedipine.[27] Finally, verapamil can cause the Stevens-Johnson syndrome.[42]

Angiotensin-converting Enzyme Inhibitors

Captopril. Captopril, a drug used for hypertension and congestive heart failure, is commonly associated with adverse side effects. Cutaneous reactions, which are the most common type, occur in about 12% of treated patients.[43] Pruritus and maculopapular eruptions are the two most frequent cutaneous reactions seen and usually resolve on stopping or reducing the drug dose.[44] Aphthous ulcers of the mouth occur frequently. Mild toxic erythema is another known complication of captopril therapy.[45] Several cases of lichen planus have occurred after the initiation of captopril, usually about six months into therapy.[46] This eruption starts with widespread erythematous scaling, mainly on the trunk, evolving into flat-topped papules with Wickham's striae. Captopril can also induce photosensitivity reactions in some patients.[27]

Two of the more interesting eruptions described with captopril are a pityriasis-like eruption[47] and pemphigus.[48] The former eruption was preceded by pruritus and contained scaly macules on the trunk, with the center of each macule darker than the surrounding uninvolved skin, and collarette of scales at the periphery. The long axis of each lesion was parallel to Langer's skin lines, giving the characteristic "fir tree" distribution. Pemphigus induced by captopril may present as symmetrical erythematous plaques, which are sore and can rapidly lose their surface, producing oozing and crusting. In the center of some lesions, flaccid bullae or erosions may be seen. A skin biopsy showed intraepithelial bullae containing a few acantholytic cells. On direct immunofluorescence, upper epidermal intercellular immunoglobulin-G (IgG) and lower epidermal intercellular C3 were detected. A case of lichen planus pemphigoides with pemphigus-like features has also been described.[49] In this patient, a bullous lichenoid eruption developed that on skin biopsy was not only consistent with lichen planus pemphigoides, but also possessed some features of pemphigus. Other reported cutaneous reactions with captopril include hypersensitivity vasculitis,[49] urticaria,[50] alopecia,[51] and onycholysis.[52] An urticarial eruption with eczematous features leading to erythroderma has been described in four patients.[43] A patient has also been described with a distinctive annular, erythematous, papular eruption on sun-exposed areas that promptly resolved on withdrawal of the drug and recurred with rechallenge.[53] Erythema multiforme is another dermatological reaction observed with captopril therapy.[27]

Two potentially serious cutaneous reactions to captopril that have been reported are exfoliative dermatitis[54] and angioneurotic edema.[55] The angioedema is a well demarcated, non-pitting edema that occurs with large erythematous areas in skin and subcutaneous tissues. It commonly involves the face, lips, and tongue, but may include the hands, feet, genitalia, and mucous membranes. In approximately 20% of instances, it is associated with respiratory symptoms, and rarely can cause respiratory distress caused by laryngospasm or shock. The mechanism of this reaction is believed to be secondary to elevated bradykinin levels from impaired kinin inactivation with angiotensin converting enzyme inhibition.

Enalapril. Unlike captopril, enalapril is rarely associated with cutaneous reactions. This is also true for patients who have reactions to captopril and are switched to enalapril. A maculopapular skin rash associated with minor oral bleeding and sloughing has been described with enalapril,[56] as has a case of erythema multiforme.[27] Angioedema has been reported with enalapril.[51] The cumulative incidence of angioedema is 0.1%, with the large majority of cases occurring during the first week of therapy. Seven patients were reported on who had angioedema reactions to both enalapril and captopril. Therefore, it appears that this reaction has a common mechanism.

OTHER ANTIARRHYTHMIC AGENTS

Procainamide. The most prominent cutaneous reaction to procainamide is drug-induced lupus erythematosus. The original description of procainamide-induced lupus was reported in 1962.[57] A patient was described with a blotchy pruritic rash over the face, chest, and forearms that had resolved and later recurred on the thighs and abdomen and was associated with intermittent swelling of the upper lip as well as other symptoms typical of drug-induced SLE. The rash was diffuse, papular, scaly, and erythematous, and disappeared two weeks after cessation of the medicine. The usual time interval from initiation of drug therapy to development of symptoms usually ranges from one to nine months. Slow acetylators of the drug appear to be at an increased risk of developing this condition, but it can occur in rapid acetylators as well, although not as quickly. The characteristic features of drug-induced lupus are arthralgias, pleuritic chest pain with roentgenographic evidence of pleuropulmonary disease, and cutaneous lesions, most commonly associated with a positive antinuclear antibody test. Drug nucleoprotein interactions that enhance the autoimmunogenicity of nuclear constituents is the most widely held theory regarding the mechanism of this disease.[58] To account for the presence of diverse autoantibodies observed in drug-induced lupus, pharmacologically distinct agents like procainamide and hydralazine must serve as adjuvants or have some immunostimulatory function in vivo.

The incidence of cutaneous drug eruptions in hospitalized medical patients receiving procainamide is 2%–3%.[59] Procainamide is also known to cause vasculitis, the second most common reaction seen.[60] Other cutaneous reactions reported with the use of procainamide include pruritus, palpable purpura, angioedema, and urticaria.[61] Maculopapular eruptions seem to occur in 1%–5% of persons treated as outpatients. In this same report, several case reports of urticarial vasculitis were described, in which erythematous, edematous papules, and plaques developed, with some hyperpigmented patches distributed over the trunk (Fig 1).[61] This reaction is pruritic, painful, or burning, and often resolves with post-inflammatory hyperpigmentation.

Quinidine. Quinidine is associated with adverse skin reactions more frequently than procainamide. This antiarrhythmic has also been shown to cause drug-induced SLE, although less frequently. Some of the minor and more common eruptions that can be seen include maculopapular

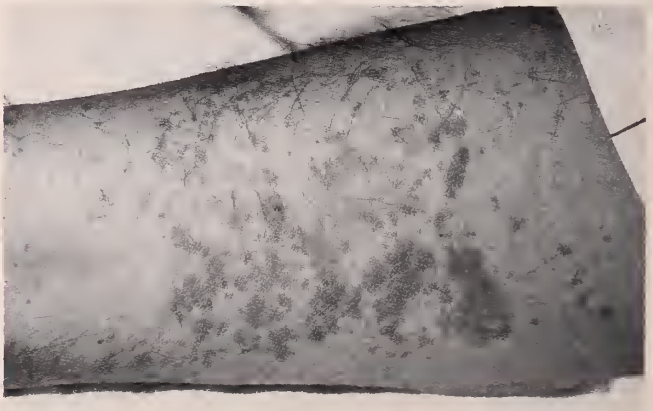

FIGURE 1. Urticarial papules and plaques on the thigh which demonstrate leukocytoclastic vasculitis on skin biopsy due to procainamide.

and scarlatiniform rashes, urticaria, and pruritus.[62] Quinidine has been known to cause thrombocytopenic purpura, which is generally reversible with discontinuation of the drug.[63]

Phototoxic reactions can occur with oral ingestion of the drug. Photodermatitis presenting as a severe eczematous, scaling dermatitis on sun-exposed areas with sharp lines of demarcation can occur, sometimes with fissures, crusting, and serous exudation.[64] This has been confirmed with oral rechallenge and is reproducible with ultraviolet-A radiation. Sun exposure has also caused a photosensitive livedo reticularis, a reticulated, violaceous, pruritic, purpuric eruption that can lead to residual pigmentary changes, usually on the legs.[65] Lichen planus with violaceous flat-topped papules on the dorsa of both hands and forearms or purplish discoloration with a lichenoid rash under both eyes was first reported in 1967.[66] Since then, it has become evident that both lichen planus (Fig 2) and photosensitive lichen planus (Fig 3) are common reaction patterns with quinidine use. Abnormalities of pigmentation are a well-known side effect of antimalarial therapy, and quinidine is structurally related to this class of drugs; therefore, it is not surprising that it has also been associated with pigmentary abnormalities. A patient has been described who had bluish-gray oval patches several centimeters in diameter over the anterior portions of both shins. Noted in addition were blue-gray striations on fingernails and toenails; the nose, ears, and forearms subsequently became similarly discolored.[67]

Like other drugs previously described, quinidine can also exacerbate psoriasis.[68] Other reactions known to occur are fixed drug eruptions and eczematoid eruptions,[69] acneiform and bullous eruptions,[27] erythroderma,[70] and erythema multiforme.[27]

Two serious adverse cutaneous reactions encountered with this drug are exfoliative dermatitis[61] and vasculitis.[63] The vasculitis can progress to hemorrhagic bullae, and immunofluorescence microscopy demonstrates granular deposits of IgM, IgA, and C3 in the dermal blood vessels of these lesions.

Dilantin. Although dilantin is more frequently used as an anticonvulsant than an antiarrhythmic agent, its side effects are worth mentioning in this review. Dilantin produces cutaneous side effects in an estimated 5% of cases.[71] The more frequently observed reactions are morbilliform and scarlatiniform eruptions that are frequently accompanied by fever, maculopapular eruptions, and lichenoid eruptions.[27] Less commonly seen are urticaria and non-thrombocytopenic purpura. Dilantin is also known to cause SLE, usually after prolonged treatment.[72] Two patients have been reported who presented with drug-induced SLE and subsequently developed Stevens-Johnson syndrome. Erythema multiforme appears to be a relatively common reaction pattern encountered with dilantin use.

A distinct life-threatening hypersensitivity reaction is reported that progresses to exfoliative dermatitis.[73] A striking aspect of this reaction is angioedema of the face that may be so severe as to prevent opening of the eyelids (Fig 4). A related report describes a fatal case of exfoliative dermatitis that was associated with severe hepatitis.[74] Another fatal reaction described with this drug is hemorrhagic erythema multiforme in a patient who presented with extensive purpuric eruptions and stupor.[64] The eruptions involved the face, neck, trunk, and extremities, including the palms and soles, and consisted of hemor-

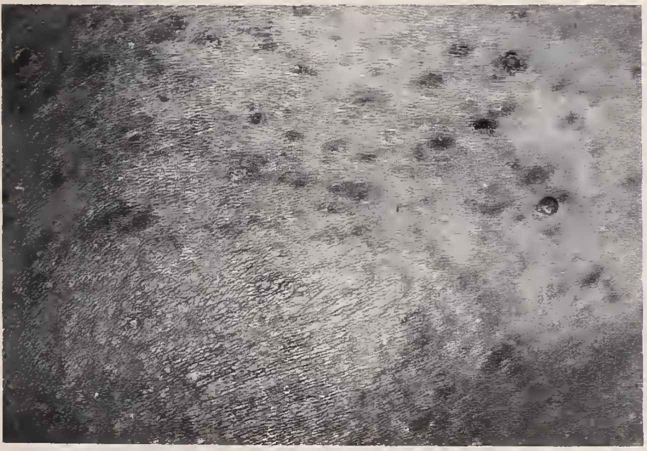
FIGURE 2. Violaceous, purple, and hyperpigmented papules of quinidine-induced lichen planus.

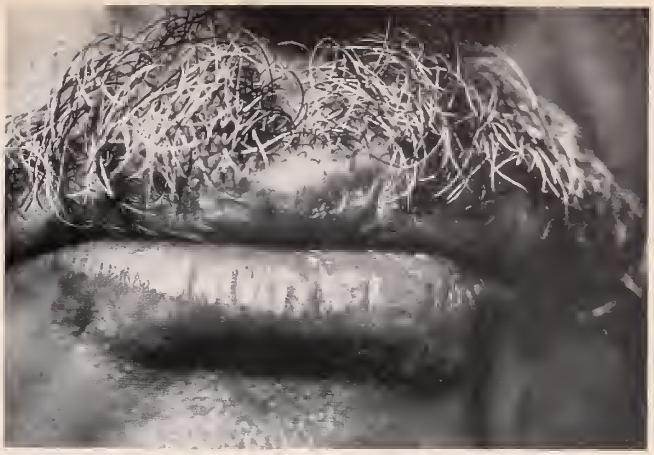
FIGURE 3. Quinidine lichenoid photodermatitis. Glistening violaceous plaques of the sun-exposed portions of the lip with a delicate network of white lines.

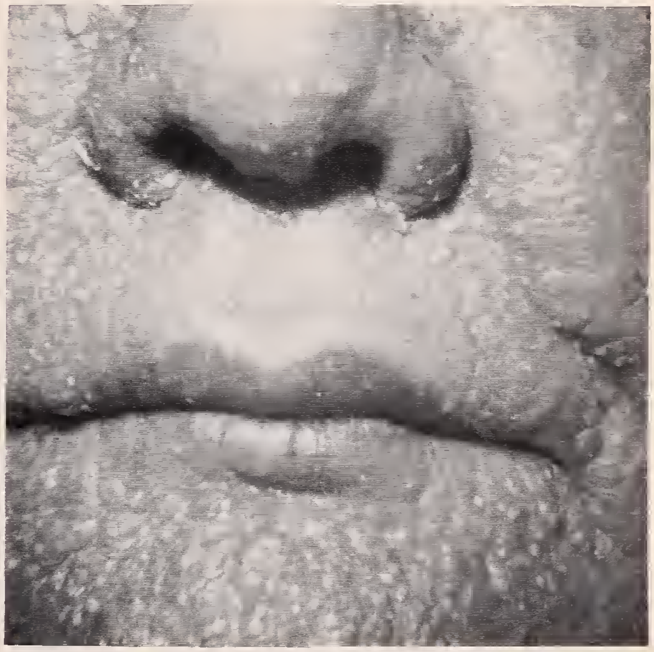
FIGURE 4. Massive edema of the face and perioral region with follicular papules and pustules characteristic of the dilantin hypersensitivity reaction.

rhagic macules and flaccid bullae. The report points out the importance of discontinuing this drug on presentation of any toxic erythema because the erythema can progress quite rapidly, with hemorrhagic tendencies.[64] Toxic epidermal necrolysis, a reaction with a 25% mortality rate, is also associated with dilantin use.[75] This typically presents with a prodrome before the symptoms of malaise, arthralgias, myalgias, headache, and fever.

A last entity to be described has been termed the pseudolymphoma mycosis fungoides syndrome (PMFS), characterized by a triad of fever, lymphadenopathy, and an erythematous macular skin eruption.[76] The skin biopsy characteristically shows infiltration of the dermis with atypical mononuclear cells with clefted, cerebriform nuclei. The overlying epidermis is hyperplastic with focal intraepidermal nests of mononuclear cells characteristic of Pautrier's microabscesses. Reported in addition is mycosis fungoides confirmed by skin biopsy and not associated with the triad of symptoms.[76] A subset of PMFS that is also known to occur with dilantin is localized cutaneous lymphoma.[77] It is felt that these are all subsets of the pseudolymphoma syndrome.

Reports of other reactions include fixed drug eruptions ($<1\%$), bullous eruptions ($<1\%$), cutaneous vasculitis ($<1\%$), spotty skin pigmentation in light-exposed areas during long-term treatment, acneiform eruptions, as well as hypertrichosis and hirsutism.[27]

Tocainide. This antiarrhythmic drug is frequently associated with adverse effects, and dermatologic reactions comprise 28% of them.[78] The mild ones are urticaria, alopecia, pruritus, and flushing. In 10% of these, the reaction was either Stevens-Johnson syndrome, erythema multiforme, or exfoliative dermatitis. Fatalities from skin reactions were reported at a rate of 0.9/100,000 prescriptions in 1985. A patient has been described with a generalized maculopapular lupoid eruption associated with tocainide use.[79] This eruption began on the abdomen, chest, and stomach, progressed with centrifugal spread of the lesions, and was associated with hemorrhage from the lips and gingivae in spite of cessation of the drug. The lesions were burgandy colored, showed some weeping and denudation, became vesicular later on, and progressed to massive necrolysis.

Amiodarone. Amiodarone is another antiarrhythmic drug frequently associated with skin reactions. By far, the most common reaction is photosensitivity, occurring in 57% of patients treated.[80] There is a wide spectrum to this reaction, ranging from increased propensity to suntan during the summer, to intense burning with erythema and swelling. Prolonged treatment is also occasionally associated with marked photosensitivity to light-exposed areas followed by a blue-gray pigmentation.[81] Skin biopsy showed that lipofuscin granules deposit in both affected and unaffected areas of skin, although they are much more prominent in affected areas. It is felt that this reaction is dose and duration dependent. Although the photosensitivity is reversible, the pigmentation is usually not. Described also is a fine erythematous, maculopapular rash with a truncal distribution that is pruritic and occurs within the first two weeks of therapy.[81] Of interest is that this rash resolved without cessation of therapy. Urticaria and pruritus are each reported to occur with a frequency of 1%–5% of those started on this drug. Psoriasis and erythema nodosum are two other rare complications of therapy.[82]

As for some of the more serious reactions to amiodarone, exfoliative dermatitis has been documented to occur after five weeks of treatment. A fatal case of toxic epidermal necrolysis was reported in which a patient subsequently died of sepsis and cardiac failure.[83] Another report relates a case of dose-dependent vasculitis that recurred several times, but only when drug levels were >2.6 mg/L.[84] A last side effect reported with the drug is proliferative papillomatosis (ioderma).[85] This reaction, presented as a necrotic tumor in the left inguinal region, was thought to be malignant and was surgically removed. However, the skin biopsy showed only nonspecific granulation tissue, and two months later new tumors appeared. Originally, they occurred at the site of excision, then in the right inguinal region, and slowly spread to the legs, developing small sinuses that discharged serous and purulent material. These lesions regressed within two weeks of discontinuation of the drug with some residual hyperpigmentation.

Propafenone. One reported cutaneous side effect of propafenone has been documented as a drug-induced SLE reaction that reappeared on oral rechallenge with the drug.[86]

CARDIAC GLYCOSIDES

Digitalis. Allergic reactions to digitalis are rare in spite of its widespread use over many years. A review published in 1958 reported urticaria, scarlatiniform eruptions, papular and vesicular lesions, and edema.[87] Included also in this review is a report of a woman who exhibited generalized pruritus and bullous lesions on her legs while taking digitalis leaf. When her therapy was changed to digitoxin, the same lesions occurred, accompanied by multiple, brown-pigmented areas with elevated, erythematous edges. Two case reports of urticaria with angioedema have been described, dating back to 1887 and 1940.[88,89] In these reports, as in most others, the reactions were always elicited with several different digitalis preparations and were reversible with discontinuation of the drug. The latter publication also mentions an eczematous reaction to digitalis.[89] Thrombocytopenia purpura is known to occur in rare instances with various digitalis formulations.[90] Digitalis is also on the list of drugs associated with fixed drug eruptions.[91] Lastly, a case report in 1981 documented a psoriasiform eruption that was induced by digoxin, confirmed on oral rechallenge, and skin biopsy, in a patient with no earlier history of psoriasis.[92]

SYMPATHOMIMETICS

Dobutamine. Few reports of cutaneous reactions can be found with this agent. Original reports describe painful gray discoloration and skin sloughing overlying and surrounding the drug infusion sites; these have been observed both with and without evidence of extravasation of the infusion.[93] Reported also is scalp pruritus that occurred during weekly 48 hour infusions with dobutamine, in the absence of any exanthems of the scalp.[94]

Norepinephrine and Epinephrine. One severe adverse reaction described with norepinephrine use is subcutaneous tissue necrosis and sloughing, often resulting in permanent scarring and rarely amputation.[95] This sloughing has also been noted to occur without direct extravasation of the drug. Intravenous use of epinephrine has also been documented to cause fixed drug eruptions.[27]

DIURETICS

Hydrochlorothiazide. Numerous adverse cutaneous reactions to hydrochlorothiazide (HCTZ) have been described. By far, the most common cutaneous side effect is photosensitivity, originally described in 1959.[96] There is a wide spectrum of photosensitive lesions that can occur: erythema; vesicles; eczema; erythematous, squamous, and papular SLE-like lesions; lichen planus; and a petechial response.[97] What is most commonly encountered is an erythematous reaction of light-exposed areas accompanied by burning or itching. Some patients will improve with the use of sunscreens only, whereas others require cessation of drug therapy before lesions will improve. In rare instances, a state of chronic photosensitivity has persisted long after the drug has been withdrawn (Fig 5).[98] This presents as a scaling erythema and histologically is an eczematous dermatitis. The combination of HCTZ and amiloride, marketed as a separate product, is associated with an extremely high rate of phototoxic eczema, accounting for 50% of all cutaneous reactions to drugs in one Danish survey.[99] In addition to the aforementioned photosensitive eruptions, subacute cutaneous lupus erythematosus has occurred with this drug.[100] This entity is a subset of SLE, in which patients have widespread non-scarring lesions in a photo-distribution pattern. The rash is erythematous and may be macular, maculopapular, or plaque-like, and has an overlying delicate scale and central hypopigmentation.

Nonphotosensitive reactions encountered with thiazides, include macular, morbilliform, petechial, and bullous[27] eruptions, as well as erythema multiforme[97] and erythema annulare centrifugum.[101] Since this diuretic is a sulfonamide, it may produce eczematous eruptions in individuals previously sensitized to other sulfonamide drugs.[62] Both thrombocytopenic purpura[102] and nonthrombocytopenic purpura[103] can occur, usually with symmetrical lesions involving the lower extremities. Pruritus and flushing are two other rare side effects of this diuretic.[103] One serious cutaneous side effect seen is necrotizing vasculitis, which is observed on the lower extremities as erythematous or purpuric lesions.[104]

Furosemide. Cutaneous reactions to this sulfonamide diuretic occur much less frequently than with HCTZ. The following eruptions have developed after treatment with furosemide: eczema, purpura, pruritus, lichenoid reactions, vesiculobullous lesions,[99] pustular lesions, pseudoporphyria, thrombocytopenia purpura, and vasculitis.[27] The last report also documents a case of Stevens-Johnson syndrome. The extent of phototoxic reactions seen with this sulfonamide drug is limited to several reports of bullous hemorrhagic eruptions occurring in light-exposed areas (Fig 6).[105] Erythema multiforme occurred in a 65-year-old man receiving furosemide for congestive heart failure.[106] Finally, bullous pemphigoid has been documented in a 78-year-old woman whose multi-drug treatment included furosemide.[107] Furosemide is another drug that has been associated with drug-induced lupus.[27] This diuretic can also cause an exfoliative dermatitis, another potentially fatal dermatologic reaction.[27]

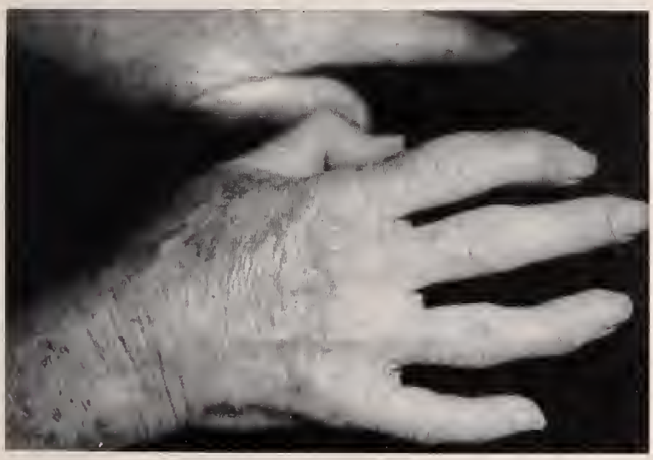
FIGURE 5. Chronic photoallergic reaction to hydrochlorothiazide with erythematous lichenified plaques in the sun-exposed body areas.

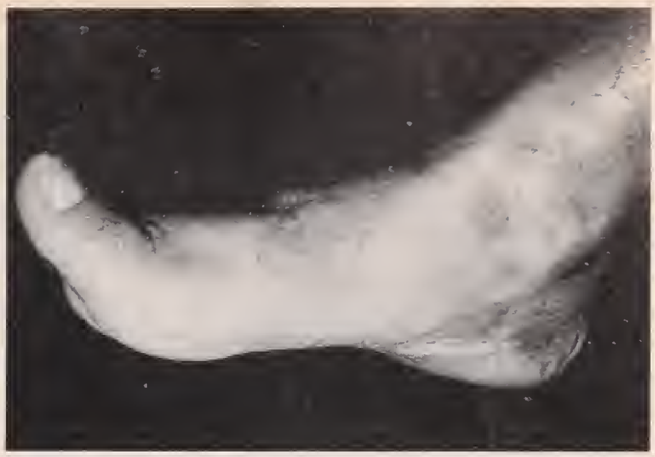
FIGURE 6. Multiple bullous fixed drug eruptions due to furosemide. Skin biopsy excluded bullous pemphigoid, bullous erythema multiforme and pseudoporphyria.

Chlorthalidone. Little information is available on adverse skin reactions to this drug. One report describing vasculitis associated with thiazides also mentions chlorthalidone as a drug that can cause vasculitis.[104] This drug has also been reported to cause photosensitive dermatitis and SLE.[27]

Spironolactone. Spironolactone, a potassium-sparing diuretic, has an extremely low, adverse-reaction profile. Eruptions previously described lichenoid reactions, thrombocytopenic purpura,[27] hirsutism, and eczematous lesions.[99]

Triamterene. Triamterene, another potassium-sparing diuretic, also has an extremely lower side effect profile. It is associated with urticaria, lichenoid reactions, photosensitive dermatitis,[108] and erythematous macular eruptions.[109] Reactions to use of this drug as a sole agent are not well studied, since it is most often used in combination with HCTZ. This combination is one of the most commonly prescribed preparations in the United States. The combination is associated with cutaneous reactions in 0.05% of users and has been shown to cause subacute cutaneous lupus erythematosus.

Acetazolamide. Very few reports are written on side effects to this diuretic. It is known to cause bullous eruptions. Documented, also, is toxic epidermal necrolysis, erythema multiforme, and thrombocytopenic purpura.[27]

Bumetanide. Bumetanide also has a very low side effect profile. Its use has been associated with cases of erythema multiforme.[27]

Ethacrynic Acid. This drug is comparable in potency to furosemide, however, it is not a sulfonamide diuretic, and reported cutaneous reactions are very rare. A Henoch-Schonlein-like eruption has been documented.[110] Necrotic hemorrhagic lesions were observed in another patient without thrombocytopenia with the histology showing vasculitis.[62]

DIRECT VASODILATOR DRUGS

Hydralazine. This vasodilator is frequently associated with side effects, including dermatologic reactions. Most frequently documented is drug-induced lupus. SLE develops in approximately 10% of patients receiving hydralazine, and skin rash in about 25%.[111] The spectrum of skin lesions described in this syndrome include erythema, erythema nodosum, pruritic papular eruptions, purpura, and most commonly the scaling and sharply dermarcated maculopapular rash typical of SLE.[112] Other lesions that have occurred with hydralazine in the setting of positive antinuclear antibody titers are periungual erythema, pyoderma gangrenosum,[112] granuloma annulare, leg ulcers,[113] vasculitis, and purpura.[114] The purpura became confluent with vesiculations, progressing to shallow ulcerations that became necrotic and required skin grafting.

Other cutaneous adverse reactions seen with this vasodilator are flushing,[115] photosensitivity, exfoliative dermatitis, livedo reticularis,[62] eczema, urticaria,[99] fixed drug eruptions,[91] and angioedema.[27] Neutrophilic dermatosis, also known as Sweet's syndrome, has occurred in association with hydralazine therapy,[116] presenting as numerous erythematous, indurated plaques on the neck, shoulders, forearm, and face.

Nitroglycerin. A major route of delivery of this drug is transdermal therefore it is not surprising that contact dermatitis is the most common cutaneous side effect. This dermatitis is usually a pruritic, erythematous, or eczematous eruption that can vesiculate. Fortunately, this is a rare complication of nitroglycerin therapy. Hendricks[117] was one of the first to report contact dermatitis with the actual drug. This was done with patch testing, eliminating the vehicle and additives as potential causes. Commonly, however, contact dermatitis is secondary to the vehicle, usually a lanolin base or the pressure-sensitive adhesive.[118] For this reason, it is advisable to try another preparation when sensitivity to one product is encountered. Another tactic used to limit skin reactions is to rotate the site of patch administration.

Minoxidil. Cutaneous reactions to minoxidil are infrequent. The most common reaction seen is hypertrichosis and hirsutism.[119] Other less-frequent effects include increased pigmentation of the skin, macular and papular lesions, and bullous eruptions.[120] A report also describes a case of Stevens-Johnson syndrome caused by minoxidil treatment that began with pruritic maculopapular lesions that subsequently progressed to large bullae and became desquamative.[120] Another report describes a patient with an erythematous weeping rash and impending exfoliation after three years of treatment, which resolved with drug withdrawal. On oral rechallenge, bullous lesions immediately reappeared.[121]

Diazoxide. Diazoxide is a vasodilator that is rarely used for management of hypertensive emergencies, and it is associated with very few side effects. Those recorded are maculopapular lesions, petechiae,[122] lichenoid reactions, and pruritis in 1%–5% of those treated.[27] Most commonly seen is hypertrichosis.

OTHER ANTIHYPERTENSIVES AGENTS

Methyldopa. Skin reactions with this drug are infrequent. The common eruptions seen with other drugs can also be seen with methyldopa: lichen planus, pruritic papular lesions, urticarial rash,[123] photosensitive eruptions, pruriginous papular eruptions, hyperpigmentation, eczematous dermatitis, and dermatitis in a seborrheic distribution.[124] Church[125] described a variety of eczematous eruptions in four patients: seborrheic, nummular, and discoid. Another patient had four nodular growths on the elbow, forehead, and face after four months of methyldopa therapy, and skin biopsy of the lesions showed a granulomatous infiltrate with multinucleated giant cells containing birefringent crystalline material.[126] Erythema multiforme, thrombocytopenia purpura,[27] generalized pruritus, and ulcerations of the lower extremities[127] have also been attributed to this agent. Methyldopa is another agent that can cause drug-induced SLE.[27]

Clonidine. Skin reactions are infrequent and include maculopapular and eczematous eruptions. Pruritus occurs in more than 5% of those who take clonidine. Pityriasis rosea, a rare drug-induced eruption, is known to occur with clonidine.[47] Cicatricial pemphigoid, a chronic disease of the mucosal surface and skin, characterized by subepidermal blister formation and scarring, is also seen with oral clonidine use.[128] Clonidine is one of the many drugs that cause SLE, with the associated cutaneous reactions seen with this entity.[129] The drug can exacerbate psoriasis.[130] A proposed mechanism of action for this latter reaction is that the inhibition of adenylate cyclase with clonidine causes a fall in intracellular cyclic AMP, which enhances epidermal cell proliferation, leading to psoriatic eruption. Another rare complication associated with clonidine use is angioedema.[27]

Reactions to clonidine are most commonly seen with transdermal delivery of the drug. Skin reactions to this preparation occur in 10%–38% of patients and seem to become more common when treatment is continued for several months.[131] Wiser et al[131] described a case of herpes simplex lesions confirmed with viral cultures that developed at the site of patch placement. During subsequent months, this same patient exhibited hyperpigmentation at the site of other patch placements, necessitating discontinuation of this drug. The spectrum of eruptions more commonly associated with transdermal clonidine use include pruritus, erythema, vesiculation,[132] and in one case, a generalized papular rash that developed after local reactions had been seen.[133]

Prazosin. This oral antihypertensive is associated with macular or maculopapular reactions in 1%–5% of patients, and pruritus in less than 1%.[27] Another cutaneous effect of this antihypertensive is a lichenoid reaction.[27]

Reserpine. Reserpine is associated with a considerable number of side effects that limit its widespread use, but cutaneous reactions are uncommon. It has been implicated in drug-induced lupus, and it can cause peripheral edema.[27]

ANTICOAGULANTS AND ANTIPLATELET DRUGS

Heparin. Heparin, used systemically and subcutaneously, has a low-side effect profile. When used systemically, heparin has been associated with alopecia, which principally involves the scalp, but may also involve the eyebrows, axillary, and pubic hair.[134] Urticaria, with and without anaphylaxis, as well as pruritus, have also been described.[135] Thrombocytopenic purpura is a well recognized reaction to heparin, and there have also been rare case reports of angioedema.[27] A more distressing reaction that occurs with intravenous use of heparin is cutaneous necrosis.[136] This presents as well-circumscribed necrotic, hemorrhagic, bullous lesions. Skin biopsy of these lesions reveals focal hemorrhage within the epidermis and adjacent dermis, and an area of necrosis covered with hemorrhagic crust. Biopsies have failed to show any evidence of vasculitis or fibrin thrombi within vessels, which is common with warfarin necrosis (described below).

Local reactions attributed to subcutaneous heparin administration consist of erythema, edema, mild cutaneous hemorrhage, and induration.[137] There can also be erythematous, indurated plaques that occasionally have eczematous surfaces at the site of injection.[27]

Warfarin. The most dreaded dermatologic complication with warfarin therapy is cutaneous necrosis, first reported in 1965.[138] It occurs three-to-five days after initiation of treatment and is rapid in onset, painful, often extensive in its involvement, and potentially life threatening. It is characterized by sudden, localized, hemorrhagic skin lesions, culminating in hemorrhagic infarcts or gangrenous necrosis. The necrosis occurs most commonly on the skin overlying adipose tissue of the abdomen, thighs, and female breasts (Fig 7). Biopsy of the margins of such lesions before the onset of necrosis shows occlusion of dermal and subcutaneous veins with fibrin thrombi, minimal inflammation, and some extravasation of red blood cells. In severe cases, this condition may require surgical debridement and skin grafting. Most other reactions to this drug are not as serious. Maculopapular, eczematous,[139] pruritic, vesicular, and scaly erythematous eruptions have all been reported.[140] Bork[27] also mentions angioedema and phototoxic reactions as other side effects of warfarin therapy. The first documented reaction to oral warfarin, reported in 1959, was transient urticaria.[141] Two other reactions known to occur, independent of elevated prothrombin time, are purpura and "purple toes" (Fig 8). Feder and Auerbach[142] reported on six patients in whom dark-blue-tinged bilateral purple discoloration of the feet developed particularly on the plantar surfaces and sides of the first two toes. Warfarin, like heparin, has also been associated with alopecia.[143]

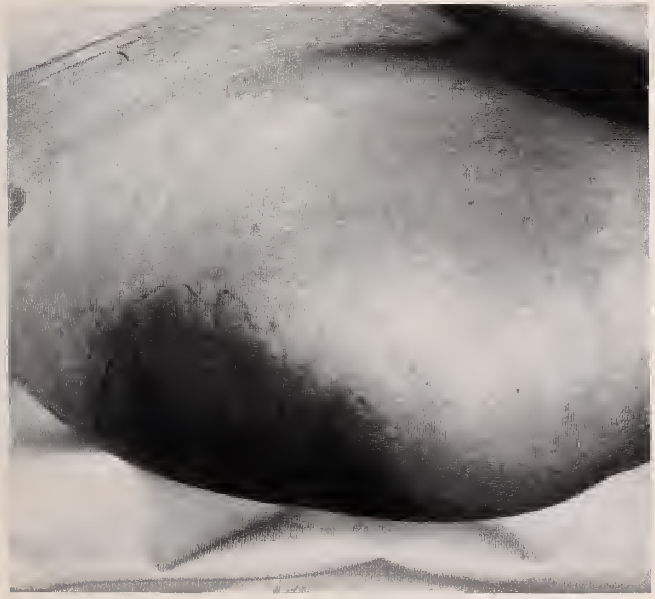

FIGURE 7. Early coumadin necrosis of the buttocks and thigh of a woman.

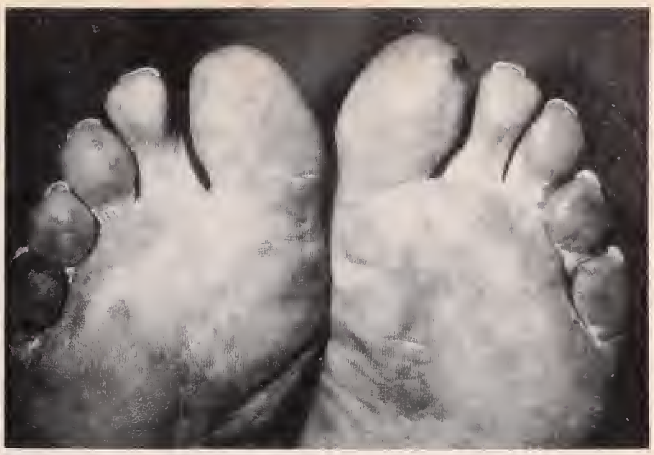

FIGURE 8. Bilateral purple discoloration of the toes and plantar surfaces of the feet due to coumadin. The "purple toes syndrome" appears the same as cholesterol microembolization.

Aspirin. Salicylate preparations are among the most commonly used drugs in the world. Although there are multiple cutaneous reactions associated with their use, the frequency appears to be quite low. The most common eruption is urticaria, which includes both the new onset and exacerbation of existing urticaria.[144] The most likely mechanism of this reaction is that aspirin acts as a histamine liberator. Anaphylaxis, angioedema, and urticaria can occur independently or in any combination. Described, also, are salicylate-induced morbilliform eruptions, flushing of the face, erythema nodosum, erythema multiforme, anaphylactoid purpura, vasculitis, thrombocytopenic purpura, herpes progenitalis, and a bullous eruption of the palms. Skin reactions described by Bork[27] include alopecia, macular or maculopapular eruptions, exfoliative dermatitis, and pruritis. Aspirin can also cause fixed drug eruptions, confirmed by readministration of the drug, toxic erythema,[144] and eczematous dermatitis. Brannen and Forbes[145] studied aspirin sensitivity using the basophil test and found that patients with a positive basophil test with aspirin developed the following reactions when exposed to the drug: lichen planus, dermatitis herpetiformis, pityriasis rosea, and parapsoriasis. Another reported cutaneous reaction is pustular psoriasis in a six-year-old given aspirin for a febrile illness.[146] The lesions spread to the entire body and resolved when aspirin was discontinued.

Dipyridamole. Reactions to this antiplatelet agent are extremely rare. A case of erythema multiforme has been reported.[27]

Lipid-lowering Agents This group of pharmacologic agents has an extremely low incidence of cutaneous adverse reactions.

Nicotinic Acid. The most common side effect seen with this drug is flushing and pruritus. Slow-release preparations are now marketed that have reduced this reaction. It is well accepted that this drug can cause acanthosis-nigricans-like lesions.[147] This presents as dark brown, slightly raised plaques with irregular borders and a smooth velvety surface. The biopsy shows hyperkeratosis, acanthosis, papillomatosis, and increased melanin in the epidermis. This lesion always disappears with discontinuation of the drug. However, since it is usually associated with malignancy, it is also important to rule out internal malignancy as a cause. Other cutaneous side effects include fixed drug eruptions and dry skin.[27]

Clofibrate. Reports of two cutaneous reactions have been made: drug-induced SLE and alopecia.[27]

Lovastatin. Lovastatin is a new drug on the market from the class of drugs known as hepatic hydroxymethyl glutlaryl coenzyme-A reductase inhibitors and has been proven to be an extremely effective lipid-lowering agent. To date, the following adverse cutaneous reactions have been reported with its use: pruritus, angioedema, urticaria, and drug-induced SLE.[148] Rashes are reported to occur in 0.4% of patients using this drug.

CONCLUSIONS

The number of cutaneous reactions observed with cardiovascular drugs are considerable. When prescribing drugs for cardiovascular disease, physicians should always take into consideration the potential adverse reactions of drug treatment. Each drug must have a clear indication, and if not clearly indicated, it should be considered contraindicated.[149]

Acknowledgments. One of the authors wishes to acknowledge the support of Mr. Stuart Troy, JD

REFERENCES

1. Waal-Manning H: Problems with practolol. *Drugs* 1975; 10:336–341.
2. Felix RH, Ive FA, Dahl MGC: Cutaneous and ocular reactions to practolol. *Br Med J* 1974; 4:321–324.
3. Wright P: Untoward effects associated with practolol administration: Oculomucocutaneous syndrome. *Br Med J* 1975; 1:595–598.
4. Raftery EB, Denman AM: Systemic lupus erythematosus syndrome induced by practolol. *Br Med J* 1973; 2:452–455
5. Stephen SA: Unwanted effect of propranolol. *Am J Cardiol* 1966; 18: 463–472.
6. Jensen H, Mikkelsen HI, Wadskov S, et al: Cutaneous reactions to propranolol (Inderal). *Acta Med Scand* 1976; 199:363–367.
7. Cochran RE, Thomson J, McQueen A, et al: Skin reactions associated with propranolol. *Arch Dermatol* 1976; 112:1173–1174.
8. Hu CH, Miller AC, Peppercorn R, et al: Generalized pustular psoriasis provoked by propranolol. *Arch Dermatol* 1985; 121:1326–1327.
9. Seides SF, Ruskin JN, Damato AN: Propranolol-induced urticaria: Successful therapy with tolamolol. *Chest* 1975; 4:496–497.
10. Pimstone B, Joffe B, Pimstone N, et al: Clinical response to long-term propranolol therapy in hyperthyroidism. *S Afr Med J* 1969; 43:1203–1205.
11. Mukul VG: Propranolol-induced Stevens-Johnson Syndrome. *J Assoc Physicians India* 1989; 37:797–798.
12. Humphreys GS, Delvin DG: Ineffectiveness of propranolol in hypertensive Jamaicans. *Br Med J* 1968; 2:601–603.
13. Van Ketel WG, Roesman-Van Waadenoyed Hernkamp A: Een op de ziekte van lyell gelijkende eruptie door propranolol. *Ned Tijdschr Geneeskd* 1977; 121:1475–1476.
14. Harrison T, Sisca TS, Wood WH: Propranolol-induced lupus syndrome? *Postgrad Med* 1976; 59:241–244.
15. Godard W, Lambert D, Gaveanou J, et al: Pemphigus induit apres traitement par association propranolol-meprobamate. *Ann Dermatol Venereol* 1980; 107:1213–1216.
16. Gokal R, Dornan TL, Ledingham JGG: Peripheral skin necrosis complicating beta-blockade. *Br Med J* 1979; 1:721–722.
17. Martin CM, Southwick EG, Maibach HI: Propranolol-induced alopecia. *Am Heart J* 1973; 86:236–237.
18. Staughton R, Sutton R, Farrell M: Beta-blockers, autoimmunity and rashes. *Lancet* 1980; 2:581.
19. Gange RW, Jones EW: Bullous lichen planus caused by labetalol. *Br Med J* 1978; 1:816–817.
20. Griffiths ID, Richardson J: Lupus-type illness associated with labetalol. *Br Med J* 1979; 2:496–497.
21. Brown RC, Cooke J, Losowsky MS: SLE syndrome probably induced by labetalol. *Postgrad Med J* 1981; 57:189–190.
22. Neumann H, Van Joost T: Adverse reactions of the skin to metoprolol and other beta-adrenoreceptor-blocking agents. *Dermatol* 1981; 162:330–335.
23. Graeber CW, Lapkin RA: Metoprolol and alopecia. *Cutis* 1981; 28: 633–634.
24. Harrower AB, Strong JA: Hyperpigmentation associated with oxprenolol administration. *Br Med J* 1977; 2:296.
25. Bensaid J, Aldigier JC, Gualde N: Systemic lupus erythematosus syndrome induced by pindolol. *Br Med J* 1979; 1:1603–604.
26. Ashford R, Staughton R, Brighton WD: Cutaneous vasculitis due to acebutolol. *Lancet* 1977; 2:462.
27. Bork K: *Cutaneous Side Effects of Drugs.* Philadelphia, WB Saunders Co, 1988, pp 3–18.
28. McEvoy GK: Drug Information 1987 American Hospital Formulary Service. Bethesda, American Society of Hospital Pharmacists Inc, pp 727–729.
29. Sheehan-Dare RA, Goodfield MD: Widespread cutaneous vasculitis associated with diltiazem. *Postgrad Med J* 1988; 64:467–468.
30. Lavrijsen APM, Dijke CJ, Vermeer BJ: Diltiazem-associated exfoliative dermatitis in a patient with psoriasis. *Acta Derm Venereol* 1986; 66:536–538.
31. Lambert DG, Dalac S, Beer F, et al: Acute generalized exanthematous pustular dermatitis induced by diltiazem. *Br J Dermatol* 1988; 118:308–309.
32. Wakeel RA, Gavin MP, Keefe M: Severe toxic erythema caused by diltiazem. *Br Med J* 1988; 296:1071–1072.
33. Sadick NS, Katz AS, Schreiber TL: Angioedema from calcium channel blockers. *J Am Acad Dermatol* 1989; 21:132–133.
34. Carmichael AJ, Paul CJ: Vasculitic leg ulcers associated with diltiazem. *Br Med J* 1988; 297:562.
35. Thomas SE, Wood ML, et al: Photosensitivity reactions associated with nifedipine. *Br Med J* 1986; 292:992.
36. Fisher JR, Padnick MB, Olstein S: Nifedipine and erythromyalgia. *Ann Intern Med* 1983; 98:671–672.
37. Bridgeman JF: Erythematous edema of the legs due to nifedipine use. *Br Med J* 1978; 1:578.
38. Reynolds NJ, Jones SK, Crossley J, et al: Exfoliative dermatitis due to nifedipine. *Br J Dermatol* 1989; 121:401–404.
39. Alcalay J, David M, Sandbank M: Cutaneous reactions to nifedipine. *Dermatol* 1987; 175:171–93.
40. Brenner S, Brau S: Vasculitis following nifedipine. *Harefuah* 1985; 108: 139–140.
41. Scolnick B, Brinberg D: Diltiazem and generalized lymphadenopathy. *Ann Intern Med* 1985; 102:588.
42. Lin AF, Baker B: Verapamil associated Stevens-Johnson syndrome. *Drug Intell & Clin Pharmacol* 1989; 23:987–988.
43. Goodfield MJ, Millard LG: Severe cutaneous reactions to captopril. *Br Med J* 1985; 290:1111.
44. Atkinson AB, Robertson JS: Captopril in the treatment of clinical hypertension and cardiac failure. *Lancet* 1979; ii:836–839.
45. Kayanakis JG, Giraud P, Fauvel JM, et al: Eosinophilia during captopril treatment. *Lancet* 1980; ii:923.
46. Cox NH, Tapson JS, Farr PM: Lichen planus associated with captopril: A further disorder demonstrating the "tin-tack" sign. *Br J Dermatol* 1989; 120: 319–321.
47. Wilkin JK, Kirkendall WM: Pityriasis rosea-like rash from captopril. *Arch Dermatol* 1982; 118:186–187.
48. Parfrey PS, Clement M, Vandenburg MJ, et al: Captopril-induced pemphigus. *Br Med J* 1980; 281:194.
49. Flageul B, Foldes C, Wallach D, et al: Captopril-induced lichen planus pemphigoides with pemphigus-like features. *Dermatologica* 1986; 173:248–255.
50. Wood SM, Mann RD: Angioedema and urticaria associated with angiotensin converting enzyme inhibitors. *Br Med J* 1987; 294:91–92.
51. Leaker B, Whitworth JA: Alopecia associated with captopril treatment. *Aust N Z J Med* 1984; 14:–866.
52. Borders JV: Captopril and onycholysis. *Ann Intern Med* 1986; 105:305–306.
53. Patri P, Nigro A, Rebora A: Lupus erythematosus-like eruption from captopril. *Acta Derm Venereol* 1985; 65:447–448.
54. Solinger AM: Exfoliative dermatitis from captopril. *Cutis* 1982; 29:473–74.
55. Slater EE, Merrill DD, Guess HA, et al: Clinical profile of angioedema associated with angiotensin converting enzyme inhibition. *J Am Med Assoc* 1988; 260:967–970.
56. Kubo SH, Cody RJ: Enalapril, a rash, and captopril. *Ann Intern Med* 1984; 100:616.
57. Ladd AT: Procainamide-induced lupus erythematosus. *N Engl J Med* 1962; 267:1357–1358.
58. Schoen RT, Trentham DE: Drug-induced lupus: An adjuvant disease? *Am J Med* 1981; 71:5–8.
59. Jick H, Miettinen OS, Shapiro S, et al: Comprehensive drug surveillance. *J Am Med Assoc* 1970; 213:1455–1460.
60. Rosin JM: Vasculitis following procainamide therapy. *Am J Med* 1967; 42:625–629.
61. Know JP, Welykyj SE, Gradini R, et al: Procainamide-induced urticarial vasculitis. *Cutis* 1988; 42:469–472.
62. Almeyda J, Levantine A: Cutaneous reactions to cardiovascular drugs. *Br J Dermatol* 1973; 88:313–319.
63. Nudelman PL, Leff IL, Howe CD: Thrombopenic purpura following quinidine. *JAMA* 1948; 137:1219–1220.
64. Pariser DM, Taylor JR: Quinidine photosensitivity. *Arch Dermatol* 1975; 111:1440–1443.
65. Marion DF, Terrien CM: Photosensitive livedo reticularis. *Arch Dermatol* 1973; 198:100–101.
66. Sarkany I: Lichen planus following quinidine therapy. *Br J Dermatol* 1967; 79:123.
67. Mahler R, Sissons W, Watters K: Pigmentation induced by quinidine therapy. *Arch Dermatol* 1986; 122:1063–1064.
68. Harwell WB: Quinidine-induced psoriasis. *J Am Acad Dermatol* 1983; 9:278.
69. Taylor DR, Potashnick R: Quinidine-induced exfoliative dermatitis. *JAMA* 1951; 145:641–642.
70. Shalit M, Flugelman MY, Harats N, et al: Quinidine-induced vasculitis. *Arch Intern Med* 1985; 145:2051–2052.
71. Ritchie EB, Kolb W: Reaction to sodium diphenyl hydantoinate. *Arch Dermatol Sylhil* 1942; 46:856–859.
72. Benton JW, Tynes B, Register HB, et al: Systemic lupus erythematosus occurring during anticonvulsive drug therapy. *JAMA* 1962; 180:115–118.

73. Stanley J, Fallon-Pellicci V: Phenytoin hypersensitivity reaction. *Arch Dermatol* 1978; 114:1350–1353.

74. Gropper AL: Diphenylhydantoin sensitivity. *N Engl J Med* 1956; 254: 522–523.

75. Gately LE, Lam MA: Phenytoin-induced toxic epidermal necrolysis. *Ann Intern Med* 1979; 91:59–60.

76. Rosenthal CJ, Noguera CA, Coppola A, et al: Pseudolymphoma with mycosis fungoides manifestations, hyperresponsiveness to diphenylhydantoin, and lymphocyte disregulation. *Cancer* 1982; 49:2305–2314.

77. Adams JD: Localized cutaneous pseudolymphoma associated with phenytoin therapy: A case report. *Aust J Dermatol* 1981; 22:28–29.

78. Arrowsmith JB, Creamer JI, Bosco L: Severe dermatologic reactions reported after treatment with tocainide. *Ann Intern Med* 1987; 107:693–696.

79. Dunn JM, Groth PE, DeSimone A: Tocainide: A severe adverse reaction. *Drug Intell Clin Pharmacol* 1988; 22:142–145.

80. Harris L, McKenna WJ, Rowland E, et al: Side effects of long-term amiodarone therapy. *Circulation* 1983; 67:45–51.

81. Vos AK, Van Ramshorst AGS, Grosfeld JCM, et al: A peculiar cutaneous pigmentation from cordarone. *Dermatol* 1972; 145:297–303.

82. Moots RJ, Banerjee A: Exfoliative dermatitis after amiodarone treatment. *Br Med J* 1988; 296:1332–1333.

83. Bencini PL, Crosti C, Sala F, et al: Toxic epidermal necrolysis and amiodarone treatment. *Arch Dermatol* 1985; 121:838.

84. Staubi M, Zimmermann A, Bircher J: Amiodarone-induced vasculitis and polyserositis. *Postgrad Med J* 1985; 61:245–47.

85. Porter JE, Zantkuyl CF: Ioderma caused by amiodarone. *Arch Dermatol* 1975; 111:1656.

86. Guindo J, Rodriguez A, Borja J, et al: Propafenone and a syndrome of the lupus erythematosus type. *Ann Intern Med* 1986; 104:589.

87. Grettve J, Johansson B: Digitalis allergy: Review of the literature and report of a case. *Cardiologia* 1958; 32: 374–376.

88. Morrow PC: *Drug Eruptions*. New York; William Wood & Co, 1887, p 118.

89. Cohen RV, Brodsky ML: Allergy to digitalis. *J Allergy* 1940; 12:69–71.

90. Berger H: Thrombocytopenic purpura following use of digitoxin. *JAMA* 1952; 148:282–283.

91. Korkij W, Soltani K: Fixed drug eruptions. *Arch Dermatol* 1984; 120: 520–524.

92. David M, Livni E, Stern E, et al: Psoriasisiform eruption induced by digoxin: Confirmed by re-exposure. *J Am Acad Dermatol* 1981; 5:702–703.

93. Hoff JV, Beatty PA, Wade JL: Dermal necrosis from dobutamine. *N Engl J Med* 1979; 300:1280.

94. McCauley CS, Blumenthal MS: Dobutamine and pururitus of the scalp. *Ann Intern Med* 1986; 105:966.

95. Oglesby JE, Baugh JH: Tissue necrosis due to norepinephrine. *Am J Surg* 1968; 115:408–412.

96. Harber LC, Lashinsky AM, Baer RL: Photosensitivity due to chlorothiazide and hydrochlorothiazide. *N Engl J Med* 1959; 261:1378–1381.

97. Addo HA, Ferguson J, Frain-Bell W: Thiazide-induced photosensitivity: A study of 33 subjects. *Br J Dermatol* 1987; 116:749–760.

98. Robinson HN, Morison WL, Hood AF: Thiazide diuretic therapy and chronic photosensitivity. *Arch Dermatol* 1985; 121:522–24.

99. Thestrup-Pedersen K: Adverse reactions in the skin from antihypertensive drugs. *Danish Med Bull* 1987; 34:3–5.

100. Reed BR, Huff JC, Jones SK, et al: Subacute cutaneous lupus erythematosus associated with hydrochlorothiazide therapy. *Ann Intern Med* 1985; 103:49–51.

101. Goette DK, Beatrice E: Erythema annulare centrifugum caused by hydrochlorothiazide-induced interstitial nephritis. *Intl J Dermatol* 1988; 27:129–130.

102. Ball P: Thrombocytopenia and purpura in patients receiving chlorothiazide and hydrochlorothiazide. *JAMA* 1960; 173:663–665.

103. Jaffe MO, Kierland RR: Purpura due to chlorothiazide. *JAMA* 1958; 168:2264–2265.

104. Bjornberg A, Gisslen H: Thiazides: A cause of necrotizing vasculitis? *Lancet* 1965; 2:982–983.

105. Burry JN, Lawrence JR: Phototoxic blisters from high frusemide dosage. *Br J Dermatol* 1976; 94:495–499.

106. Gibson TP, Blue P: Erythema multiforme and furosemide therapy. *JAMA* 1970; 212:1709.

107. Fellner MJ, Katz JM: Occurrence of bullous phemphigoid after furosemide therapy. *Arch Dermatol* 1976; 112:75–77.

108. Darken M, McBurney EI: Subacute cutaneous lupus erythematosus-like drug eruption due to combination diuretic hydrochlorothiazide and triamterene. *J Am Acad Dermatol* 1988; 18:38–42.

109. Baume P, Ratcliffe FJ, Corry JR: Triamterene, a new natriuretic agent. *Am J Med Sci* 1963; 245:668–75.

110. Bar-On H, Eisenberg S, Eliakim M: Clinical experience with ethacrynic acid, with reference to a possible complication of the Henoch-Schoenlein type. *Isr J Med Sci* 1967; 3:113–118.

111. Lee SL, Rivero I, Siegel M: Activation of systemic lupus erythematosus by drugs. *Arch Intern Med* 1966; 117:620–626.

112. Peterson II: Hydralazine-induced systemic lupus erythematosus presenting as pyoderma gangrenosum-like ulcers. *J Am Acad Dermatol* 1984; 10:379–384.

113. Kiddin MW, Wiliamson RCN: Hydralazine-induced SLE-like syndrome presenting as a leg ulcer. *Br Med J* 1979; 2:1330.

114. Petty R, Medley DRK: Cutaneous manifestations of hydralazine toxicity. *Br Med J* 1980; 280:482.

115. Wilkin JK: Vasodilator rosacea. *Arch Dermatol* 1980; 116:598.

116. Sequeira W, Polisky RB, Alrenga DP: Neutrophilic dermatosis (Sweet's Syndrome). *Am J Med* 1986; 81:558–560.

117. Hendricks AA, Dec WD: Contact dermatitis due to nitroglycerin ointment. *Arch Dermatol* 1979; 115:853–855.

118. Fisher AA: Dermatitis due to transdermal therapeutic systems. *Cutis* 1984; 34:526–531.

119. Burton JL, Marshall A: Hypertrichosis due to minoxidil. *Br J Dermatol* 1979; 101:593–595.

120. DiSantis DJ, Flanagan J: Minoxidil-induced Steven-Johnson syndrome. *Arch Intern Med* 1981; 141:1515.

121. Ackerman BH, Townsend ME, Golden W, et al: Pruritic rash with actinic keratosis and impending exfoliation in a patient with hypertension managed with minoxidil. *Drug Intell Clin Pharmacol* 1988; 22:702–703.

122. Kuan P: Dangers of diazoxide. *Br Med J* 1973; 1:114.

123. Holt PJA, Navaratnam A: Lichenoid eruption due to methyldopa. *Br Med J* 1974; 3:234.

124. Vaillant L, LeMarchand D, Grognard C, et al: Photosensitivity to methyldopa. *Arch Dermatol* 1988; 124:326–327.

125. Church R: Eczema provoked by methyldopa. *Br J Dermatol* 1974; 91: 373–378.

126. Wells JD, Kurtay M, Lochner JC, et al: Granulomatous skin lesions and alpha-methyldopa. *Ann Intern Med* 1974; 81:701–702.

127. Mackie BS: Drug-induced ulcer of the lip. *Br J Dermatol* 1967; 79:106–110.

128. Van Joost T, Faber WR, Manuel HR: Drug-induced anogenital cicatricial pemphigoid. *Br J Dermatol* 1980; 102:715–718.

129. Witman G, Davis R: A lupus erythematosus syndrome induced by clonidine hydrochloride. *RI Med J* 1981; 64:147–150.

130. Wilkin J: Exacerbation of psoriasis during clonidine therapy. *Arch Dermatol* 1981; 117:4.

131. Wiser TH, Kazakis AM, LaCivita CL: Transdermal clonidine: An association with recurrent herpes simplex and hyperpigmentation. *J Am Acad Dermatol* 1987; 17:143–144.

132. Dick JBC, Northridge DB, Lawson AAH: Skin reactions to long-term transdermal clonidine. *Lancet* 1987; 1:516.

133. McMahon FG, Weber MA: Allergic skin reactions to transdermal clonidine. *Lancet* 1983; 2:850–851.

134. Hirschboeck JS, Madison FW, Pisciotta AV: Alopecia and other toxic effects of heparin and synthetic heparinoids. *Am J Med Sci* 1954; 227:279–282.

135. Chernoff AI: Anaphylactic reaction following injection of heparin. *N Engl J Med* 1950; 242:315–319.

136. Kelly RA, Gelfand JA, Pincus SH: Cutaneous necrosis caused by systemically administered heparin. *JAMA* 1981; 246:1582–1583.

137. O'Toole RD: Heparin: Adverse reaction. *Ann Intern Med* 1973; 79:759.

138. Nalbandian RM, Mader IJ, Barrett JL, et al: Petechiae, ecchymoses, and necrosis of skin induced by coumarin congeners. *JAMA* 1965; 192:107–112.

139. Adams CW, Pass BJ: Extensive dermatitis due to warfarin sodium. *Circulation* 1960; 22:947–948.

140. Schiff BL, Kern AB: Cutaneous reactions to anticoagulants. *Arch Dermatol* 1968; 98:136–137.

141. Sheps SG, Gifford RW: Urticaria after administration of warfarin sodium. *Am J Cardiol* 1959; 3:118–120.

142. Feder W, Auerbach R: "Purple Toes": An uncommon sequela of oral coumarin drug therapy. *Ann Intern Med* 1961; 55:911–917.

143. Baker H, Levene GM: Cutaneous reactions to anticoagulants. *Br J Dermatol* 1969; 81:236–238.

144. Baker H, Moore-Robinson M: Cutaneous responses to aspirin and its derivatives. *Br J Dermatol* 1970; 82:319–321.

145. Brannen M, Forbes AF: Salicylate sensitivity. *Texas J Med* 1966; 62:58–60.

146. Shelley WB: Birch pollen and aspirin psoriasis. *JAMA* 1964; 189:985–988.

147. Tromovithch TA, Jacobs PH, Kern S: Acanthosis nigricans-like lesions from nicotinic acid. *Arch Dermatol* 1964; 89:222–223.

148. Tobert JA, Shear CL, Chremos AN, et al: Clinical experience with lovastatin. *Am J Cardiol* 1990; 65:23F–26F.

149. Harrison CE, Davies NE: Unusual reactions to common cardiovasculavascular drugs. *J Med Assoc Georgia* 1970; 59:117–120.

The Golden Years or The Wasteland

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As the twentieth century comes to a close, it behooves us to come to grips with a burgeoning gerontological problem. It requires the formulation of an imaginative, sophisticated, and well-financed program for the care of our chronically ill and aged population. Social philosophers observe that one of the significant criteria of a civilized society's worth is the scope of its care for the aged.

Improved standards of living, modern preventive medicine, advances in bioscience, and medical technology are responsible for an expanding aged population. During the past ten years there have been scholarly articles published, highlighting society's burden: the mounting numbers of septagenarians and octogenarians in the population who often have multiple chronic diseases and the increasing prevalence of Alzheimer's disease with aging.[1,2,3] In addition, many new periodicals dedicated to studies of the aged are being published.

Dr Robert Butler,[4] Dr Edward L. Schneider, [5,6,7] and other notable contributors[8,9,10,11] have written with thoroughness about the geriatric scene. Prominent is their emphasis on the need for more gerontological research and an expansion of a disciplined, academic teaching ambience in nursing homes, where possible.

Most of us are familiar with senior citizens who have an octogenarian parent who is enfeebled, homebound, and living at a distance. Occasionally, that parent will end his or her life in a nursing home. The nursing home is chosen because an elderly child cannot provide the essential nursing care due to lack of expertise, lack of space at home, or because of his or her own limiting infirmities.

For the concerned family of the disabled elderly, contact with a nursing home evokes negative images: alienation and loneliness, bed restraints, the odor of urine, unfinished meals, and lack of compassionate care.[12] Inevitable for the middle-aged and elderly children, there is unresolved guilt. They see a parent surrounded by demons of nothingness . . . warehoused.

After visiting several nursing homes, I remained with a gray and depressing after-image. First, to my surprise, there was a penetrating quiet . . . an absence of voices. Lined up in the corridors, many patients appeared to be captives in their wheelchairs. Prominent was the fixed, wrinkled dignity of their faces. Most were dozing, some bent to the side, or jackknifed forward. Not one was reading a newspaper or a magazine, or talking to a neighbor. With the support of a "walker" or a cane, an occasional patient was inching along the hall. Apathy. Frozen immobility. A void of loneliness. The occasional TV set had no audience. Three out of four were women. Daumier, the great 19th century cartoonist, might have sent a dramatic black-on-white message were he to depict this scene.

My first encounter was with a stone-faced woman (a victim of Parkinson's disease) in soft flannel strap restraints. I squeezed one of her smooth, gnarled hands, which fluttered like a captive, small bird's wings in mine, and I said, "Good morning." Not looking up, whispering, she said, "I know it's morning. There are cornflakes on my tray. Why are they prolonging my life?" With a forced smile, I retreated and said, "Have a good day." I sweated.

From somewhere down the hall I heard occasional weak, garbled cries. In one of the rooms, an aged woman lay in her cribbed bed, staring at the ceiling, eyelids rarely blinking. Hanging on a pole at her bedside was a bag of brownish fluid from which a plastic tube led to her gastrostomy site. Farther along, my guide and I discovered that the man from Room 703, fully clothed, was now asleep in the bed in Room 709. The bed was soaked with urine. The true occupant of 709 was absent because of a dental appointment outside of the nursing home.

At the nurses' station there was a black bulletin board with large white print announcing: Today is Sunday . . . The sun is shining . . . Catholic Mass at 9:30 AM . . . Methodist services at 11:00 AM . . . First night of Passover tomorrow. On another visit the sign read thus: Exercises at 9:30 AM . . . Puppy Pets at 11:00 AM . . . Barbecue at noon in the patio . . . Kids' Klasses at 2:30 PM . . . Talking Books tomorrow at 2:00 PM . . . "Sing-Along" at 4:00 PM.

The aforementioned activities are a noble try to exercise the cerebral circuits and to overcome the boredom. However, the penetrating and gruesome fact is that a nursing home resident not only loses his or her autonomy, but is, in this ambience, psychosocially neutralized and diminished. The banker, the physicist, the house painter, the shoemaker, and the former social worker are brought to equal status: they are infantilized and caught in a Greek tragedy which they cannot escape. Day and night are a "living deadness." The dawn, the midday sun, and the dusk are the same. Yet, outside, there are variations which enrich the atmosphere . . . countryside, flowerbeds, grazing animals.

So much for the surface. Under the skin of nursing home patients is a textbook of geriatric problems, a grab bag of multi-organ diseases such as congestive heart failure, chronic obstructive lung disease, cardiac arrhythmias, the neurological deficits after cerebral vascular occlusions, and a confusing spectrum of cerebral impairment. Add to this, visual and auditory losses, osteoarthritis, gait disorder, and the unstable urinary bladder.[13,14] Because of the multiplicity of chronic disease, most of the patients are on multiple drug therapy. However, we are forced to recognize that the daily mix of drugs may bring on side reactions such as cardiac dysfunction, further loss of bladder control, insomnia, lack

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of motor coordination, diarrhea, dehydration, cognitive losses, and even mania.

Not all of the nursing home inmates fit into a pattern of apathy, mental decline, and depression. Some, like many elderly in the mainstream, possess amazing cognitive functions and intellectual agility. Excluding those with Alzheimer's disease, the mix would be a gold mine for the research neurophysiologist interested in magnetoencephalography and positron emission tomography.

After I had finished a few mini-tours at different homes and had browsed through some of the recent gerontological literature, I adopted one of the patients at a local, non-teaching nursing facility, hoping thus to penetrate reality. My only request was that the adoptee be a male who was mentally competent and that he was willing to accept me as a regular visitor. Later, I would also adopt a female patient.

The adoptee chosen for me was a 65-year-old engineer who had already spent one year at the nursing home. A former hydrogen-bomb-maker at one of our most prestigious research laboratories, he looked like Persian Gulf hero, Lt. General H. Norman Schwarzkopf. A large man, mildly dyspneic and cyanotic, he was linked by nasal catheter to a tall tank of oxygen. Because he worked with uranium ore in the early 1950s, and had a long exposure to fluorine gas, I surmised that his respiratory difficulties were related to the former occupational hazard. His medical regimen also included nine different drugs daily!

The man was desperate for companionship. Amazingly bright, exuding genius, sitting up, feet over the side of the bed, he talked on and on, euphorically, rarely pausing, for the next 40 minutes. I interrupted him, playing doctor; and I took his pulse: 120, with frequent premature beats (fibrillating?). Suddenly, and with apologies, he fell back on his pillow, apparently exhausted. I left with guilt that I had taxed him. I promised to visit regularly and to start a weekly correspondence. Tearfully, he thanked me. I wrote suggesting that we prepare topics for weekly discussion. A part of his first letter to me was as follows:

> My Dear Doctor: There are so many possible ingredients for our conversational salads. Next time let's talk about one of the following: "Designing explosives," "Technical versus people problems," or "Producing ^{235}U and ^{238}U." etc, etc.

We started on ^{235}U and ^{238}U. I understood some. Over about six months we discussed President George Bush's failure to address urgent social and environmental problems at home, the revolutions in eastern Europe, the psyche of the Arab world, and other subjects—even Treasury bills. He gasped uncomplainingly through my one-hour visits.

Suddenly he was transported to another nursing facility in his home community many miles away. I continued to write to him, hoping that our exchanges would be an exciting means of communication. However, he was remiss as a correspondent. I later learned that he was deterred by his now Parkinson-like script. Frequently, at about 6:00 AM, I telephoned his night nurse and left him a message. About nine months later, driven by guilt, I made the long drive over scenic mountain roads to visit him. My surprise appearance was intensely emotional for both of us. My tears welled up. He was exuberant. He was able to sit up briefly. The skin of his hands was now purple, as were his lips. His neck veins were bulging, and his pulse was 104 and regular. However, his personality had not changed. A nebulizer in one hand, oxygen flowing into his nasal catheter, he lectured to me on Middle East politics. We agonized about poor Mikhail Gorbachev; and we philosophized about how often the rage for utopia brought on terror. With the erudite jargon of his former workplace, he said, "The 'critical mass' of right men is not yet on the scene to push these fools in the right direction." Finally, he added, "I sent my wife on a one week's vacation to Hawaii because it is our 46th wedding anniversary. You know, Doc? You're a friend." He beamed and then masked a brief sob. He was hoarse and gasping. On the long trip home, dazzlingly colorful scenes: in the distance, broad and high mesas, fields of red poppies, white-bellied Oregon juncos skimming over tall grasses, a lone bald eagle soaring majestically above. My guilt was worsening. I was now speeding away at 75 miles per hour.

Most medical schools briefly expose their students to the non-stimulating atmosphere of a nursing facility. Because of the absence of diagnostic interventions and intensive treatment programs there, one suspects that the students will find little to excite them. The intellectual stimulations ever present in the intensive care units, the emergency and operating rooms, and the nursing areas of acute care hospitals are more attractive. However, unfortunately, in these settings eyeball-to-eyeball exposure with the patient and intimate communication are minimal. The patient becomes a "case" . . . alienated. Pages of laboratory data, the results of invasive procedures and the treatment protocols are the main topics, discussed out in the hall or in the conference room. An almost depersonalized scenario.

It occurred to me that rather than a one to two month, unsupervised stay at an exotic place like a mountain village in the Peruvian Andes, fourth year medical students would be better enriched by a mandatory month or two of clinical clerkship in a teaching nursing home. In such a collegial atmosphere, students could be given hands-on exposure to long-term care and to the frustrating problems of communicating with the helpless elderly . . . patients who never get well. It would be another soil for nurturing the young physician-to-be, and it would challenge him to walk the tightrope of multiple chronic disease and multiple medication. Students would be constantly reminded not to impose the image of the nursing home inmate on those somewhat limited elderly still in the mainstream of society. As an aside, the teaching gerontologist might mention Marcus Cato, the great Roman politician and philosopher, who began the study of Greek at the age of 80 and eventually thrust himself on his sword when he was sick and helpless at the age of 85. He might also point to 83-year-old Chancellor Konrad Adenauer, standup comic George Burns at 92, and Artur Rubinstein still concertizing in his mid-80s.

Ideally, teaching rounds[15] for students should be the teamwork of an academic gerontologist, a teaching nurse, and a social worker. Other participants at conferences might include a cardiologist, a neurologist, a gerontological psychiatrist, and an academic clinical pharmacologist. The biology of aging[16] and the challenges of polypharmacy[17,18,19] should be high on the students' reading agenda.

Conferences might give pause for some philosophic discussions of society's moral and ethical responsibilities toward the aged, such discussions leading to the literature on "living wills," legal wranglings over the right to die with dignity, and euthanasia.[20] A medical ethicist and a theologian could enrich the effort even more.

Fortunately, more than 95% of our elderly are coping, either alone or as part of a family scene, out in the mainstream, or they are included in a community network that may be offering assistance with shopping, cleaning, bathing, doctors' appointments, payment of bills, laundry, and even library borrowing. Some have radio-telephonic communication devices that alert networks to deal with urgent situations. However, when the family or the community cannot adopt the impaired elderly for total care, or if they are inconsistent, transfer to a nursing home is imperative. For most patients, I was told, death ensues there in about two- or two-and-a-half years—a long night's journey.

Obviously, in years to come, physicians will spend more of their time at nursing homes. Although they may not be able to cure their patients, their ultimate role must be to provide expertise, making the lives of these elderly comfortable. Moreover, unless the staff, at all levels, delivers unwavering, dignified, and compassionate nursing care, the best of these scenarios will be wanting. A possible unhappy fact is that at the lower rungs of many a nursing home staff the desired quality of commitment may be lacking. This is the area in which there is frequent turnover of personnel.

Visionaries among our social workers, psychologists, gerontologists, and average persons in the mainstream should be able to enrich the atmosphere of the "nursing home". . . a place which, truly, is not a "home."

Wishful thinking: Congress will be magnanimous in funding research on the afflictions of the aged; there will be adequate funding for the training of a proud and elite corps of highly-paid personnel for the staffing of nursing homes; and ultimately, as in the Netherlands and Scandinavia, there will be ample government funds for totally relieving the patient, or the family, of the financial burden of a nursing home.

The care of our aged is one of the moral and intellectual challenges of society today. Platforms in outer space can wait; and so can the new F-22s at $100 million each.

I have lived long enough. My way of life
Is fallen in sere, the yellow leaf,
And that which should accompany old age,
As honour, love, obedience, troops of friends,
I must not look to have, but in their stead
Curses, not loud but deep . . .

William Shakespeare (1564–1616)
Macbeth: 5.3, lines 22–27

REFERENCES

1. Deary IJ, Whalley LJ: Recent research on the causes of Alzheimer's disease. *Brit Med J* 1988; 297:807–809.
2. Glenner G: The pathobiology of Alzheimer's disease. *Ann Rev Med* 1989; 40:45–51.
3. Telephonic communication: Alzheimer's Disease and Related Disorders Association Inc., Chicago, Illinois.
4. Butler R: The Teaching nursing home. *JAMA* 1981; 14:1435–1437.
5. Schneider EL: Teaching nursing homes. *N Engl J Med* 1983; 6:336–337.
6. Schneider EL, Org M, Aung ML: Teaching nursing homes revisited: Survey of affiliations between American medical schools and long-term care facilities. *JAMA* 1987; 20:2771–2775.
7. Schneider EL, Guralnik JM: The aging of America–Impact on health care costs. *JAMA* 1980; 17:2335–2340.
8. Woolliscroft JO, Calhoun JG, Maxim BR: Medical education in facilities for the elderly. Impact on medical students, facility staff, and residents. *JAMA* 1984; 24:3382–3385.
9. Ahronheim JC: Pitfalls of the teaching nursing home. A case for balanced geriatric education. *N Engl J Med* 1983; 6:335–336.
10. Fries JF: Aging, natural death, and the compression of morbidity. *N Engl J Med* 1980; 3:130–135.
11. Reisenberg D: The teaching nursing home. A golden annex to the ivory tower. *JAMA* 1987; 22:3119–3120.
12. Vladeck BC: *Unloving Care: The Nursing Home Tragedy*. New York: Basic Books, 1980.
13. Resnick NM, Yalla S: Destrusor hyperactivity with impaired contractile function (an unrecognized but common cause of incontinence in elderly patients). *JAMA* 1980; 22:3076–3081.
14. Resnick NM, Yalla S: Management of urinary incontinence in the elderly. *N Engl J Med* 1985; 13:800–805.
15. Schneider EL (ed): *The Teaching Nursing Home: A New Approach to Geriatric Research, Education, and Clinical Care*. New York: The Raven Press, 1985.
16. Finch C, Schneider EL: *Handbook of the Biology of Aging*, ed 2. New York: Van Nostrand Reinhold. 1985.
17. Vestral RE, Dawson GW: Pharmacology and aging, in Finch CE and Schneider EL: *Handbook of the Biology of Aging*, ed 2. New York: Van Nostrand Reinhold, 1985, pp 744–819.
18. Reidenberg MM: Drug therapy in the elderly. The problem from the point of view of a clinical pharmacologist. *Clin Pharmacol* 1987; 42:677–680.
19. Buechler JR, Malloy W: Drug therapy in the elderly. How to achieve optimum results. *Postgraduate Medicine* 1989; 6:87–89.
20. De Wachter MAM: Active euthanasia in the Netherlands. *JAMA* 1989; 23:3316–3319.

Ethical considerations concerning the HIV-positive physician

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An extensive literature deals with the ethical issues and dilemmas in the acquired immunodeficiency syndrome (AIDS) and human immunodeficiency virus (HIV) infection.[1–4] Some of these issues include HIV testing, confidentiality, partner notification, informed consent, stigmatization and discrimination, costs of treatment and research, public health versus civil liberties, truth telling, and the refusal to care for seropositive persons.

On July 27, 1990, the Centers for Disease Control (CDC) reported the possible transmission of HIV from an HIV-positive dentist to a patient as a result of patient care,[5] and subsequent information indicated that this dentist infected several of his patients.[6] The widespread public hysteria and panic after this report was fueled by the mass media and the nation's newspapers and magazines. Many professional groups including the American Medical Association (AMA) hurriedly issued guidelines for physicians infected with HIV. Some of these guidelines and recommendations appear to have been "unwarranted, imprecise and not in the public interest."[7]

The case of the dentist cited above catapulted to center stage the numerous ethical, medical, and public policy issues surrounding HIV-infected health care workers. Does an HIV-infected health care worker pose a risk to patients? How much risk? What constitutes allowable risk? Should all health care workers be tested for HIV? Does the patient's right to know outweigh the health care worker's right to confidentiality?[8] Should HIV-infected health care professionals be restricted in the practice of invasive or non-invasive procedures? Are such restrictions discriminatory? Two extreme positions are being widely debated. "One, based on prejudice and irrational fear, would require testing and restrictions on a wide range of health care professionals. The other, based on civil libertarian concerns, would set no limits."[9]

ETHICAL ISSUES IN MANDATORY HIV TESTING

Many politicians and concerned citizens advocate mandatory AIDS testing for health professionals as a public health measure. Others argue that AIDS testing against a person's will violates that person's individual rights. Many philosophers and ethicists argue that individuals have rights (eg, a right to privacy) that cannot be violated even in the interest of the public good. Even if mandatory AIDS testing were shown to be an effective public health measure, these philosophers would argue that this fact does not justify the violation of individual freedom that mandatory testing would imply. In this view, the concept that individuals have inalienable rights that take precedence over the common good is fundamental to our political and social traditions. Indeed, this view has been the dominant theme in European and American political thought since the late 18th century.

Although individuals may have the right to refuse testing, this does not mean they have a right to willfully or negligently infect others. Even if AIDS can be spread from health professionals to patients, one can argue that mandatory testing is immoral, but the physician who knows or suspects he has AIDS has an ethical obligation to avoid procedures that have a risk of infecting his patients or at least to warn patients on whom he performs invasive procedures of that risk. It follows that one can consistently oppose mandatory AIDS testing, yet still advocate penalties for those who willfully harm others by infecting them with a disease.

How does one reconcile the view that mandatory AIDS testing violates the rights of those tested with the necessity to protect other individuals from disease? The "Typhoid Mary" example is often cited by advocates of various public health measures as an obvious case where restrictions on freedom are necessary to maintain public safety. There is, however, a fundamental distinction here. The question of whether the rights of an infected and highly contagious individual may be restricted is a *different* ethical question from whether the rights of a large group, such as all health care professionals, whose members are presumably mostly free of communicable disease can have their freedom and privacy violated for reasons of public health. The courts have made an analogous distinction, regarding freedom from unreasonable search and seizure. One might countenance a police search, with a court-ordered warrant, of the home of someone strongly suspected of having committed a crime, but not allow the police search of all the homes in a neighborhood (even a high crime neighborhood) in hopes of turning up criminal activity. Similar arguments have also

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Prepared as an educational and informational report by the Committee on Bioethical Issues of the Medical Society of the State of New York.

been raised, especially by civil liberties groups, against routine alcohol testing of automobile drivers, although the courts do not always uphold these arguments. These groups might not disapprove of the police stopping a driver whose erratic behavior represents a threat to other individuals. On the other hand, they would argue that routine detention of drivers for alcohol testing in hopes of detecting a driver with an elevated alcohol level represents a fundamental violation of their and indeed all our civil liberties. The same argument can be made regarding mandatory AIDS testing of health professionals or other groups.

OCCUPATIONAL RISK OF CONTRACTING HIV

The risk from occupational exposure to HIV has been evaluated prospectively in several thousand health care providers with a history of accidental parenteral inoculation with HIV-infected material. Cumulative results indicate that the rate of nosocomial HIV transmission is approximately 0.4% per parenteral needle-stick exposure.[10–11] Needlestick injuries are common[12–13] especially among resident physicians[14] but can be prevented or reduced in frequency by appropriate precautions.[15] The risk to health care workers is thus exceedingly low but does exist, and fear persists.[16]

Type of exposure, size of inoculum and stage of HIV infection may all play a role in the risk of transmission.[15] Surgeons are acknowledged to be at special risk of contracting HIV from an infected patient because of the frequency and magnitude of blood exposure in the operating room. Some surgeons advocate universal testing of patients before surgery. The various pros and cons of such routine testing without specific informed consent, even if confidentiality can be assured, have been summarized previously.[17]

Some physicians are reluctant to care for patients with AIDS.[18] There is historic precedent for such an attitude. A number of famous physicians, including Galen and Sydenham, fled from patients with contagious epidemic diseases. On the other hand, many of their colleagues, at considerable personal risk, remained behind to care for the sick.[19]

One physician questions whether patients have the right to infect their doctors.[20] Compulsory isolation of patients with tuberculosis was carried out without public outcry. The infringements of individual rights were seen to be necessary to protect the community. By the same reasoning, "AIDS is a question of infectivity and not of morality. The responsibility of the physician is to prevent spread of infection into the community."[20] Hence, high-risk groups should be tested, partners of seropositive patients should be notified, and prostitutes who are HIV-infected and continue to work should be isolated.[20]

One can take serious issue with these recommendations. The subject of widespread HIV testing continues to be debated. Even among HIV-infected patients screened with their informed consent, the dilemma for the physician is the duty of confidentiality to the patient versus the duty to warn spouses and other persons at risk.[21]

RISK TO PATIENTS FROM HIV-POSITIVE PHYSICIANS

In January 1989, newspapers and television stations reported the identity of a Nashville, Tennessee, surgeon who had AIDS. Concern about surgeon-to-patient transmission of HIV was widespread and persisted in spite of reassurances from health authorities. HIV antibody testing was therefore offered to the surgeon's patients. Only one of 616 patients was found to be HIV-positive, and he was an intravenous drug user whose medical history suggested that he already had AIDS at the time of surgery.[22] The investigators concluded that: "the risks to patients operated on by HIV-infected surgeons are most likely quite low." They supported recommendations for the individualized assessment of HIV-infected health care workers.[22] Three earlier reports addressed the issue of HIV transmission from infected physicians to patients.[23–25] Not a single case of such transmission was found.

Hepatitis B virus transmission from surgeon to patient is well documented.[26–30] It is therefore not surprising that HIV transmission to several patients from an infected dentist was recently demonstrated.[5,6] The probability of surgeon-to-patient HIV transmission is estimated to be between 1 per 100,000 and 1 per 1 million operations.[31] The exposure rate is strongly influenced by the type of procedure. Ophthalmic surgery has the lowest risk, and pelvic surgery has the highest. In spite of the extremely low risk to patients many national medical organizations issued statements and guidelines that distressed many practicing physicians.

Both the AMA and the American Dental Association (ADA) called on HIV-infected physicians and dentists to stop performing invasive procedures or to disclose their HIV status to patients scheduled for surgery.[32] At the same time, the AMA and ADA opposed mandatory testing. The American Academy of Orthopedic Surgeons said that HIV-infected orthopedists should stop performing invasive procedures, except in emergencies or after obtaining informed consent from patients.[33] The California Medical Association issued a policy that HIV-infected physicians should not perform invasive procedures that carried a high risk of glove or skin laceration.[34] In New Jersey, a Superior Court judge ruled that a surgeon infected with the AIDS virus must inform patients before operating on them.[35] The Medical Society of New Jersey called for universal testing of hospitalized patients and of health care workers, including physicians,[36] but did not address the issue of whether HIV-infected physicians should restrict their patient contact.

In February 1991, the Centers for Disease Control (CDC) convened a two-day meeting in Atlanta to review current information on the risks of blood-borne HIV transmission from health care workers to patients. After considering the numerous testimonies heard at the conference, the CDC-drafted guidelines recommending that infected physicians voluntarily stop performing procedures that carry a risk of transmission, or seek the approval of local committees. Speakers from medical, dental, public health, labor, and advocacy groups overwhelmingly stressed the minuscule risk of HIV transmission from a health care worker to a patient. The risk of health care workers getting HIV from patients is much greater. The CDC was cautioned to "avoid a quick fix for a tough problem."[37] The common themes that emerged from the conference were (1) develop, promulgate, and implement a high standard of infection control; (2) invent safer instruments and techniques; (3) collect more data and perform more studies to determine the real transmission rate from health care workers to patients; (4) shift the emphasis on avoiding infection from where it is rare—the operating room or dental office—to where it is rampant.[37]

RESPONSE TO THE OVERREACTION TO THE ISSUE OF HIV-INFECTED PHYSICIANS

The American College of Physicians, at the CDC conference cited above, reaffirmed its 1988 position paper stating that "HIV- infected physicians, if otherwise able to care for patients, present virtually no risk of HIV transmission to their patients." The American College of Emergency Physicians stated that it believes health care workers who are HIV positive should not be precluded from performing normal medical services where the performance of those services does not constitute a significant threat of harm to patients or others. In addition, health care workers should not be required to disclose their HIV status if their performance of a procedure does not pose a significant risk of transmission.[32] The NYS Department of Health issued guidelines in January 1991 stating that health care facilities in New York State are not required to disclose information to patients about the HIV status of personnel, including physicians and others who perform invasive procedures.[38]

The AMA and ADA recommendations that HIV-infected physicians and dentists disclose their HIV status to patients and/or to cease performing invasive procedures were criticized as "unwarranted, imprecise, and not in the public interest."[7] Physicians could be deprived of their livelihood, either through abandoning their practices or having their patients abandon them. The HIV-infected physician is also a patient and should have the same rights of privacy and confidentiality as any other person. Both physicians and patients share the duty of disclosing HIV status when appropriate so that both parties can take precautions.[39]

HIV-INFECTED DENTISTS

The controversy over the HIV-infected health care professionals was catapulted to center stage when the CDC demonstrated HIV transmission from a dentist to five of his patients.[5–6] An extensive literature exists about AIDS in dental practice[40] and the legal and ethical dilemmas posed when a dental professional becomes HIV-infected.[41] In spite of infrequent compliance with recommended infection control precautions, frequent occupational exposure to persons at increased risk for HIV infection, and frequent accidental puncturing of the skin with sharp instruments, dental professionals are at low occupational risk for HIV infection.[42] A national survey of 300 dentists showed that 44% doubted the possibility of dentist-to-patient transmission of HIV and did not believe the CDC case report. They did believe, however, that infected dentists should refrain either from clinical work or modify their procedures.[42]

REASONABLE APPROACHES TO THE HIV-INFECTED PHYSICIAN

A reasonable framework for protecting from stigmatization an HIV-infected physician in the absence of demonstrated material risks "should be based on the same fiduciary foundation that grounds the ethical obligation of physicians not to harm patients. Physicians may be obliged voluntarily to refrain from risky practice, but this determination should be made on an individual basis between the infected physician and his personal doctor in consultation with infection control personnel."[8]

The Deans of seven medical schools in the New York area and the presidents of the major New York teaching hospitals stated that testing of all health professionals for HIV infection and the removal of those who test positive from all invasive patient care procedures is not logical.[43] The experience with hepatitis B virus suggests that risk of transmission is very low and can be reduced further by adherence to basic infection control procedures. The vast majority of hepatitis B transmissions are associated with failure or breaks in these procedures. The main risks for HIV transmission are unprotected sexual activity and intravenous drug use, and attention should be focused on these issues. The above authors agree to the following:

> Physicians, dentists, and other health workers who are functionally impaired from any cause and potentially harmful to their patients should not be involved in patient care. Testing for HIV and reporting HIV test results in these individuals will not address this need and will deflect attention from the far larger and more serious problem of the epidemic of HIV infection.[43]

Some of the questions under debate are the following: Should health care professionals be required to disclose their serologic status to patients? Should HIV-infected health care professionals be restricted in the practice of noninvasive procedures? Should HIV-infected health care professionals be restricted in the practice of invasive procedures? If health care professionals should be restricted, does it follow that they must be subject to screening? Would professional rules limiting the right to practice invasive procedures violate anti-discrimination statutes?[9]

Public policy in this area should be scientifically rational, should protect the public, and should also protect the health care worker from discrimination.[44] Universal testing of all health care workers and patients has no place in such a policy. At the CDC-sponsored open meeting on the prevention of HIV transmission from health care workers to patients, ninety speakers from more than 70 organizations argued against mandatory testing.[45]

Regrettably, HIV testing has become a political issue rather than a public health issue.[46] To loosen the stringent New York State requirements on HIV-testing, including informed voluntary and specific consent, several medical societies in New York State unsuccessfully sued the Commissioner of Health in 1989 to compel him to define AIDS and HIV infection as sexually transmitted and communicable diseases. The AMA also believes that HIV infection should be classified as a "sexually transmitted disease." However, public health authorities are reluctant to do so, in part because of the enormous financial burden involved. Screening and testing would have to be accompanied by education, counselling, and treatment for those found to be positive. One investigator points out that: "The effort to sustain a set of policies treating HIV-infection as fundamentally different from all other public health threats will be increasingly difficult. Inevitably, HIV exceptionalism will be viewed as a relic of the epidemic's first years"[47]

PRACTICAL SUGGESTIONS REGARDING THE HIV-INFECTED PHYSICIAN

A physician accidentally infected with HIV while working as a house officer at Johns Hopkins suffered not only from the disease but from personal, professional, and economic ostracism and isolation.[48] Clearly, HIV-infected persons, whether physicians or lay people, "need to be protected against discrimination and hysteria, but doing so requires social and political measures, not epidemiologic ones. Jobs, housing, and insurance benefits, for example, should be protected by statute."[49]

An ethic of autonomy, if it is to be an ethic at all, must be an ethic of individual responsibility; however is individual responsibility a sufficient basis for public confidence?[50] The University of Washington in Seattle has established a thorough and well-considered policy on health care workers infected with HIV. In part, its summary section reads as follows:

1. The HIV and/or hepatitis B seropositive individual shall abstain from performing invasive procedures (described elsewhere) involving high risk of exposing the patient to his or her body fluids whether or not his or her employer is notified.
2. An individual who is at high risk for HIV and/or hepatitis B infection, and whose duties and responsibilities include performing invasive procedures, shall be tested for HIV and hepatitis B or shall abstain from performing invasive procedures . . . whether or not his or her employer or supervisor is notified.[50]

The responsibility of disclosure or withdrawal from certain procedures is left to the health care worker.

A distinguished national faculty met at a conference, December 7–8, 1990, in New Brunswick, New Jersey, on policy options concerning the HIV-positive health professional. They concluded that: "Policies that support infected physicians and other professionals are more likely to be effective than those which exclude and punish. Counseling, confidentiality, job protection, financial indemnification, and, where indicated, retraining and reassignment are not only more humane, but more likely to be more workable."[51] The following points were also stressed:

> Rigid exclusionary measures are not justified by current risk data.
>
> Policies structured around the discovery and exclusion of the irresponsible seem inherently unjust and, in any case, will not work.
>
> Social and collegial support for infected health care workers will encourage responsible behavior.
>
> "Responsible behavior," arguably including avoidance of some procedures and surely including scrupulous infection control measures, always entails obtaining sound advice from colleagues and relevant experts.
>
> Vigorous, sustained education coupled with voluntary compliance still represents the best overall policy direction.[51]

The 1991 House of Delegates of the Medical Society of the State of New York adopted a resolution, stating that physicians and medical students have the responsibility to prevent transmission of communicable diseases to their patients. Furthermore, physicians and medical students should, whenever appropriate, determine their HIV status. In addition, if a physician's ability to practice medicine is impaired, either physically or mentally by HIV infection or any other disease, he or she should not practice medicine. Finally, the resolution asserted that if a physician or medical student is HIV seropositive but not impaired, he or she should not engage in any professional activity for which there is scientific evidence of risk of disease transmission to the patient.

On July 12, 1991, the CDC issued its long-awaited guidelines explicitly addressing the threat that may be posed to patients by HIV-infected health care workers. The guidelines call for voluntary AIDS testing of health care workers and require those who test positive to inform their patients before performing exposure-prone procedures.

Only a few days after these guidelines and recommendations were released, a Long Island dentist died of AIDS. Hundreds of panic stricken patients of the dentist sought HIV testing and counseling. Mass hysteria was again aggravated and fueled by the media and newspapers. On July 18, 1991, the US Senate voted 81 to 18 for a Jesse Helms–sponsored proposal that would mandate prison terms of at least ten years and fines of up to $10,000 for health workers who know they have AIDS but fail to inform patients on whom they perform invasive procedures. The Senate then voted 99 to 0 on a leadership bill, directing the states to require health professionals who perform invasive procedures to be HIV tested.[52] This hysterical outburst on the part of the US Senate reflects the irrationality that is rampant throughout the land with regards to the AIDS-HIV epidemic.

The senatorial hysteria may have been fueled in part by the July 1, 1991, *Newsweek* cover story "Doctors With AIDS." In a Gallup poll taken for the magazine, 94% of respondents said all physicians should be required to tell patients if they are infected with HIV, and 65% said they would discontinue all treatment offered by infected health care workers.

The state health departments in New York and Michigan and the city health department in San Francisco announced formal opposition to the CDC quidelines concerning exposure-prone procedures that infected health care workers should not perform. In addition, the ADA and the California Medical Association announced opposition to the guidelines.[53]

The CDC guidelines and agenda proposed by Brennan[54] states that the benefits of screening patients and health care workers are marginal and outweighed by the costs. Nevertheless, given the ethical commitment of health care professionals to patients, the threshold may need to be lower than for patients. A clear definition is needed for "seriously invasive procedures." Hospitals should create policies and make resources available to HIV-positive health care providers. The rights and confidentiality of the provider must be weighed against the patient's need to know.

The right to privacy means the right to refuse HIV testing, but it does not include the right to harm others. The right to confidentiality means the right to withhold disclosure of HIV status but not the right to put others at significant risk. If surgeons are to undergo elective screening to protect their patients, patients should certainly undergo screening to protect their surgeons. The risk of the surgeon acquiring HIV from the patient is much greater than the risk of the patient acquiring it from the surgeon.

Some institutions and medical organizations have not examined in depth the ethical issues involved in dealing with an HIV-infected infected physician, "but have promulgated restrictive, perhaps irrational policies regarding workers with HIV-infection."[54] Health care institutions should take the initiative in designing rational and reasonable policies "to monitor, to detect, and to control the behavior of persons who are, or who might be, HIV-positive."[50] The new CDC guidelines maybe helpful in this regard. Finally HIV-positive physicians must be responsible and ensure their patients' safety. These physicians in turn must be assured that their colleagues will support their responsibility in tangible ways.[50]

Acknowledgment. The authors are indebted to Mrs Ann Amore for typing the manuscript.

REFERENCES

1. Kelly K: AIDS and ethics: An overview. *Gen Hosp Psychiatry* 1987; 331–340.
2. Rosner F, Brennessel DJ: Ethical issues in AIDS and H1V 1nfections, in Rapoza N (ed): *HIV Infection and Disease*. Chicago, AMA, 1989, pp 193–202.
3. Judd, FK, Biggs BA, Burrows GD: Ethical issues and acquired immunodeficiency syndrome (AIDS). *Aus NZJ Psychiatry* 1989; 23;523–528.
4. Lo B: Ethical dilemmas in H1V infection. *J Am Podiat Med Assoc* 1990; 80:26–30.
5. Possible transmission of human immunodeficiency virus to a patient during an invasive dental procedure. *MMWR* 1990; 39:489–493.
6. Update transmission of HIV infection during an invasive dental procedure—Florida. *MMWR* 1991; 40:21–33.
7. Lederman MM, Mehlman MJ: Physicians infected with HIV. *JAMA* 1991; 265:2337–2338.
8. Gramelspacher GP, Miles SH, Cassel CK: When the doctor has AIDS. *J Infect Dis* 1990; 162:534–537.
9. Gostin L: The HIV infected health care professional. Public policy, discrimination and safety. *Arch Int Med* 1991; 151:663–665.
10. Gerberding JL: AIDS and the health care provider; coping with risks, fears and risk reduction strategies, in Rapoza N (ed): *HIV Infection and Disease*. 1989, Chicago, AMA, pp 35–46.
11. Henderson DK, Fahey BJ, Willy M, et al: Risk for occupational transmission of human immunodeficiency virus type 1 (HIV-1) associated with clinical exposures. A prospective evaluation. *Ann Intern Med* 1990; 113:740–746.
12. Gerberding JL, Littell C, Tarkington A, et al: Risk of exposure of surgical personnel to patients' blood during surgery at San Francisco General Hospital. *N Engl J Med* 1990; 322: 1788–1793.
13. Jagger J, Hunt EH, Brand-Elnaggar J, et al: Rates of needle-stick injury caused by various devices in a university hospital. *N Engl J Med* 1988; 319:284–288.
14. Heald AL, Ransohoff DF: Needle-stick injuries among resident physicians. *J Gen Intern Med* 1990; 5:389–393.
15. Klein RS: Universal precautions for preventing occupational exposures to human immunodeficiency virus type 1. *Amer J Med* 1991; 90:141–144.
16. Gerbert B, Maguire B, Badner V, et al: Why fear persists: Health care professionals and AIDS. *JAMA* 1988; 260:3481–3.
17. Brennen TA: Transmission of the human immunodeficiency virus in the health care setting—time for action. *N Engl J Med* 1991; 324:1504–1509.
18. Rizzo JA, Marder WD, Willke RJ: Physician contact with and attitudes toward HIV-seropositive patients: Results from a national survey. *Med Care* 1990; 28:251–60.
19. Zuger A, Miles SH: Physicians, AIDS and occupational risk: Historic traditions and ethical obligations. *JAMA* 1987; 258:1924–1928.
20. O'Connor TW: Do patients have the right to infect their doctor? *Aust NZJ Surg* 1990; 60:157–162.
21. Udell JL: AIDS and the duty to warn. *Health Matrix* 1990; 6:31–35.
22. Mishu B, Schaffner W, Horan JM, et al: A surgeon with AIDS: Lack of evidence of transmission to patients. *JAMA* 1990; 264;467–470.
23. Sacks JJ: AIDS in a surgeon. *N Engl J Med* 1985; 313:1017–1018.
24. Armstrong FP, Miner JC, Wolfe WH: 1nvestigation of a health care worker with symptomatic HIV infection: An epidemiologic approach. *Milit Med* 1987; 152:414–418.
25. Porter JD, Cruickshank JG, Gentle PH, et al: Management of patients treated by surgeon with HIV infection. *Lancet* 1990: 335:113–114.
26. Lettau LA, Smith JD, Williams D, et al: Transmission of hepatitis B with resultant restriction of surgical practice. *JAMA* 1986: 255:934–937.
27. Centers for Disease Control. Hepatitis B among dental patients in Indiana. *MMWR* 1985: 34:73–75.
28. Coutinho RA, van Lent PA, Stootjesdijk L, et al. Hepatitis B from doctors. *Lancet* 1982; 2:345–346.
29. Carl M, Blakey DL, Francis DP, et al: Interruption of hepatitis B transmission by modification of a gynecologist's surgical technique. *Lancet* 1982; 1:731–733.
30. Welch J, Webster M, Tilzey AJ, et al: Hepatitis B infections after gynecological surgery. *Lancet* 1989; 1:205–207.
31. Rhame FS: The HIV infected surgeon. *JAMA* 1990; 264:507–508.
32. Frandzel S: Organizations defend their positions at CDC conference on HIV-infected health care workers. *AIDS Patient Care* 1991; 5:129–133.
33. Staver S: HIV-infected orthopedists urged to curtail surgery. *Amer Med News* March 25, 1991, pp 2, 18.
34. Staver S: Doctors with HIV must avoid some surgeries—CMA. *Amer Med News* April 1, 1991, p 9.
35. Sullivan JF: Judge rules surgeon infected with AIDS must tell patients. *NY Times*, April 26, 1991 pp B1, B2.
36. Kerr P: Test all hospital patients for AIDS, New Jersey doctors urge. *NY Times* May 1, 1991, pp B1, B5.
37. Goldsmith MF: Physicians and dentists tell the CDC: "Avoid quick fix for a tough problem." *JAMA* 1991; 265:1221–1222.
38. New York State Department of Health. Policy Statement and Guidelines. Health Care Facilities and HIV-Infected Medical Personnel. Jan 1991.
39. Whitehead ED: All share duty of disclosing HIV status when appropriate. *Amer Med News*, March 18, 1991, p 25.
40. Knobel GJ: AIDS in dental practice: Hazards, infection control, medico-legal and ethical aspects. *J Forensic Odonto-Stomatology* 1986; 4:15–28.
41. Klein RS, Phelan JA, Freeman K, et al: Low occupational risk of human immunodeficiency virus infection among dental professionals. *N Engl J Med* 1988; 318:86–90.
42. Gerbert B, Bleecker T, Miyasoki C, et al: Possible health care professional-to-patient HIV transmission: Dentists' reactions to a Centers for Disease Control Report. *JAMA* 1991; 265:1845–1848.
43. Barondess JA, Farber SJ, Rogers DE, et al: The risk of contracting HIV infection in the course of health care. *JAMA* 1991; 265:1872–1873.
44. Landesman SH: The HIV-positive health professional: Policy options for individuals, institutions and states. Public policy and the public-observations from the front line. *Arch Int Med* 1991; 151:655–657.
45. Check WA: Experts cite flaws of mandatory HIV testing of health care workers. *ACP Observer* 1991; 11:12–13.
46. Liturn MS: HIV should be issue of health, not politics. *Amer Med News* March 18, 1991, p 21.
47. Bayer R: Public health policy and the AIDS epidemic: An end to H1V exceptionalism? *N Engl J Med* 1991; 324:1500–1504.
48. Aoun H: When a house officer gets AIDS. *N Engl J Med* 1989; 321:693–696.
49. Angell M: A dual approach to the AIDS epidemic. *N Engl J Med* 1991; 324:1498–1500.
50. Jonsen AR: Is individual responsibility a sufficient basis for public confidence? *Arch Int Med* 1991; 151:660–662.
51. Price DM: What should we do about HIV-positive health professionals? *Arch Int Med* 1991; 151:658–659.
52. Tolchin M: Senate adopts tough measures on health care workers. Backs prison and fine for failure to tell patients. *NY Times*, July 19, 1991, pp A1, A14.
53. Staver S: Opposition mounts against CDC plan for "exposure-prone" list. *Amer Med News* Oct 28, 1991, pp 1, 28, 29.
54. Brennan TA: Transmission of the human immunodeficiency virus in the health care setting—time for action. *N Engl J Med* 1991; 324:1504–1509.

Primary and secondary aortoenteric fistula and thoracic aortic aneurysm

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In 1825, Sir Astley Cooper[1] first described a case of aortointestinal fistula as a complication of abdominal aortic aneurysm. Brock[2] in 1953, described the first case of a secondary aortoenteric fistula detected at autopsy after the use of a homograft. In 1957, Heberer[3] reported the first successful management of an aortoenteric fistula. He used an endoaneurysmorraphy technique to treat a saccular aneurysm that had progressed to a fistula. In 1958, McKenzie et al[4] described the first successful repair of a fistula between an aortic homograft and the duodenum. In 1959, Grupska[5] reported the first repair of a fistula between the bifurcation of a Dacron graft and the duodenum. Since then, there have been several reviews of primary aortoenteric fistulas, most of which have been single case reports or studies using small numbers of patients. With the advent of more aggressive surgical management of abdominal aortic aneurysms, the reports of secondary aortoenteric fistulas have been increasing. Both primary and secondary aortoenteric fistula pose diagnostic and therapeutic challenges.

CASE REPORT

A 62-year-old Caucasian man was admitted to Flushing Hospital Medical Center with a history of upper abdominal pain and hematemesis of one week duration. Upper gastrointestinal endoscopy revealed a "greater curvature gastric ulcer." He was placed on an H-2 blocker and followed-up as an outpatient. The next month he was readmitted with hematemesis. A pulsating abdominal mass was palpable. On repeat upper endoscopy "an erosion was seen in the third portion of the duodenum." Within 12 hours, the patient had a bout of severe hematochezia and hypotension. An exploratory laparotomy revealed an infrarenal abdominal aortic aneurysm and an aortoduodenal fistula into the third portion of the duodenum. The aneurysm was resected and replaced with a Dacron graft. The portion of the duodenum involved with the fistula was resected, and an end-to-end duodenoduodenostomy was performed. No omentum was placed between the graft and the duodenoduodenostomy.

The patient had an uneventful postoperative course and was discharged 12 days after surgery. Approximately one year later, he was admitted with left-sided abdominal pain but no evidence of bleeding. A barium enema and aortogram were performed, and both were interpreted as negative. He was discharged, only to be readmitted one month later with severe hematochezia. At laparotomy, an abscess around the proximal aortic suture line was noted with erosion into the transverse colon. The aortic graft was removed; the aortic stump was debrided, closed with polypropylene sutures, and reinforced with a patch of omentum. The defect in the colon was resected and an end-to-end anastomosis was performed. After the abdomen was closed an axillary bifemoral bypass was performed. The patient was eventually discharged without any significant morbidity. Approximately two years later, he underwent resection of a thoracic aortic aneurysm. Subsequently, he moved to Florida and has been lost to follow-up.

DISCUSSION

Aortoenteric fistulas can be classified into two groups: primary and secondary. Primary fistulas arise de novo from several underlying causes, including tuberculosis, and syphilitic and mycotic infections. Since the advent of antibiotic therapy, the majority of cases have been the result of atheromatous degeneration of the infrarenal aorta. Other reported causes have been carcinoma, ulcers, gallstones, diverticulitis, appendicitis, and foreign bodies.[6] Primary aortoenteric fistulas are uncommon, with Bergqvist[7] reporting 249 cases in his review of the world literature, including postmortem cases. Secondary aortoenteric fistulas result from previous aortic surgery and are a well recognized complication of aortic prosthesis; they occur in 0.6%–4.0% of cases.

The pathogenesis of both primary and secondary aortoenteric fistulas depends on the direct adhesion of a segment of intestine to the aneurysm or to the aortic prosthesis. The latter usually occurs at the proximal anastomosis, followed by progressive erosion through the bowel wall and secondary contamination by intestinal flora. The ensuing infection further contributes to necrosis of the adjacent aortic wall or prosthesis suture line, with gastrointestinal hemorrhage as a final consequence. Busutill et al[9] have presented experimental data suggesting that a sufficient concentration of bacteria must be present for a secondary fistula to form. Buchbinder et al[10] and other investigators[11,12] looking at both experimental and clinical conditions, conclude that the pathogenesis of secondary aortoenteric fistulas is still controversial but that both mechanical and infective factors play an important role in their development. Secondary aortoenteric fistulas have been classified further into true fistulas and paraprosthetic fistulas. True fistulas have a complete communication between the lumen of the aorta and the intestine, whereas "paraprosthetic fistulas," a term coined by Elliot et al[13] consists of a communication between the intestine and the external surface of the vascular prosthesis without actual fistulization into the vascular lumen.

Primary aortoenteric fistulas are diferent to diagnose unless suspected. Of the reported cases, less then half had correct diagnoses before surgery.[1] Abdominal and back pain along with gastrointestinal tract bleeding occurred in only 50% of the reported cases.[15] More than 60% of the patients survived their initial bleeding episode, and fewer then 30% died during the first six hours after rupture.[16] The triad of pain, hemorrhage, and a pulsatile mass, although suggestive of such a fistula, is rarely present. Fiberoptic endoscopy is the most useful and frequently recommended initial diagnostic procedure and should be carried down through the third and fourth portions of the duodenum to visualize the mucosal defect associated with a fistula. Although the fistula may not be visualized, the absence of an actively bleeding lesion in the esophagus, stomach, or proximal duodenum, by exclusion, supports the diagnosis of a fistula.[17]

Secondary aortoenteric fistulas are reported more often than primary ones but present a diagnostic challenge. Several reports[18,19] have illustrated the value of computed tomography (CT) in demonstrating the accumulations of fluid and air around grafts, the presence of anastomotic aneurysms, or direct erosion of aortic prostheses into adjacent loops of intestine. Aortog-

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raphy may demonstrate a false aneurysm that was not palpable on physical examination.[20] The findings of extravasated dye in the intestinal lumen, however, is rarely found.[21] Aortography is also important in planning surgical intervention because it may provide information on the need for lower extremity revascularization and/or graft excision. Recently, the use of ^{111}In-labeled leukocyte scanning has been reported to detect perigraft inflammation associated with fistula formation and may be useful when the results of other studies are equivocal. It is useful to remember the premise of Garrett et al[23] who stated that gastrointestinal hemorrhage after aortic surgery is due to an aortoenteric fistula until proven otherwise.

In a large series of secondary aortoenteric fistulas reviewed,[24] 30% of patients presented with fever, 26% with gastrointestinal bleeding, 22% with fever and bleeding, and 4% with graft failure. The so-called "herald bleed" of aortoenteric fistula has been reported in only 20% of patients in several large series. Other patients have presented with recurrent fever and chills of unknown origin, groin abscess, and renal failure. Martin and Coperman[25] emphasized the importance of septic emboli to the skin of the legs, whereas Becker et al[26] reported a patient with an infected aortic graft who had peticheal rash and increased temperature of one leg. Obviously, a high index of suspicion is required for correct diagnosis using a combination of endoscopy, CT scans, and angiography endoscopy being primarily of use in ruling out other sources of bleeding.

The treatment of primary aortoenteric fistulas has ranged from the early attempts at aneurysm resection, intestinal repair, and extra-anatomic bypass to, more recently, aortic reconstruction with prosthesis and intestinal repair. The more recent literature suggests that primary aortoenteric fistula is best treated by resection of the aneurysm with graft replacement and closure of the intestinal defect, followed by the interposition of a vascularized pedicle of omentum between the intestinal and the vascular anastomoses.[27,28] The recommendation for the use of antibiotics both during and after surgery and in long-term postoperative therapy, seems to be on the basis of the results of intraoperative cultures of the aortic wall. Macbeth et al[29] recommend that aortic wall and periaortic debridement be done to decrease contamination, since when aortic wall cultures were positive, suture line disruption occurred 57% of the time compared with no disruptions when the cultures were negative. Sweeney and Gadacz[30] recommended 4–6 weeks of antibiotic therapy if the intraoperative cultures are positive.

Operations for secondary aortoenteric fistulas can be divided into two groups on the basis of whether the aortic prostheses are removed or replaced. Several sources[31-34] recommend total removal of the previous graft, oversewing the aortic stump, and extra-anatomic bypass. Although this radical procedure is routinely recommended, there is significant in-hospital mortality, ranging upward from 50%,[34] and morbidity, with the incidence of aortic stump disruption as high as 33%[34] and of limb amputation up to 25%.[34] This had led some groups to look for alternative approaches. Walker et al[24] reported on 23 patients treated for secondary aortoenteric fistulas by excision of grafts with replacement, intestinal repairs, and insertion of pedicles of omentum between the intestinal and vascular anastomoses, with only a 17% incidence of graft-related problems. There were five hospital deaths, none being graft-related. Three patients developed false aneurysms, suffered additional hemorrhagic events, and died. A recent report by Thomas and Baird also supported the plan for graft replacement, which was successfully carried out on four patients. Several other reports on the use of graft replacement,[36,37] however, have not had the same success rate as that reported by Walker et al.[24]

Harris et al[38] reported on 14 patients managed by various methods. Five were treated by direct repairs and insertion of new grafts, four died in the immediate postoperative period, and five were treated with graft removal and extra-anatomic bypass resulting in a 60% short-term survival and 40% long-term survival.

Several factors seem to have an impact on the success of graft replacement. In one of the early reports, Elliot et al[13] pointed out that overt retroperitoneal sepsis, positive intraoperative cultures, and positive femoral artery blood cultures with negative venous blood cultures are clear indications for graft removal and extra-anatomic bypass. More recent reports have emphasized other factors, as well, and have included complete coverage of the retroperitoneal vascular anastomosis with a vascularized piece of omentum to separate it completely from the intestinal anastomosis or repair.

With the increasing number of operations for abdominal aortic aneurysms, it is likely that secondary aortoenteric fistulas will continue to occur even with the most meticulous techniques. Both require a high index of suspicion for diagnosis and individualized surgical approaches. The recommended management for primary aortoenteric fistula is less controversial and consists of resection of the aneurysm and graft replacement, repair or resection of the intestine, and omental interposition to separate completely the vascular and intestinal anastomoses.

Secondary aortoenteric fistulas are difficult to diagnose and equally difficult to cure. The diagnosis may depend on only clinical suspicion in patients with gastrointestinal hemorrhage after aortic surgery, or on the use of upper gastrointestinal endoscopy, CT scanning, aortography, and even ^{111}In scanning. The final diagnosis may be made only at laparotomy. There are a number of options for the management of secondary aortoenteric fistulas: removal of the prosthesis, intestinal repair, and extra-anatomic bypass; replacement of the prosthesis, intestinal repair, and omental interposition; and in rare cases, local repair of the fistula with omental interposition. The overall mortality remains high, and there is no single surgical approach that is clearly most successful. The more recently introduced graft replacement and intestinal repair with omental interposition may be preferable to extra-anatomic bypass due to a seemingly lower mortality. The patient that we describe here did not have omental interposition after the first repair. Perhaps if he had, the secondary aortoenteric fistula would not have occurred.

Acknowledgment. The authors thank Ms Dorothy Bellere for her assistance in the preparation of this manuscript.

REFERENCES

1. Cooper A: *Lectures on the principles and practice of surgery with additional notes and cases by F. Tyrue,* 5th ed. Philadelphia, Haswell, Barrington and Haswell, 1839.
2. Brock RC: Aortic homografting: A report of six successful cases. *Guys Hosp Rep* 1953; 102:204–228.
3. Heberer A: Diagnosis and treatment of aneurysms of the abdominal aorta. *Germ Med Mon* 1957; 2:203.
4. McKenzie RJ, Buell AH, Pearson SC: Aneurysms of an aortic homograft with rupture into the duodenum. *Arch Surg* 1958; 77:965.
5. Case record of the Massachusetts General Hospital (case 45522). *N Engl J Med* 1959; 261:1339.
6. Steffes BC, O'Leary JP: Primary aortoduodenal fistulas: A case report and review of the literature. *Am Surg* 1980; 46:121–129.
7. Bergqvist D: Aortoenteric fistula. *Acta Chir Scand*. 1987; 153:81–86.
8. Bunt TJ: Synthetic vascular graft infections. II. Graft-enteric erosions erosions and graft-enteric fistulas. *Surgery* 1983; 94:1.
9. Busutill RW, Rees W, Baker JD, et al: Pathogenesis of aortoduodenal fistula. Experimental and clinical correlates. *Surgery* 1979; 85:1–13.
10. Buchbinder D, Leather R, Shah D, et al: Pathologic interactions between prosthetic aortic grafts and the gastrointestinal tract: Clinical problems and a new experimental approach. *Am J Surg* 1980; 140:192–198.
11. Humphries AW, Young JR, DeWolfe VG, et al: Complications of abdominal aortic surgery. *Arch Surg* 1963; 86:57.
12. Shaigany A, Gillespie L, Mock JP, et al: Aortoenteric fistula: A complication of renal artery bypass graft. *Arch Intern Med* 1976; 136:930.
13. Elliot JP, Smith RF, Szilagyi DE: Proceedings: Aortoenteric and paraprosthetic enteric fistulas: Problems of diagnosis and management. *Arch Surg* 1974; 108:479–490.
14. Brenowitz JB, Williams ED: Successful repair of a primary aortoduodenal fistula. *Am Surg* 1976; 42:265–267.
15. Reckless JP, McColl I, Taylor GW: Aortoenteric fistulae: An uncommon complication of abdominal aortic aneurysm. *Br J Surg* 1972; 59:458.
16. Dinapoli RP, Schirger A, Kirblin JW: Abdominal aortic aneurysm with bleeding into the gastrointestinal tract. *Mayo Clin Proc* 1964; 37:354–357.
17. Mir-Madjlessi SH: Endoscopic diagnosis of aortoduodenal fistula. *Gastrointest Endosc* 1973; 19: 187–188.
18. Lineaweaver WC, Clore F, Alexander RH: Computed tomographic diagnosis of acute aortoiliac catastrophies. *Arch Surg* 1982; 117:1095–1097.

19. Kukora JS, Rushton FW, Cranston PE: New computed tomographic signs of aortoenteric fistula. *Arch Surg* 1984; 119:1073–1075.

20. Pelz DM, Rankin RN: Alternate bleeding sites in suspected graft-enteric fistula. *AJR* 1981; 136:707–709.

21. Kleinman LH, Towne JB, Bernhard VM: A diagnostic and therapeutic approach to aortoenteric fistulas: Clinical experience with 20 patients. *Surgery* 1979; 86:868–877.

22. Lawrence PF, Dries DJ, Alazraki N: Indium 111 labeled leukocyte scanning for detection of prosthetic graft infections. *J Vasc Surg* 1985; 2:165–173.

23. Garrett HE, Beall AC, Jordon GL, et al: Surgical considerations of massive gastrointestinal tract hemorrhage caused by aortoduodenal fistula. *Am J Surg* 1963; 105:6–12.

24. Walker WE, Cooley DA, Duncan JM, et al: The management of aortoduodenal fistula by in-situ replacement of the infected abdominal aortic graft. *Ann Surg* 1987; 205:727–732.

25. Martin A, Coperman PWM: Aorto-jejunal fistula from rupture of teflon graft with septic emboli in the skin. *Br Med J* 1967; 2:155–156.

26. Becker RM, Cockroft D, Poirier NL: Excision of infected aortic bifurcation graft and axillofemoral bypass: Case report. *Surgery* 1975; 77:718–721.

27. Pfeiffer RB Jr: Successful repair of three primary aortoduodenal fistulas. *Arch Surg* 1982; 117: 1098–1099.

28. Daugherty M, Shearer R, Ernst CB: Primary aortoduodenal fistula: Extra anatomic vascular reconstruction not required for successful management. *Surgery* 1979; 86:399–400.

29. Macbeth GA, Rubin JR, McIntyre KF, et al: The revelance of arterial wall microbiology to the treatment of prosthetic graft infection: Graft infection vs. arterial infection. *J Vasc Surg* 1984; 1:750–756.

30. Sweeney MS, Gadacz TR: Primary aortoduodenal fistula: Manifestation, diagnosis and treatment. *Surgery* 1984; 96:492–496.

31. Bernhard VM, Towne JB (eds): *Complications in Vascular Surgery*. New York, Grune and Stratton, 1980, pp 517–530.

32. Najarian JS, Delaney JP (eds): *Advances in Vascular Surgery*. Chicago, Year Book Medical Publishers, 1983, pp 156–157.

33. Reilly LM, Altman H, Tubsy RJ: Late results following surgical management of vascular graft infections. *J Vasc Surg* 1984; 1:36–44.

34. O'Hara PJ, Hertzer NR, Beven EE, et al: Surgical management of infected abdominal aortic grafts: Review of a 25 year experience. *J Vasc Surg* 1986; 86:725–731.

35. Perdue GD, Smith RB, Ansley JD, et al: Impending aortoenteric hemorrhage: The effect of early recognition on improved outcome. *Ann Surg* 1980; 192:237–243.

36. Harris JP, Sheil AG, Stephen MS, et al: Lessons learnt in the management of aortoenteric fistulae. *J Cardiovasc Surg* 1987; 28:449–452.

Metastatic hemangiopericytoma of the breast

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Hemangiopericytoma is an uncommon vascular tumor first described in 1942 by Stout and Murray.[1] The tumor is believed to arise from pericapillary contractile cells, the pericytes of Zimmerman. The tumor most often involves the musculoskeletal system and skin, especially of the lower extremities.[2,3] Less common sites of involvement include the pelvic fossa, retroperitoneum, lung, central nervous system, and bowel. Metastases can occur by both lymphatic and hematogenous routes. Lung and skeleton are the most frequently reported sites of metastases.[3]

The breast is a rare site for primary hemangiopericytoma; 15 cases have been reported previously.[4–10] Metastatic hemangiopericytoma of the breast has been documented only twice.[11,12] We report an additional instance of metastatic hemangiopericytoma of the breast discovered by routine mammography and diagnosed by stereotaxic, fine-needle aspiration biopsy. The patient had a previous resection of a hemangiopericytoma of the buttock.

CASE REPORT

A 41-year-old woman was found to have a palpable, right breast mass in 1982. Removal of the mass led to a diagnosis of fibroadenoma.

Three years later, the patient presented with an 11.0-cm mass in the right buttock. The mass was noted to be warm to the touch and exhibited several superficial distended veins. A vascular soft tissue lesion was suspected, and a pelvic arteriogram was performed to define the vascularity. Feeding vessels from the inferior gluteal and circumflex femoral arteries were identified, and the lesion was characterized as a hemangioma on the basis of radiologic findings. The feeding vessels were embolized with gelfoam in anticipation of excision of the mass. Wide excision of the mass was performed, and a diagnosis of hemangiopericytoma was made. The tumor had focal areas of pleomorphism and hypercellularity and was therefore considered to have malignant potential. The patient's regular follow-up showed no evidence of local recurrence.

Four years later, the patient had a routine mammogram using a low-dose technique, disclosing a 2.4-cm diameter nodule in the upper outer quadrant of the left breast. This mass was not present on a mammogram performed two years earlier (Fig 1). No mass was palpable in either breast, and there was no axillary adenopathy. A stereotaxic, fine-needle aspiration biopsy of the nodule was performed and was diagnosed as a hemangiopericytoma.

A chest film and computed tomography scans of the chest, abdomen, pelvis, and brain showed no evidence of tumor. Liver function tests and bone scan using ^{99M}Tm methylene diphosphonate were normal. The breast mass was widely excised after mammographic wire localization.

Smears obtained from the stereotaxic, fine-needle aspiration showed dispersed blood and multiple fragments of a cellular, spindle-cell neoplasm composed of cells with ovoid nuclei, inconspicuous nucleoli, and delicate cytoplasm having ill-defined borders. The neoplasm showed abundant small vessels lined by endothelial cells predominantly obscured by the neoplastic cells. Mitotic figures were seen. The findings were consistent with a diagnosis of malignant hemangiopericytoma.

Gross inspection of the excised mass revealed a soft, pink, focally hemorrhagic tumor measuring 2 × 1.7 × 1.2 cm (Fig 2). It was well-delineated from the surrounding adipose tissue by a thin capsule. On histologic evaluation, the tumor was markedly cellular and composed of numerous endothelial-lined channels surrounded by irregularly proliferating, closely packed neoplastic pericytes, with plump, oval- to spindle-shaped nuclei and inconspicuous nucleoli (Fig 3). The vascular spaces were compressed and poorly outlined. There were 2–3 mitotic figures/10 high-power fields, and there was focal microcystic degeneration. Necrosis was not seen.

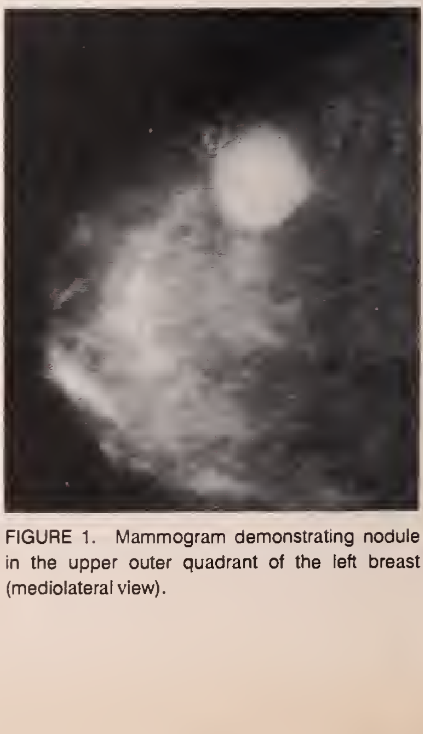

FIGURE 1. Mammogram demonstrating nodule in the upper outer quadrant of the left breast (mediolateral view).

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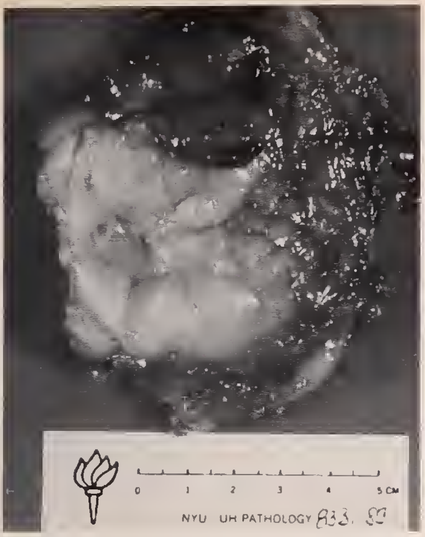

FIGURE 2. Cut section of tumor in the upper part of the specimen is distinct from the adjacent breast tumor.

The histologic characteristics of the previously excised tumor of the right buttock were reviewed. The tumor was located in the subcutis and had been completely excised. The lesion was round, measured 6.5 cm in diameter, and was covered by a thin capsule. Cross sections revealed a soft, pink tumor with occasional slit-like empty vascular spaces, blood-filled vessels, and strands of fibrous tissue. It was associated with the wall of a blood vessel. Microscopic analysis confirmed the diagnosis of malignant hemangiopericytoma with findings similar to those found in the breast mass; however, the buttock mass was less cellular and had few if any mitotic figures (Fig 4). In spite of the low mitotic rate, focal areas of pleomorphism and hypercellularity were felt to be indicative of malignant potential.

DISCUSSION

In 1942, Stout and Murray[1] first described hemangiopericytoma as a tumor derived from capillary pericytes and characterized by endothelial tubes with surrounding rounded or elongated cells and a supporting meshwork of reticulin fibers. Hemangiopericytoma has since become a well-recognized tumor, and by 1969, 224 cases had been reported.[3]

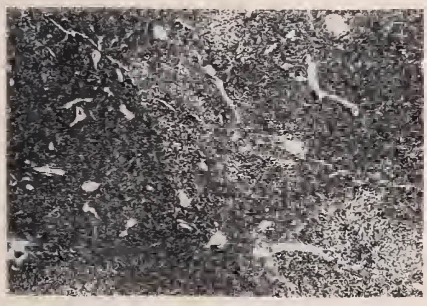

FIGURE 3. The hemangiopericytoma is histologically characterized by small blood vessels surrounded by neoplastic pericytes (hematoxylin and eosin stain; original magnification × 100).

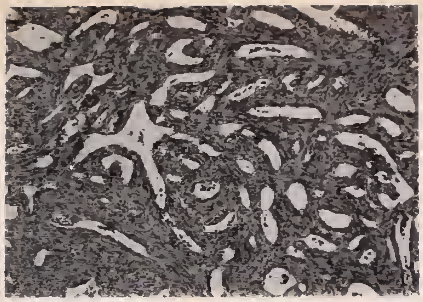

FIGURE 4. The primary hemangiopericytoma of the buttock demonstrates blood vessels surrounded by neoplastic pericytes (hematoxylin and eosin stain; original magnification × 100).

The reported median age of patients with hemangiopericytoma is the fifth decade, with ages ranging from birth to 92 years.[3] The tumor usually presents as a painless, enlarging mass.[13] The musculoskeletal system and skin (especially of the lower extremities), retroperitoneum, and pelvis are the most common sites of occurrence.[2,3,13] Hemangiopericytoma can reach a very large size, with tumors up to 25 cm in greatest dimension and 1,800 g in weight having been reported.[14] On gross examination, they may be firm, rubbery, soft, or friable and usually have a well-defined capsule. Hemangiopericytomas are histologically characterized by tightly packed, plump spindle cells, the pericytes of Zimmerman, and surrounding thin-walled vascular spaces. Enzinger and Smith[2] have described histologic criteria of malignancy for these tumors. Increased cellularity, mitotic activity (4 mitotic figures/10 high-power fields), and foci of necrosis or hemorrhage are characteristic findings.

The most common sites of metastases are lung and skeleton.[2] Up to 79% of histologically malignant tumors metastasize, and the overall incidence of metastasis is 50%.[13] Local recurrence is related to the site and grade of the primary tumor. Tumors within the central nervous system are among those with the highest morbidity, with a recurrence rate of 80%.[3] This likely reflects inadequate excision of the primary tumor.

Wide excision is the treatment of choice for hemangiopericytomas.[2,3] Amputation may be necessary in some circumstances to achieve adequate surgical margins. High-dose radiation therapy causes some tumor regression and has been recommended as an adjunct to surgical excision.[15] The use of chemotherapy, particularly doxorubicin, has produced remission in many instances of metastatic hemangiopericytoma.[16]

Long-term survival is related to tumor histology and size. The ten-year survival rate is 77% for tumors with benign features (>3 mitotic figures/10 high-power fields), and 29% for tumors with malignant features (four or more mitotic figures; tumor necrosis). Tumors smaller than 6.5 cm are associated with a ten-year survival rate of 92%, whereas the rate is 63% for tumors larger than 6.5 cm.[2]

Fifteen primary hemangiopericytomas of the breast have been reported.[4–10] These tumors characteristically presented as circumscribed, slow-growing solitary masses. Wide excision was the usual treatment, except for particularly large tumors, which were treated by mastectomy. Primary hemangiopericytomas of the breast are low-grade tumors, and neither local recurrence nor distant metastases have been described.[4]

Metastatic hemangiopericytoma of the breast has been reported twice, with the orbit and retroperitoneum being the primary sites of malignancy.[11,12] The orbital tumor metastasized to the breast one year after primary excision, and the retroperitoneal tumor did so after six years. Both metastatic breast tumors were treated by local excision.

Stereotaxic, fine-needle aspiration biopsy has become a valuable tool in the diagnosis of nonpalpable breast masses discovered by mammography. Stereotaxic localization is performed by taking multiple radiographic images of the lesion from different standard angles and plotting the coordinates of the lesion in three dimensions. Fine-needle aspiration biopsy of the lesion is then performed. Dowlatshahi et al[17] studied 528 breast masses using this technique and reported a 95% sensitivity and 91% specificity rate.

The case presented here represents the third reported metastatic hemangiopericytoma to the breast. Unlike the previously reported primary and metastatic hemangiopericytomas of the breast, this lesion was a nonpalpable mass discovered by routine mammography. Stereotaxic, fine-needle aspiration biopsy confirmed the diagnosis, and the patient underwent wide excision of the breast tumor.

REFERENCES

1. Stout AP, Murray MR: Hemangiopericytoma: A vascular tumor featuring Zimmerman's pericytes. *Ann Surg* 1942; 116:26–33.

2. Enzinger FM, Smith BH: Hemangiopericytoma: An analysis of 106 cases. *Hum Pathol* 1976; 7: 61–82.

3. Backwinkel KD, Diddams JA: Hemangiopericytoma: Report of a case and comprehensive review of the literature. *Cancer* 1970; 25:896–901.

4. Arias-Stella Jr J, Rosen PP: Hemangiopericytoma of the breast. *Mod Pathol* 1988; 1:98–103.

5. Callery CD, Rosen PP, Kinne DW: Sarcoma of the breast. *Ann Surg* 1986; 201:527–532.

6. Donnell RM, Rosen PP, Lieberman PH, et al: Angiosarcoma and other vascular tumors of the breast. *Am J Surg Pathol* 1981; 5:629–642.

7. Kaufman SL, Murray MR: Hemangiopericytoma in children. *Cancer* 1960;13:695–710.

8. Khushbakhat R, Mittal KR, Gerald WR, et al: Hemangiopericytoma of the breast: Report of a case with ultrastructual and immunohistochemical findings. *Hum Pathol* 1986; 17:1181–1183.

9. Tavassoli FA, Weiss S: Hemangiopericytoma of the breast. *Am J Surg Pathol* 1981; 5:745–752.

10. Volmer J, Pickarz H, Javtzke G: Vascular tumor in the region of the breast. *Virchows Arch [A]* 1980; 385:201–214.

11. Panda A, Dayal Y, Singhal V, et al: Haemangiopericytoma. *Br J Ophthalmol* 1984; 68:124–127.
12. Kindblom L-G, Ullman A: Malignant hemangiopericytoma with admixed glandular structures in breast and lung metastases. *Appl Pathol* 1983; 1:50–59.
13. McMaster MJ, Soule EH, Ivins JC: Hemangiopericytoma: A clinicopathologic study and long-term followup of 60 patients. *Cancer* 1975; 36:2232–2244.
14. Gerner RE, Moore GE, Pickren JW: Hemangiopericytoma. *Ann Surg* 1974; 179:128–132.
15. Mira JG, Chu FC, Fortner JG: The role of radiotherapy in the management of malignant hemangiopericytoma. *Cancer* 1977; 39:1254–1259.
16. Wong PP, Yagoda A: Chemotherapy of malignant hemangiopericytoma. *Cancer* 1978; 41:1256–1260.
17. Dowlatshahi K, Gent HJ, Schmidit R, et al: Nonpalpable breast tumors: Diagnosis with stereotaxic localization and fine-needle aspiration. *Radiology* 1989; 170:427–433.

Spontaneous rupture of the spleen as the initial manifestation of acute myeloid leukemia

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Nontraumatic rupture of the spleen is a well recognized but rare complication of hematologic malignancies. Rokitansky,[1] in 1861, was the first to describe splenic rupture in a patient with leukemia. Since then, only 40 cases have been described in the English literature.[2] As an initial manifestation, splenic rupture is even less common, and rarer still in acute leukemias, for which only five cases have been reported with splenic rupture as the presenting manifestation.[4,5,6] To date, no more than 15 cases of splenic rupture have been reported with acute myeloid leukemia (AML).[3,7,10] We report a case of AML presenting as spontaneous rupture of the spleen.

CASE REPORT

A 61-year-old woman with no medical history of note arrived in the emergency department with the complaints of malaise, dizziness, cold and clammy skin, and vague abdominal discomfort of a 24-hour duration. Her blood pressure was 70-mm Hg systolic; heart rate was 94 beats/min; respiratory rate was 20/min; and temperature was 100.1 °F. She had scattered ecchymoses on her extremities. Her abdomen was soft with bowel sounds present, and there was mild epigastric tenderness. There was no rebound or guarding. Rectal examination was negative for occult blood. Her white blood cell count was 58×10^3 μL with 6% blasts; hemoglobin was 11.8 g/dL; platelets were 35×10^3 μL. Bone marrow aspiration and biopsy in the emergency department revealed 90% cellularity, mostly myeloblasts with an myeloid-erythroid ratio of 10:1.

The patient, suspected of having sepsis, received vigorous fluid hydration. She was placed on triple antibiotic therapy consisting of intravenous mezlocillin, 3 g every 4 hours; intravenous gentamicin, 60 mg every 12 hours after a loading dose of 100 mg; intravenous oxacillin, 1 g every 4 hours; and was started on oral allopurinol by mouth, 300 mg daily. Within 24 hours, her hemoglobin dropped to 7.4 g/dL and platelets dropped to 28,000 μL. She went into acute respiratory distress and required intubation. Her prothrombin time was 17 seconds (control, 11.7 sec); partial thromboplastin time was 36 seconds (control, 31 sec); fibrin degradation products were >40 mg/dL; and fibrinogen, 95 mg/dL (normal, 200–400 mg/dL). Her arterial blood gases revealed a metabolic acidosis with a pH of 6.87. She was given aggressive fluid hydration, fresh frozen plasma, platelets, and bicarbonate. She remained hypotensive and died after resuscitative efforts failed.

The autopsy revealed a soft, moist spleen weighing 350 g with a tear in the lower pole. There was 3,000 mL of blood effusion in the peritoneal cavity. The other organs were intact. Microscopic study showed leukemic infiltration into the spleen and several other organs.

DISCUSSION

Nontraumatic rupture of the spleen in acute leukemia is a rare event with an incidence of 0.72%.[2] It frequently goes unrecognized and results in a 100% mortality without surgical intervention. With surgery, the success rate approaches 78% for the immediate postoperative period.[3]

In 1964, Hynes et al[11] described the following mechanisms as possible causes of splenic rupture in leukemia: leukemic infiltration of the splenic capsule, splenic infarction leading to capsular hemorrhage, and coagulopathy in leukemia. A definite cause has not been found because splenic involvement in hematologic malignancies is common, but splenic rupture is rare.[3] The size of the spleen, type of underlying malignancy, and age and gender of the patient do not seem to have any predictive value. Interestingly, splenic rupture has not been described in patients in complete remission.

There are no specific signs or symptoms pathognomonic for splenic rupture. Hypotension, tachycardia, tachypnea, abdominal tenderness and rigidity, shifting dullness, shoulder pain (Kehr's sign), nausea and vomiting, dizziness, and syncope may all occur independently or in combination.[2] Fever may or may not be present and the vital signs may be normal.[3] The only symptom that seems to be consistently present is abdominal pain.[2] However, it is not always severe, acute, or localized. It may easily be confused with abdominal aortic aneurysm, pancreatitis, perforated viscus, biliary tract disease, small bowel obstruction, mesenteric thrombosis, and splenic abscess.[3,12] Theoretically, it may also be masked in patients experiencing gastrointestinal toxicity from chemotherapeutic agents.

The diagnosis usually requires a high index of suspicion because the clinical picture is often blurred by coexisting clinical problems (ie, sepsis). The confirmatory studies are usually radiologic (eg, abdominal films, nuclear scanning, abdominal sonography, computed tomography). Paracentesis is a very effective diagnostic procedure to show intra-abdominal hemorrhage[3] but is not always safe in a patient who may be thrombocytopenic or exhibiting coagulation defects.

This patient's abdominal pain did not progress beyond a mild and vague discomfort. In addition, the initial presentation of hypotension and fever without obvious bleeding or trauma suggested a diagnosis of sepsis. Although the possibility of a splenic rupture was suspected and a liver-spleen scan was ordered, it was considered a remote possibility and the urgency of the situation was not recognized.

In conclusion, in leukemic patients not in remission, the differential diagnosis of any abdominal pain should include the possibility of splenic rupture. The abdominal examination, vital signs, and hematocrit should be closely monitored. Although splenic rupture is a rare complication of leukemia, it is universally fatal unless suspected and treated.

REFERENCES

1. Rokitansky KP: Zwei falle von spontaner zerreissung der milz der deuesten zeit. Wohenblatt der zeitcchrift der K.K. *Gensellschaaft der Aerzte in Wein* 1961; 17:42–44.
2. Canady MR, Welling RE, Strobel SL: Splenic rupture in leukemia. *J Surg Oncol* 1989; 41:194–197.
3. Bauer TW, Haskins GE, Armitage J: Splenic rupture in patients with hematologic malignancies. *Cancer* 1981; 48:2729–2733.

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4. Wolfson IN, Croce EJ, Fite FK: Acute leukemia with rupture of the spleen as the initial symptom. *N Engl J Med* 1954; 251:735–737.
5. Salers JG: Acute leukemia presenting as a ruptured spleen: Report of a case. *Rocky M Med J* 1977; 74:319–320.
6. Johnson CS, Rosen PJ, Sheehan WW: Acute lymphocytic leukemia manifesting as splenic rupture. *Am J Clin Pathol* 1979; 72:118–121.
7. Sarode R, Varma SC, Sakhuja U, et al: Spontaneous rupture of the spleen in acute myeloid leukemia. *J Assoc Physicians India* 1988; 6:4000.
8. Yin JA: Spontaneous splenic rupture in acute monocytic leukemia. [letter] *Eur J Haematol* 1988; 40:279.
9. Wong P, Takabayashi K, Sugiura Y, et al: Splenic rupture in acute megakaryoblastic leukemia. *Jpn J Med* 1987; 26:234–236.
10. Sonobe H, Uchida H, Doi K, et al: Spontaneous rupture of the spleen in acute myeloid leukemia. *Acta Pathol Jpn* 1981; 31:309–315.
11. Hynes HE, Silverstein MN, Fawcett KJ: Spontaneous rupture of spleen in acute leukemia. *Cancer* 1964; 17:1356–1360.
12. Hawkins JA, Mower WR, Nelson, FW: Acute abdominal conditions in patients with leukemia. *Am J Surg* 1985; 150:739–742.

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THE ABBOTT TREATMENT OF SCOLIOSIS

Scoliosis is of two main types, functional and fixed. In the first the spine is flexible, and can be placed in the normal or overcorrected position by a voluntary muscular action of the patient. It is cured by the development of musculature sufficient to maintain the normal poise. With this type we shall not deal except inasmuch as it is the ideal sought in the handling of the second type—the fixed scoliosis. Here we have a spine which cannot be made to take a normal position by muscular action. Beyond the mildest degrees, the power necessary to place it in the normal position may vary from a comparatively small amount of properly applied pressure to an amount of force the patient cannot endure.

In 1912 E. G. Abbott announced that he had developed a method by which the deformity of fixed lateral curvature could be over-corrected rapidly and safely. The keystone of his method, gained from experimental work on the cadaver and the living subject, is the application of the correcting forces with the patient in a flexed position. In flexion, the spinal articular processes are unlocked and the position of the vertebrae can be more readily changed; whereas, in extension the articular processes are locked and the vertebrae are held more rigidly. Fixed lateral curvature is an exaggeration of a position of flexion and side-bending, the early stages of which all of us can and do assume every day. Applying the same reasoning which we daily employ in the reduction of dislocations, it is readily seen that the deformity can best be corrected by forcing its parts to re-travel the path of its development. Having placed the patient in a flexed position, for which an anaesthetic is not necessary or desirable, the correcting forces are applied to the points on which they are to act by means of traction bandages. The high shoulder is pulled downward and backward, the low shoulder upward and forward; the high hip downward and backward, the low hip upward and forward. The convex deformity of the chest is pulled laterally toward the concave side, and at the same time backwards, increasing flexion.

The thighs are flexed on the abdomen to increase the flexion of the spine. The hollowed-out concave side of the chest is filled with thick felt pads. Over all a heavy plaster jacket is applied. In this jacket a large window is cut over the concave side of the chest, and small windows are cut in the front and opposite side of the jacket through which felt pads can be applied, making pressure backwards on the anterior deformity of the chest and laterally against the convexity of the deformed chest. In other words, the idea is to force by a combination of pressure and chest movements the deformed vertebrae and ribs to re-travel the path of development of the *deformity.* The huge window opposite the concave side of the chest allows freedom for this change. In working out the latter part of this method, Abbott took long-established methods and modified them as it seemed necessary.

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HOWARD L. PRINCE, MD
(*NY State J Med* 1913; 13:335–337)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy

TO THE EDITOR: The treatment of fungal peritonitis with amphotericin B without catheter removal has yielded poor results, and the recommendation has been that catheter removal should be followed-up by 10–14 days of amphotericin B therapy.[1] We describe a patient in whom early catheter removal after recognition of fungal peritonitis led to microbiological and clinical resolution without amphotericin B treatment.

Case Report. A 70-year-old black man presented to the emergency room with lethargy and progressive weakness and was found to have renal failure. He was cachectic with bitemporal wasting and normal vital signs. Initial laboratory values were sodium 129 mEq/L, potassium 7.9 mEq/L, chloride 109 mEq/L, CO_2 11 mEq/L, glucose 123 mg/dL, blood urea nitrogen 145 mg/dL, creatinine 12 mg/dL, hemoglobin 8.3 gm/dL. He was started on peritoneal dialysis and was getting 1.5 L of 2.5% dianeal solution per exchange and four exchanges a day. A bone marrow biopsy revealed stage four multiple myeloma. His course was complicated by a bout of *Acinetobacter calcacaeticus* peritonitis which responded to intraperitoneal and intravenous antibiotic therapy. Two months after peritoneal dialysis was initiated, he became febrile and developed abdominal pain with mild diffuse tenderness. The peritoneal fluid cell count was consistent with peritonitis, and he was started on intraperitoneal vancomycin (1 gm loading, 15 mg/L maintenance) and gentamicin (80 mg loading, 8 mg/L maintenance). Cultures of peritoneal dialysis fluid were negative for bacteria, but two cultures taken during a three day period were positive for *Candida albicans.* The Tenckoff catheter was removed, and the antibiotics stopped. The patient became afebrile and his abdominal pain resolved within 24 hours. Subsequent cultures were negative for *C albicans*, and he was switched to hemodialysis. After the creation of a permanent arteriovenous graft, the patient was discharged. He has remained well and is currently hemodialysed in an ambulatory dialysis unit.

Discussion. Fungal peritonitis accounts for a small percentage of all continous ambulatory peritoneal dialysis peritonitis, but the morbidity and mortality is high.[2] Early catheter removal without treatment with antifungal agents has been previously described.[3] Apparently, clinicians have been discouraged from that practice because of the high mortality rate of untreated *Candida* peritonitis.[4] Our patient, although febrile and suffering abdominal pain during his bout of peritonitis, did not appear toxic or unstable. This encouraged us to withhold amphotericin B, a well-known toxic drug, while monitoring him closely. This suggests that it may be possible to achieve complete resolution in some cases of fungal peritonitis with just catheter removal, especially if the situation is recognized early in a stable patient. This would be highly desirable in situations such as acute reversible renal failure where further renal insult needs to be avoided.

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1. Keane WF, Everett ED, Fine RN, et al: Continuous ambulatory peritoneal dialysis (CAPD) peritonitis treatment recommendations: 1989 update. *Peritoneal Dialysis International* 1989; 9:247–256.

2. Bint AJ, et al: Diagnosis and management of peritonitis in continous ambulatory peritoneal dialysis. *Lancet* April 11; 1987; 845–848.

3. Mandell IN, Ahem MJ, Kliger VT, et al: Candida peritonitis complicating peritoneal dialysis: Successful treatment with low dose amphotericin B therapy. *Clin Nephrol* 1976; 6:492–496.

4. Reeves O, Ripepi A, Carter RE, et al: Candida peritonitis in a quadriplegic: Treatment with amphotericin B. *South Med J* 1972; 65:325–328.

How we tell our doctors about pain

TO THE EDITOR: I am a rheumatologist in Rochester, NY. I have just finished reading Jacqueline Galloway's special article in the September 1991 issue of the *Journal* entitled, “How we tell our doctor about pain.”[1] I think it is the most sensitive, realistic, and useful article that I have ever read on chronic pain, coping with it, and relating it to the physician. I am extremely grateful for it, and we have posted it in our patient waiting room. If the author has written any other articles on this topic or, if she is available for speaking engagements, I would be very interested.

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1. Galloway J: How we tell our doctor about pain. *NY State J Med* 1991; 91:399–400.

The physician and the poor

TO THE EDITOR: In the November 1991 issue of the *Journal*, Dr Edgar M. Housepian unjustly criticizes Dr Eli Ginzberg, a most thorough and honest observer, for proclaiming that we have an overproduction of physicians and that this is costly to our health care system.[1]

Of course, we have a surplus, and it is increasing each year. It is, along with lack of primary care physicians, overspecialization, and the exorbitant reimbursement for technical procedures, a major cause of our escalating health care costs. Dr Ginzberg and most careful analysts have contended this for years.[2] In 1985, the American Medical Association recognized a surplus but did little or nothing about it. Dr John K. Iglehart, another responsible analyst, comments that “most Western nations have a common problem . . . they are training more physicians than they seem prepared to accommodate, but few have decided how many physicians are enough.[3]

We license more physicians than retire each year, and in spite of a modest decline, this situation will persist well into the future. In the year 2010, it is estimated that there will still be a net increase of over 3,000 annually.

Before Congress initiated an increase in physicians in the 1960s (in a misguided attempt to pressure physicians to practice in less desirable locations), physicians easily achieved their financial goals. Now, meeting increased competition and fewer patients, to meet their goals physicians increase their services. Each practitioner generates hundreds of thousands of dollars in health care expenses a year. Is it surprising that costs escalate as more practitioners enter an overcrowded profession and carve out their livelihood?

Prompt and drastic reduction in physician supply is an essential part of any health care plan. It should be the first step while other measures are being considered. Congress and state legislatures are reluctant to get involved in the details of medical student numbers, residency training positions, and the licensing of foreign medical graduates. Unfortunately, the medical profession is also uninterested. Are we waiting until fee-for-services medicine is history!

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1. Housepian EM: The physician and the poor (letter). *NY State J Med* 1991; 91:508.

2. Gershberg H: Physician supply and the

health care crisis (letter). *Ann Int Med* 1990; 113: 996.

3. Iglehart JK: Health policy report. Canada's health care system faces its problems. *N Engl J Med* 1990; 322:562–568.

TO THE EDITOR: Dr Eli Ginzberg's response to Dr Housepian's comments regarding his commentary "The Physician and the Poor" was arrogant.[1–3] Instead of answering the items brought up, he simply denied the comments were applicable to him without bothering to say why. Perhaps he agreed that the medical instruments and the drugs are overpriced, and the cost of health management bureaucracy excessive but had no courage to say so.

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1. Ginzberg E: The physician and the poor. *NY State J Med* 1991; 91:236–237.
2. Housepian EM: The physician and the poor (letter). *NY State J Med* 1991; 91:508.
3. Ginzberg E: In reply (letter). *NY State J Med* 1991; 91:508.

FROM THE LIBRARY

NOTE ON BEEF JUICE

There are conditions of frequent occurrence to almost every member of this Society, in which the patient's life turns on the question of adequate sustentation. I refer to cases of great asthenia due to protracted and exhausting illness, to hemorrhage, shock, septicaemia and operations of various kinds.

For more than fifteen years, in the states named, I have at times used besides cream, milk, eggs and alcohol, from four to thirty-two ounces of beef juice in twenty-four hours. The average amount may appear large, but the effect has seemed so favorable in the experience of myself and many of my professional friends in Rochester, that I take the liberty of referring to a process by which the juice is easily expressed as well as to its liberal use, for the information of those not already familiar with both. In diet books it is stated that beef juice is prepared by broiling the beef, cutting it into small pieces, and squeezing out the juice with a lemon squeezer. Very early I found that it was almost impossible to obtain any considerable quantity by this method. I therefore, in 1870, had a meat squeezer made on the principle of the lemon squeezer, with arms 32 inches long, and several years ago exhibited this instrument with its uses at a meeting of the Central New York Medical Association. Even with this, the expression of the juice in the desired amount, day after day, always proved laborious and exhausting. In 1880 it was discarded and there was substituted for it, what is known as the *Fruit Press.* By this, it is easy to express the juice in any desired quantity.

Fresh and juicy lean beef should be finely chopped by the butcher. A pound or more of this is thoroughly heated, in an open dish, over the fire, with constant stirring to keep it from burning. It is then quickly transferred to the cylinder and by the screw pressure of the instrument, the juice flows readily. Thus obtained it is a bright red fluid, becoming darker after standing, of a specific gravity of 1.032, acid, rendered solid by boiling, and as it cools, becoming rapidly coated with fat. Before being administered this is eliminated by passing it two or three times through a fine strainer.

WILLIAM S. ELY
(*Trans Med Soc Stat of NY* 1982, pp 124–126)

BOOK REVIEWS

HISTORY OF SYPHILIS

By Claude Quétel. Translated by Judith Braddock and Brian Pike. 342 pp, illustrated. Baltimore, Md, The Johns Hopkins University Press. 1990. $39.95 (hardcover)

In the introduction to this book, the author views the history of social diseases as a unique window to unusual insight into a society. He chose to focus on syphilis, the most feared and serious venereal disease, because the terror it has elicited through the ages has extended its influence to the level of a social and cultural phenomenon. This history not only discusses clinical, therapeutic, and epidemiologic aspects of syphilis, but also includes the profound influence of syphilis on mortality, literature and politics. The author, Claude Quétel, is an historian and researcher at the Centre Nationale de la Récherche Scientifique in Paris and a founding member of the Société Internationale de l'Histoire de la Psychiatrie et de la Psychoanalyse.

The book presents the history of syphilis chronologically in ten chapters, each discussing a different era, beginning in 1495 and extending to the present. In the conclusion, the author gives a very broad overview of all the sexually transmitted diseases, presented briefly in tabular form, and shows how AIDS has assumed the mantle of terror historically reserved for syphilis. This work has been translated from French into English and occasionally the phraseology is somewhat ponderous. On the whole, however, this book is very readable and is very carefully referenced. Following the text is a 40-page section of notes. The number of citations per chapter ranges from 34 to 124. There is no general bibliography because the author feels that list would be much too long. The book also includes a chronology of landmarks in the history of syphilis which extends from 1495 and 1965, and a very adequate index.

This volume will be interesting both to physicians caring for patients with sexually transmitted diseases and people who would like to understand the origins and ramifications of the social stigma surrounding patients with AIDS. Viewed from our present perspective, some of the treatment modalities and control measures employed in the past appear either silly, harmful, or unduly restrictive. Most of the discriminatory practices toward patients with syphilis were directly related to fear. Today similar fear has invoked discriminatory measures toward patients with AIDS.

Quétel also makes the interesting observation that fear of sexually transmitted diseases has been a factor limiting the number of sexual partners for most people. The only time in history when this fear was absent and the number of sexual partners increased dramatically was the approximately 25-year period which began with the widespread use of penicillin and ended with the onset of the AIDS epidemic.

The author clearly achieved his objective for this work. The major limitation of his book is its major emphasis on the history of syphilis in France, compared with the meager discussion of syphilis and its social, cultural, and political ramifications in other countries.

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AIDS AND OTHER SEXUALLY TRANSMITTED DISEASES—ADOLESCENT MEDICINE: STATE OF THE ART REVIEWS

Edited by Manuel Schydlower, MD, and Mary-Ann Shafer, MD. 237 pp, illustrated. Philadelphia, Pa, Hanley & Belfus, Inc, 1990 $28.00 (hardcover)

This text, edited by Drs Schydlower and Shafer, was written to provide a state of the art review of the major sexually transmitted diseases affecting adolescents. It is part of a series developed in cooperation with the Section on Adolescent Health of the American Academy of Pediatrics, evaluating important clinical issues concerning adolescent health and morbidity. As noted in the preface, the authors who have contributed to the text include the most prominent names in adolescent medicine and venereology. The style is fluid and concise, and the chapters are well referenced. The information is both accurate and timely, and provides both a foundation for the novice, as well as some challenging perspectives for the experienced clinician. The chapters are designed around clinical syndromes and issues concerning sexually transmitted diseases; these include specific organisms (eg, human papillomavirus and *Chlamydia*), epidemiologic principles regarding transmission and control in adolescents, a risk model for AIDS, issues about homosexual youth, laboratory testing, psychobehavioral factors, and historical perspectives regarding sexually transmitted diseases with some interesting comparisons to HIV infections of today.

This book is well suited for the individual practitioner or clinical staffs that care for adolescent patients. It describes the approach to adolescent patients regarding sexually transmitted diseases, methods for screening, treatment guidelines, and issues concerning epidemiology, compliance, and behavioral and educational factors. Although it does not have the breadth of the textbook by Holmes, Mardh, Sparling, and Weisner, it is more concise and easier to manage. Both texts have much less data on the male rather than female patient, with the exception of the chapter on homosexual youth; the opening chapter by Cates does comment on the paucity of information in the literature regarding males. Other texts on adolescent medicine or adolescent and pediatric gynecology are not as comprehensive in the discussion of sexually transmitted diseases as is the text under review here. The authors have achieved their objective, to provide a thorough and timely review of the sexually transmitted diseases of adolescents.

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THE TOXICITY OF ANTICANCER DRUGS

Edited by Garth Powis, DPhil, and Miles P. Hacker, PhD. 228 pp. Elmsford, NY, Pergamon Press, Inc, 1991. $49.50 (hardcover)

The goal of this compact volume is to "fill the gap" of information on the basic mechanisms of toxicity of antineoplastic agents. The contributing authors are generally clinical pharmacologists or oncologists. Organized in a series of chapters devoted to the major classes of anticancer drugs, the book provides a relatively complete summary of the toxicities of these agents. Most sections contain at least a brief discussion of the mechanism of anti-tumor activity for each of the drugs but more complete sources for this information are certainly available.

The book does bring together in one source a compendium of information about the toxicity of antineoplastic agents. The discussions are thorough and extensively referenced. The discussion of many of the most clinically significant toxicities includes some aspects of prevention and treatment. The section on platinum-based drugs, for example, contains a discussion of their emetogenic effect and treatment strategies. Similarly, there is a summary of anthracycline cardiotoxicity and protective treatments in the appropriate section.

This is not a volume whose main use will be as a frequent reference in the clinic, out-patient office, or hospital chemotherapy unit. Practical information relating to the acute treatment of chemotherapeutic toxicity is relatively limited. Some clinically troublesome toxicities, such as vincristine extravasation, receive only passing mention. Nevertheless, the educational and informational value of the book is high.

For those interested in clinical pharmacology, there is a good chapter on the usefulness of animal studies in predicting human toxicity of new antineoplastic drugs.

This chapter goes far in providing explanations of the role of animal models and experiments in the drug development process, relating to oncology treatments. A provocative chapter is also included on the development of second tumors after antineoplastic therapy.

The most severe criticism of the book relates to the copious references which, in virtually every chapter, seem largely outdated. The clinical application of biological response modifiers, such as interferon, interleukin, and tumor necrosis factor, and the understanding of their toxicity, has been explosive in the past two years. Yet, in the chapter dealing with the toxicity of these agents, only 16 of 181 references are from 1989, and just one is from 1990 (in press). Furthermore, the references throughout the book are identified in the text by author name and date. This bibliographic style makes it extremely difficult for a reader to locate the text related to a reference that has caught the eye while skimming a chapter's bibliography.

The book is well-written, with a straightforward style that may unfortunately discourage the casual reader.

The book is a solid academic summary of basic knowledge regarding antineoplastic toxicities. Its use will be for the student, practitioner or researcher seeking a complete discussion of these drugs' toxic effects. The book will not provide as much current information as one might expect, nor will it provide many specific treatment options for dealing with the often significant toxicities of the antineoplastic agents.

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CARBON MONOXIDE POISONING

By K. K. Jain, MD. 220 pp, illustrated. St Louis, Mo, Warren H. Green, Inc, 1990. $37.50 (softcover)

Although the relationship between carbon monoxide poisoning and suicide is well known, not enough emphasis has been given to the health effects of sub-lethal exposure to carbon monoxide. While there have been some publications devoted to the health effects of a specific environmental contaminant, those discussed rarely have the potential health impact of sub-lethal exposure to carbon monoxide. This is true in both terms of the number of individuals exposed and the excess morbidity and/or mortality caused. This book therefore is a much-needed addition to the medical/environmental health literature.

The author provides a wealth of information through a well-written, concise, and interesting treatment of the subject matter. The topic is introduced via an extremely interesting historical perspective of carbon monoxide poisoning. By including a series of experiments conducted by John Haldane in 1895, the author elegantly demonstrates that a great deal of knowledge was known about the mechanism of carbon monoxide poisoning nearly 100 years ago and makes the reader wonder why so many years had to elapse before the medical profession and society addressed the potential impact on health directly.

The author then presents a discussion of the biochemical, physical, and physiological aspects of carbon monoxide exposure in terms of the physical and chemical properties of the gas, its intake, distribution, and fate in the body, as well as its effect on oxygen transport and metabolism at both the organ system and cellular level. This is presented in a clear and concise manner to provide much information in a limited number of pages.

Subsequent chapters deal with the sources of carbon monoxide pollution, the epidemiology of carbon monoxide morbidity and mortality, the effects of carbon monoxide on various organ systems, pathological and clinical features of toxic carbon monoxide exposure, as well as the diagnosis and treatment of carbon monoxide poisoning. It was particularly rewarding to see an entire chapter devoted to the health effects of carbon monoxide derived from cigarette smoke. Cigarette smoke is the most common means of exposure to carbon monoxide, and thus the major mechanism by which this poisoning occurs. The extent of the resulting morbidity and/or mortality caused by such exposure is too often overshadowed by the association of cigarette smoke with cancer.

The author has successfully grappled with the problem of consolidating an extensive and well-dispersed body of journal publications and monographs into a clearly written reference work. Although some detail has not been included, this book affords the reader a clear and concise overview of the numerous health effects and treatment of carbon monoxide poisoning. A large number of references is provided at the end of each chapter to facilitate in-depth study of a particular subject area. Although it was the author's intent to gear this book to physicians, this book will be a valuable addition to the libraries of other health professionals having an interest in environmentally induced disease.

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RHEUMATOLOGY FOR THE HEALTH CARE PROFESSIONAL

Edited by Harry Spiera, MD, and Irwin Oreskes, PhD. 369 pp, St Louis, Mo, Warren H. Green, Inc, 1991. $42.50 (softcover)

In rheumatology, perhaps more than any subspecialty of medicine, the spectrum of disease entities and their pathophysiology are broad. The goal of this book is to provide an informational base of rheumatologic topics for the non-physician health professional. The targeted readership could range from physical therapist to physicians-in-training. The text is multi-authored by medical experts in their respective areas of interest.

The initial chapters give an overview of basic joint biology and a well-written discussion of the inflammatory mediators. Specific rheumatologic disease entities are described with historical perspectives, etiopathogenesis, clinical manifestations, and therapeutic considerations. There is no redundancy in the material presented. Topics range from "Local Musculoskeletal Problems" to the description of the "Diagnostic Process" with separate reference lists at the conclusion of each chapter.

Rheumatologic therapy and surgical alternatives with rational approaches to therapeutic decision-making are elucidated. Physical, occupational, psychological, cultural and social aspects of chronic arthritic diseases are discussed. Professional foundations and reference sources for patients and health care providers are listed.

Overall, the authors present an organized, coherent and up-to-date overview of major rheumatologic topics. This text achieves the authors' stated objective to provide an informational source for the non-physician health professional caring for the arthritic patient today.

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CLINICAL TESTS OF VISION

By Lars Frisen, MD. 222 pp, illustrated, New York, Raven Press, 1990. $59.00 (softcover)

This volume discusses the foundations, methods of examination, and interpretation of the full range of visual tests. Included are discussions of evaluation of acuity, contrast sensitivity, hue discrimination and saturation, metamorphopsia, dysmetropsia, and central and peripheral vision and perimetry. The salient features of each test are explained and up-to-date references provided for those seeking further practical information. The discussions are concise and limited to the subject at hand as much as is practicable.

Fully three quarters of the book are devoted to the evaluation of visual fields, both kinetic and static, with attention to localization of lesions. The last chapter is a test to help one apply what was learned in the previous chapters. It includes clinical scenarios with visual fields and provides a discussion and localization for each lesion.

The author, Lars Frisen, MD, PhD, is a clinician and vision researcher in the Department of Ophthalmology at the University of Gotenborg, Sweden. He provides 150 illustrations with the text, including 13 in full color.

This book will be of interest to those in need of a complete review of visual testing or as a reference for physicians who examine patients with visual impairments.

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PROGRESS IN CARDIOLOGY

By Douglas P. Zipes, MD, and Derek J. Rowlands, MD. 250 pp. illustrated. Philadelphia, Lea & Febiger, 1990. $39.50 (softcover)

As would be expected, Drs Douglas Zipes and Derek Rowlands selected an outstanding group of contributors for this book whose major theme relates to new technological developments regarding noninvasive imaging and the study of the heart.

I believe that having people working in the field write detailed chapters is a better approach to the review of these complex technological problems than having a single editor trying to abstract from the literature some bits and pieces from several articles and then writing a book of abstracts. I certainly believe the approach in this particular book is quite good.

The title, *Progress in Cardiology,* may be slightly misleading. As indicated in the preface, Zipes and Rowlands claim that they were going to talk principally about the study of cardiac patients outside the catheterization laboratory. I believe, however, that catheterization laboratory topics are very important and presumably will be addressed as they indicated in a future volume.

I have continued to be frustrated by the use of alphabet jargon in medical writing. Many times the alphabet soup is difficult to interpret and, on some occasions, one can spend long periods going back to review an entire article trying to find out what NCU (not clearly understood) and other similar abbreviations mean. I would suggest that most such abbreviations be eliminated, not only in this type of book, but also in all medical articles and books, with possibly the exception of abstracts. One to two chapters were quite difficult to read, and I was forced to go back to the beginning to find the definition of some abbreviations.

I am also concerned about using inanimate objects as subjects of active verbs. For example, "the electrocardiogram shows." I am also somewhat offended by phrases such as "the heart valve or heart muscle was not interrogated." I have trouble understanding how one can interrogate an inanimate object.

In spite of these caveats, however, I believe the book is well done. I personally like the concept of chapters written by people working in the field and anticipate that the following volumes will expand the topics to cover the entire concept of progress in cardiology.

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PHACOEMULSIFICATION SURGERY

By Terence M. Devine, MD, and William Banko, MD. 130 pp, illustrated. Oxford, England, Pergamon Press, Inc, 1991. $125.00 (hardcover)

In spite of the development of phacoemulsification as a means of cataract removal in the late 1960s, and its more recent promotion by ophthalmic equipment manufacturers and advocate ophthalmologists, there has remained no advanced surgical techniques text on the subject until the present volume edited by Devine and Banko.

The editors' approach to the subject as described in the preface is to ". . . teach the fundamental concepts and choices necessary to develop a personalized surgical technique." Their goal is achieved by outlining the various current techniques of 11 contributing ophthalmologists supplemented by individual views regarding the benefits of a particular approach. Since no comparison is made between approaches and the techniques rely on an evolving technology, the text risks faddism. Its importance, however, lies in the compilation of preferences of individual surgeons through which the reader might distill a technique variation that might prove helpful. Other current texts on phacoemulsification surgery such as Maloney and Grindle's, *Textbook of Phacoemulsification* or Koch's, *Conveting to Phacoemulsification*, take the readers through the initial steps in learning phacoemulsification as well as descriptions of some advanced technique variations.

The book is organized into five sections, all concerned with phacoemulsification. They are as listed: "History of Phacoemulsification," "How to Set the Dials," "Surgical Procedure," "Special Considerations," and "Complications." All chapters are written in an easily readable style accompanied by several excellent tables which list the individual contributors' preferences on machine settings, anesthesia techniques, and wound-closure techniques. There is also a table listing current equipment types with individual machine specifications listed. However, the drawings and photographs accompanying the text are of uneven quality, with many drawings of lens and instrument orientation that are diagrammatic rather than demonstrating proper technique. Many of the operative photographs are poorly focused. Notably absent are diagrams of the various suturing and astigmatic correction techniques.

The main body of work is devoted to chapter two, "How to Set the Dials" and chapter three, "Surgical Procedure." In chapter two, the readers will find a good elemental discussion of fluid dynamics and instrument design variations. A more complete discussion giving the reader understanding of the physics involved rather than simple dial setting, should have included Poiseuille's law and its application to real fluids as well as the physics of phacoemulsification energy transfer in different surgical situations. Chapter three is true to the editors' purpose with many technique variations discussed, especially capsulorrhexis, phacoemulsification approaches, and cortical cleanup. Absent from the text are steps in learning phacoemulsification, endocapsular and different lens insertion techniques, as well as current implant lens technology and surface modification.

In summary, a reader interested in a compilation of current, phacoemulsification technique variations by several distinguished ophthalmologists would be well served by purchasing this text. A reader interested in learning phacoemulsification should consider either Koch's or Maloney and Gimble's texts. An alternative to the textbooks might be an obsessive perusal of the "throw away" ophthalmic literature and attendance at key ophthalmic meetings and phacoemulsification courses. The latter alternative is a far more costly, albeit more current, choice to purchasing Devine and Banko's comprehensive overview on current techniques.

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PROGRESS IN CARDIOLOGY

Edited by Douglas P. Zipes, MD, and Derek J. Rowlands, MD. 138 pp. Philadelphia, Lea & Febiger, 1991. $39.50 (softcover)

Progress in Cardiology is a biannual publication dealing, as it says on the inside cover, with "Recent Conclusions, Collaborative Studies, and Conceptual Breakthroughs—From the Molecular Basis of Cardiac Disturbances to Decisions Made at the Bedside." The year 1991 is the fourth year in which this publication has appeared, and the one under review is the first for this year–thus, the designation 4/1 Progress in Cardiology.

This issue, the editors state in their introduction, is a "blend of multiple themes, including echocardiography (particularly transesophageal echocardiology), coronary artery disease, and congestive heart failure." Important aspects of each topic are discussed in detail by one or more of the twenty authors. The section on echocardiography deals principally with the advantages of the transesophageal approach, as compared with the transthoracic approach, in the definition of such problems as infective endocarditis and atrial thrombi. The illustrations are clear and convincing.

The section on coronary artery disease is divided into interesting components: Cardiovascular Actions of Fish Oils and Omega-3 Fatty Acids, Evaluation and Management of the Patient after Myocardial Infarction, Silent Myocardial Ischemia, and Clinical Implications of the Cardiac Arrhythmia Suppression Trial (CAST).

The section on Congestive Heart Failure contains subtitles on Epidemiologic Considerations in Heart Failure, Ventricular Arrhythmias and Sudden Cardiac Death, Renin-Angiotensin-Aldosterone System, and Atrial Natriuretic Factor and Arginine Vasopressin, with the promise that there will be discussions of other topics related to congestive heart failure in the second issue of the 1991 series.

Long ago it became evident, as the rate of scientific discovery accelerated, that textbooks were usually out-dated by the time they were published. Even those with many concurrently working authors required too much time to compile to be current on completion. The solution appeared to lie in the publication of reviews or monographs of limited subject material that could be produced in months instead of years. Yearbooks, annual reviews, and periodic clinics became more popular for gathering the latest accumulation of data and opinions on medical subjects.

Progress in Cardiology is another such attempt to keep cardiologists abreast of current knowledge and opinion in areas that are developing rapidly. However, it is really more that just another attempt, for the reputation of the editors for accuracy and clarity in research and teaching practically guarantees the excellent quality of the entries in their publication.

It is of some interest that theirs is a biannual journal rather than a bimonthly or quarterly one, as so many are. Perhaps, it is because their articles are more succinct and to the point, or perhaps it is because their priorities are more restrictive. Whatever the reason, having read one of their issues, one looks forward eagerly to the next.

The sections in this publication are written primarily for cardiologists and internists with large cardiac practices. They are full of practical and theoretical material.

Of all the periodic publications in cardiology, and there are many, this one, *Progress in Cardiology*, is one of the best and deserves to be read.

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PRINCIPLES AND PRACTICE OF PEDIATRICS

Edited by Frank A. Oski, MD, Catherine D. DeAngelis, MD, Ralph D. Feigin, MD, Joseph B. Warshaw, MD. 2,000 pp, illustrated. Philadelphia, JB Lippincott Co, 1990. $99.50 (hardcover)

The purpose of this comprehensive text is to meet the needs of students of pediatrics whether they are medical students, house officers, or practitioners. The book is intended to be both comprehensive and cohesive. The authors' goal has been to accurately represent the depth and breadth of pediatrics as it is practiced today. I think these goals have been partially accomplished.

The credentials of the editor-in-chief, Dr Frank A. Oski, and the editors Dr Catherine D. DeAngelis, Dr Ralph D. Feigin, and Dr Joseph B Warshaw are impeccable. Dr Oski is well-known for the outstanding insight and foresight that he has demonstrated over the years in the field of pediatrics. Similar to any comprehensive text such as this, there is an extensive list of contributors (261 to be exact). The majority of the contributors represents faculties from the respective institutions of the editors.

This book seeks to establish a niche among the several other well-known comprehensive textbooks in general pediatrics. To this end, the editors have divided the book into a unique arrangement, use five main subsections: general pediatrics, the fetus and the newborn, ambulatory pediatrics, the sick or hospitalized patient, and a miscellaneous compendium of facts. Although this format might be considered somewhat different, overall the table of contents is quite similar to those of other competing textbooks. I found the organization somewhat contrived. Many of the topics could have easily slid from one section to another. The format led to some redundancy in presentations and made some topics difficult to find.

The first section provides an overview of general pediatrics. The interested reader will particularly enjoy the several introductory sections that cover the history and current status of pediatrics in the United States, the ethics of pediatric practice, and medical economics. There is also a useful chapter on reading and interpreting the medical literature. The remainder of the chapter covers several different topics that could have very easily been placed in the other sections. The section on the fetus and the newborn very nicely covers most of the relevant topics in this area. The third section on ambulatory pediatrics ambitiously attempts to provide an all encompassing overview. The fourth section on the care of the sick or hospitalized patient begins with a discussion of aspects of intensive care management and continues with a disease-oriented approach to subdividing the topics under this heading. The final section is really a hodgepodge of useful tidbits including interpreting laboratory tests, identifying inborn errors of metabolism, assisting with syndrome diagnosis, an outline of selected pediatric procedures, and an overview of presenting signs and symptoms.

Textbooks like this one relying on the contributions of a large number of contributors, potentially suffer from variation in quality, accuracy, writing style, and clarity. This book displays some of these problems. For instance, the section on asthma presents a somewhat narrow approach to the acute and chronic aspects of management. The section on the pathophysiology of body fluids includes a discussion of the management of shock. That section does not include a good clinical definition of shock and the management suggested, fluid administration over one to two hours, is simply wrong in my view. I believe the reader is done a disservice by the superficial presentation of medicines in sections such as the one in intubation.

Personally, I found the subdivisions of this text less than helpful. I was also less than pleased with its overall balance. For instance, the collective space devoted to the topics of pharyngitis, otitis, sinusitis, and the management of the febrile child is less than that of the single section on skeletal dysplasias. I realize, however, that the appreciation for a general textbook such as this one is a very individual matter. What makes sense to one reader may not seem logical to the next. I certainly feel that this publication is on equal footing with other publications in the field.

If the reader is interested in a textbook in general pediatrics, I suggest the following rule. Look at the table of contents. Is this similar to how you might arrange a filing cabinet? If not, it is probably not the right textbook for you. Does the textbook emphasize things that are important to you? Compare chapters on topics with which you are very familiar. By following the above rules, and then perusing areas of interest, one will probably be able to purchase the textbook which provides the best personal fit.

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THE BREAST: COMPREHENSIVE MANAGEMENT OF BENIGN AND MALIGNANT DISEASES

Edited by Kirby I. Bland, MD, and Edward M. Copeland, III, MD,. 1,154 pp, illustrated. Philadelphia, WB Saunders, 1990. $150.00 (hardcover)

The editor of this book set out to present a "comprehensive and readable text on the basic principles necessary for the diagnosis and treatment of various benign and malignant breast diseases." Generally, Bland and Copeland have admirably achieved this goal in this multi-authored text.

The book touches on every aspect of breast disease but does so in a somewhat uneven fashion. For example, the practical management of the patient with overt gross cysts, a very common condition, receives scant if any attention. Yet, many other far less common conditions that will be rarely encountered by the average practitioner are presented in much greater detail. There are also some problems engendered by the fact that different authors present related subjects in separate chapters without an attempt to synthesize the material into usable form for the reader. Mammillary fistula and periareolar abscess in the non-lacting breast are discussed in one chapter, whereas duct ectasia, which may be related to these conditions, is described in another chapter. However, there is little attempt to reconcile any differences or similarities existing between these entities. The uneven treatment of various subjects is exemplified by the surprisingly detailed descriptions of such currently outmoded surgical procedures as the classical Halstedian radical mastectomy and the extended radical mastectomy.

The reader is indeed presented with an enormous compendium of data and summaries of studies on various aspects of breast disease. However, it is somewhat difficult for the average person to place all of these data into perspective such that the material can be applied directly to the patient. It would have been helpful to have offered some editorial comment on some of the more controversial subjects presented by various authors.

The book is generally well-written but the organization leaves something to be desired. I found it difficult to find the treatment of some subjects. For example, one of the most common procedures currently in use, such as the excision of non-palpable, needle-localized mammographic lesions, is presented in a chapter on the choice of operations for breast cancer. Several subjects such as lobular carcinoma in situ are discussed in scattered places, rather than being presented in one more comprehensive section. The illustrations are clear and useful. References at the end of each chapter are comprehensive and complete, and it will be of great value to the student of breast disease.

In spite of the criticisms cited above, the purchase of this book will be mandatory for all physicians who treat patients with disease of the breast because the book provides such an extensive and comprehensive review of all aspects of the subject. It is an extremely useful addition to the literature on this subject and compares extremely favorably with other texts on the same subject.

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THE MERCK MANUAL OF GERIATRICS

Edited by William B. Abrams, MD, Robert Berkow, MD, and Andrew J. Fletcher, MBBCHIR. 1,267 pages. Rahway, NJ, Merck, Sharp & Dome Research Laboratories, 1990. $20.00 (hardcover)

The Merck Manual has done it again, this time in geriatrics. One hundred and four chapters from undisputed experts have been carefully edited into a truly exceptional reference manual. That this is a manual written by and for clinicians is immediately evident from its format.

The problem-oriented approach section helps non-geriatricians organize their priorities toward the dominant principle of functional assessment characterized by activities of daily living, mobility, cognition, continence and nutrition, thereby often avoiding the quagmire of a seemingly endless list of diseases which so frequently seem to bother the physician more than the patient.

The chapters on inappropriate antidiuretic hormone, hypothermia, and syncope offers concise and precise information, and those on falls and gait disorders, incontinence and chronic pain are alone worth the modest price of the volume.

The section on specific approaches covers the formation of a patient care plan from a comprehensive functional assessment by a multidisciplinary team, which has been enshrined in long-term care by OBRA '87 as the Minimum Data Set. Although not in itself a physician-oriented tool, it is admirably designed to expose quickly any deterioration in function that so often is the earliest sign of acute disease in the elderly. The chapters devoted to pharmacodynamics, pharmacokinetic, and pre- and post-operative care are basic to the care of the elderly, and the excellent section on geriatric rehabilitation sheds light into a black hole for many of us. One should not even attempt the additional qualification examination without reading these chapters.

The organ systems section is the marathon performance for which Merck manuals are justly famous. Now, however, each chapter is prefaced by a sometimes delicate comparison between normal changes of aging and disease-determined changes, a distinction frequently without precise boundaries. I was particularly drawn to statements such as "The liver ages gracefully" and the concept expressed in the section on geriatric sexuality that "the longer time to arousal makes the geriatric male a more satisfying lover."

The special issue section is an informative one and covers such topics as epidemiology, elder abuse and legal/ethical issues, including the current major topic of advance directives. The extensive tables of geriatric laboratory values show conventional and system international values side-by-side, and ample examples are given of instruments used in the assessment of activities of daily living, cognition, balance, and gait.

The editors are to be congratulated for having admirably succeeded in uniting the thoughts of a hundred experts without overt signs of disagreement. A brief period of awed silence is appropriate. This volume belongs at the desk of any physician who presently treats older patients and wants the satisfaction of treating them just a bit better.

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SURGERY OF THE EAR

Fourth Edition. Edited by Micheal E. Glasscock, III, and George E. Shambaugh, Jr. 640 pp, illustrated. Philadelphia, WB Saunders, 1990. $125.00 (hardcover).

This volume, first published in 1959 and now in its fourth edition, has established itself over the past decades as one of the better, single-volume textbooks of otology and now neuro-otology and skull-base surgery.

The book has evolved from that of its original single author, George E. Shambaugh, Jr, MD, to an edited version including the work of 12 qualified contributors and three editor/authors. Drs Shambaugh, Glasscock, and the contributing authors have established themselves through practice and publications as experts in the field. The text retains, as the editors phrase it, "the easy to read style" that Dr Shambaugh imparted to the original edition.

As a single volume, *Surgery of the Ear* cannot cover every aspect of the field in exhaustive technical detail. Indeed, the topics of almost every chapter have been expanded into entire textbooks. The text, divided into four sections, covers all aspects of ear surgery, including embryology, anatomy, diagnosis, infections, deafness, facial nerve, vertigo, tumors, and implantable devices. Temporal bone dissection and surgical anatomy are covered in the appendix.

Neuro-otology and particularly skull-base surgery are rapidly expanding fields, which have naturally grown from the ear surgeon's knowledge of temporal bone anatomy. With the addition of several new chapters, the book is up-to-date on the current developments in these areas. Intraoperative facial and acoustic nerve monitoring receive an entire chapter, as do implantable hearing devices.

Cerebellopontine angle lesions are thoroughly presented in Chapter 23, although the author has biased the discussion of surgical results toward Dr Glasscock's experience even though others have had equal or larger series. The imaging chapter discusses the use of the magnetic resonance imaging, computed tomography, and other modalities to study the temporal bone.

The chapters covering infectious complications or otitis media are complete. Forty-six pages are devoted to these potentially disastrous but now less frequent complications. Mastoid operations, often confusing to the beginner, are clearly diagrammed and explained in two chapters. The section entitled "Surgery of Deafness" contain seven chapters, six of which take the reader through the mechanics of hearing and surgical corrections of the various forms of conductive hearing losses. Of historical interest only, the chapter discussing conventional radiology could be shortened from its current 25 pages.

This book is well illustrated with anatomic drawings, radiographs, and photographs. Surgical procedures are clearly visualized. The current editors keep the appropriate amount of historical material, providing not only interesting readers but a sense of the development and evolution of techniques which form the basics of contemporary temporal bone surgery. One very nice aspect of the book, preserved from previous editions, is the photographs and descriptions of important historical figures in otology, placed at the beginning of the chapters.

This book will always prove to be a valuable reference to residents, practicing otolaryngologists and otologists. It should be read in its entirety, however, by anyone contemplating ear surgery or the treatment of ear diseases.

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EYE TRAUMA

Edited by Bradford J. Shingleton, MD; Peter S. Hersh, MD; Kenneth R. Kenyon, MD. 427 pp, illustrated. St Louis, Mo, Mosby-Year Book, Inc, 1991. $99.00 (hardcover)

The editors of this rather comprehensive book, Drs Shingleton, Hersh, and Kenyon, comment in their introduction that the management of ocular injuries "embraces the full spectrum of ophthalmology practice" and that "eye trauma is evolving into a subspecialty of its own."

These two comments juxtapose uncomfortably as we head into the last decade of the 20th century. On the one hand, medicine is still madly sub-specializing itself into ever narrower areas of medical-surgical expertise and at the same time seeking to encourage more broadly educated physicians so that they might take a firmer, more participatory hand in all aspects of their field of endeavor, from primary to tertiary.

I will not argue the merits of these trends. Suffice it to say that *Eye Trauma* is certainly a book that should be used by all ophthalmologists on both scores. For those ophthalmologists not wishing to delve into the depths of eye trauma as a "subspecialty" this book will allow understanding and definition of the limits and scope of the work they do wish to undertake. For those of us who have taken on roles within tertiary eye care facilities, with the imprimatur "eye trauma centers," this book provides as complete a compendium of the subject as any available today.

This book is a unique effort of 52 contributors. There are 37 chapters divided nicely into six sections. The photographs, including 38 color plates, are of good quality, and the art work is excellent.

Except for several minor lapses, this book has been extremely well edited. Where there is overlap in areas of contiguous interest, such as in the problem of intraocular infection, a discussion of which might be entertained in several chapters relating to penetrating injuries, the authors wisely direct the reader to chapters specifically charged to detail this information.

Ophthalmologists tend to be a fussy lot, and we are capable of pontificating over a subject for many pages. We are inescapably drawn to the fine details of our craft, for it is the details that make for excellence. However, we have all seen the forest lost through the trees. *Eye Trauma* has beautifully balanced the need for detail with the clear understanding that this is, after all, a book for clinicians seeking to put their knowledge efficiently and securely to work. As an example, a chapter on the pathophysiology of anterior segment wound healing does not bog down in the rarefied aspects of laboratory and experimental work in tissue healing. The chapter is solidly in support of the body of work in the chapters on anterior segment repair to either side.

The comprehensive aspects of this book are reflected in the detailed chapters on ocular as well as adnexal and orbital trauma. Finally, the editors include chapters on epidemiology and the medical legal consequences of caring for eye trauma.

I was pleasantly surprised to find that although a plethora of authors participated in this book, the quality of each of its chapters is quite uniform as are the writing styles. Several other books relating to eye trauma have been published in recent years. I believe that *Eye Trauma* will take a rightful place as a major contribution to the effort to expand our ability to care for the injured eye. It is an excellent source of reference for the established clinician, and an important text for use by residents in ophthalmology.

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THE DEVELOPMENT OF MEDICAL TECHNIQUES AND TREATMENTS: FROM LEECHES TO HEART SURGERY.

By Marin Duke, MD. 252 pp, Madison, Ct, International Universities Press, Inc, 1991. $32.50 (hardcover)

I admire anyone, especially a busy colleague, who takes the time and has the patience to do the research necessary to write a book; it requires a very special talent, and for this creation I salute Dr Duke. His book is interesting but is of little value to the well-informed physician, especially the medical history "buff."

I enjoyed the first chapter on cardiopulmonary resusciation, "The Breath of Life"; my own long-time interest in *Citizens CPR* attracted my attention. I found little new in the sections on clinical techniques, pharmacology, and contemporary cardiac care. However, the early history of the events that led to the discoveries intrigued me.

This pleasant, easy-to-read book will be of special value to undergraduate students interested in the medical sciences, as well as the laity and first year medical students. The rise and fall of leeches will fascinate them.

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VAGINAL SURGERY

Third Edition. By David H. Nichols, MD, and Clyde L. Randall, MD. 463 pp, illustrated. Baltimore, Md, Williams & Wilkins, 1989. $76.00 (hardcover)

This third edition is one of the classic books that should be read by all gynecologic surgeons interested in perfecting their skills in vaginal surgery. It has been considerably expanded compared with the previous editions. An important aspect of the book is the in-depth discussion about the applied anatomy of the pelvis and the pathophysiologic basis of the various conditions that would necessitate surgical correction. The advantages and drawbacks of the various procedures and the reasons for selecting a particular procedure are well discussed. The authors have included discussion on pre- and postoperative care, appropriate surgical instruments, and suture material used for vaginal surgery. The surgical procedures have been described step-by-step in great detail, helping the reader to perfect his own technique.

It is hoped that future editions would include details of some other surgical procedures (eg, the Pyerea procedure and its modifications) that have been successfully used for urinary stress incontinence by other surgeons. Some color plates from actual surgical procedures should also be included in future editions to stress the importance of good dissection for visualizing tissue planes, as is apparent when one observes Dr Nichols perform a surgical procedure. This textbook is a treatise that is easy to read. It will be a valuable companion for all residents as well as gynecologists interested in becoming proficient in vaginal surgery.

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OBITUARY LISTINGS

Sara Bass, MD, New York. Died September 13, 1991; age 87. Dr Bass was a 1929 graduate of Cornell University Medical College. She was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. Her memberships included the American Society of Anesthesiologists, Inc, the American Medical Women's Association, the New York County Medical Society, and the Medical Society of the State of New York.

Heinz Bieringer, MD, Saratoga Springs. Died December 12, 1991; age 82. Dr Bieringer was a 1934 graduate of the Medizinische Fakultaet der Ludwig Maximiliams Universitaet, Munchen, Bayern, Germany. He was a Diplomate of the American Board of Family Practice and a Fellow of the American Academy of Family Physicians. He was a member of the Medical Society of the County of Saratoga and the Medical Society of the State of New York.

George Loveridge Bowen, MD, Blue Bell, Pennsylvania. Died October 24, 1991; age 96. Dr Bowen was a 1925 graduate of George Washington University School of Medicine and Health Science, Washington, DC. He was a Diplomate of the American Board of Obstetrics and Gynecology, a Fellow of the American College of Obstetricians and Gynecologist, and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the New York Obstetrical Society, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Earland Cyrus, MD, Brooklyn. Died May 15, 1991; age 84. Dr Cyrus was a 1942 graduate of Howard University College of Medicine, Washington, DC. He was a member of the American Academy of Family Physicians, Medical Society of the County of Kings, and the Medical Society of the State of New York.

Leonard Isaac Ehrlich, MD, Great Neck. Died January 10, 1992; age 78. Dr Ehrlich was a 1938 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Samuel A. Feldman, MD, Pompano Beach, Florida. Died September 23, 1991; age 86. Dr Feldman was a 1931 graduate of the University of Maryland School of Medicine, Baltimore, Maryland. He was a Diplomate of the American Board of Internal Medicine. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Vincenzo Ferraiuolo, MD, Franklin Square. Died January 16, 1992; age 66. Dr Ferraiuolo was a 1963 graduate of the Facolta di Medicina e Chirurgia, Universita di Napoli-I, Naples, Italy. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Fred Joseph Fumia, MD, Pittsford. Died December 31, 1991; age 88. Dr Fumia was a 1931 graduate of Albany Medical College. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

H. Louis George, MD, Watertown. Died December 1, 1991; age 89. Dr George was a 1927 graduate of Harvard Medical School. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Jefferson and the Medical Society of the State of New York.

Murray Greenwald, MD, Brooklyn. Died October 25, 1991; age 91. Dr Greenwald was a 1925 graduate of Boston University School of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Louis A. Goldstein, MD, Rochester. Died January 27, 1992; age 85. Dr Goldstein was a 1932 graduate of the University of Rochester School of Medicine and Dentistry. He was a Diplomate of the American Board of Orthopedic Surgery and a Fellow of the American College of Surgeons. His memberships included the New York Academy of Medicine, the American Orthopaedic Association, the American Academy of Orthopaedic Surgeons, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Anthony Joseph Granata, MD, New Rochelle. Died October 7, 1991; age 74. Dr Granata was a 1943 graduate of the Facolta di Medicina e Chirurgia, Universita di Bologna, Bologna, Italy. He was a Diplomate of the American Board of Family Practice. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

David G. Greene, MD, Buffalo. Died October 30, 1991; age 77. Dr Greene was a 1940 graduate of Harvard Medical School. He was a Diplomate of the American Board of Internal Medicine, a Fellow of the American College of Physicians, a Fellow of the American College of Cardiology, and a Fellow of the American College of Chest Physicians. He was a member of the American Physiological Society, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Murray Greenwald, MD, Brooklyn. Died October 25, 1991; age 91. Dr Greenwald was a 1925 graduate of Boston University School of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Myron B. Harkavy, MD, Woodmere. Died January 29, 1992; age 66. Dr Harkavy was a 1951 graduate of New York Medical College. He was a Diplomate of the American Board of Internal Medicine. He was a member of the American Geriatrics Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Jack W. Herrmann, MD, Buffalo. Died December 31, 1991; age 74. Dr Herrmann was a 1941 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a Diplomate of the American Board of Surgery, a Diplomate of the American Board of Thoracic Surgery and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Milton Holtzman, MD, Rockville Centre. Died December 9, 1991; age 71. Dr Holtzman was a 1945 graduate of Middlesex University School of Medicine, Waltham, Massachusetts. He was a Diplomate of the American Board of Physical Medicine and Rehabilitation. His memberships included the New York Academy of Medicine, the New York Society for Physical Medicine and Rehabilitation, the American Academy of Physical Medicine and Rehabilitation, the Nassau County Medical Society, and the Medical Society of the State of New York.

Suzanne Adele L. Howe, MD, New York. Died September 29, 1991; age 78. Dr Howe was a 1940 graduate of Cornell University Medical College. She was a Diplomate of the American Board of Otolaryngology. Her memberships included the American Medical Women's Association, the American Academy of Otolaryngology, the New York County Medical Society and the Medical Society of the State of New York.

Leif G. Jensen, MD, Orlando, Florida. Died December 9, 1991; age 96. Dr Jensen was a 1923 graduate of Cornell University Medical College. He was a Fellow of the American College of Surgeons. He was a member of the Richmond County Medical Society and the Medical Society of the State of New York.

Glenn Eugene Jones, MD, Grand Island. Died January 11, 1992; age 72. Dr Jones was a 1948 graduate of the University of Rochester School of Medicine and Dentistry. He was a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology. His memberships included the New York Academy of Medicine, the Radiological Society of New York State, the Radiological Society of North America, Inc, the Medical Society of the County of Niagara, and the Medical Society of the State of New York.

Daniel Kaplowitz, MD, New York. Died December 25, 1991; age 75. Dr Kaplowitz was a 1943 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the American Academy of Psychoanalysis, the New York County Medical Society, and the Medical Society of the State of New York.

Alexander Karetzky, MD, New York. Died November 28, 1991; age 87. Dr Karetzky was a 1928 graduate of the State University of New York Health Science Center at Brooklyn, College of Medicine. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Jules M. Koch, MD, New York. Died October 5, 1991; age 84. Dr Koch was a 1932 graduate of New York Medical College. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Cardiology. His memberships included the New York Academy of Medicine, the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Bronx County Medical Society, and the Medical Society of the State of New York.

Edith Lind Kristeller, MD, New York. Died January 26, 1991; age 81. Dr Kristeller was a 1947 graduate of the Medical College of Pennsylvania. She was a Diplomate of the American Board of Physical Medicine and Rehabilitation and a Fellow of the American College of Rheumatology. Her memberships included the New York Academy of Medicine, the New York Society for Physical Medicine and Rehabilitation, the American Academy of Physical Medicine and Rehabilitation, the American Medical Women's Association, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Louis Langman, MD, Sagaponack. Died October 1, 1991; age 89. Dr Langman was a 1927 graduate of Columbia University College of Physicians and Surgeons. He was a Diplomate of the American Board of Obstetrics and Gynecology and a Fellow of the American College of Surgeons. He was a member of the New York Academy of Medicine, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Stuart Jay Leavitt, MD, New York. Died January 1, 1992; age 60. Dr Leavitt was a 1956 graduate of New York Medical College. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the New York County Medical Society, and the Medical Society of the State of New York.

Erwin Levy, MD, New York. Died November 10, 1991; age 85. Dr Levy was a 1930 graduate of the Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

Tera Lieblich, MD, Forest Hills. Died April 23, 1991; age 84. Dr Lieblich was a 1933 graduate of Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. She was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Milton Roland Louria, MD, New York. Died November 28, 1991; age 94. Dr Louria was a 1928 graduate of Columbia University College of Physicians and Surgeons. He was a Fellow of the American College of Chest Physicians. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

John Baptiste Mauro, MD, Teaneck, New Jersey. Died November 5, 1991; age 96. Dr Mauro was a 1921 graduate of the University of Vermont College of Medicine, Burlington. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Richard Michael Messura, MD, Fairport. Died December 22, 1991; age 62. Dr Messura was a 1963 graduate of Georgetown University School of Medicine. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

Salvatore Louis Noto, MD, Freeport. Died January 17, 1991; age 74. Dr Noto was a 1942 graduate of Boston University School of Medicine. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the New York Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Milton Ford Quander, MD, New York. Died September 21, 1991; age 80. Dr Quander was a 1943 graduate of Howard University School of Medicine. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Joseph Mead Riley, MD, Fair Haven. Died March 31, 1991; age 86. Dr Riley was a 1930 graduate of Georgetown University School of Medicine. He was a member of the Medical Society of the County of Oswego and the Medical Society of the State of New York.

Bessie L. Silver Rini, MD, Mount Vernon. Died November 1, 1991; age 94. Dr Rini was a 1923 graduate of the State University of New York Health Science Center at Brooklyn, College of Medicine. She was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Gustav Rosbasch, MD, Rochester. Died January 7, 1992; age 91. Dr Rosbasch was a 1924 graduate of Medizinische Fakultaet, Albert Ludwigs Universitaet, Freiburg im Breisgau, Baden-Wurttemberg, Germany. His memberships included the American Geriatrics Society, the American Academy of Family Physicians, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Harold Rosenberg, MD, Sarasota, Florida. Died November 23, 1991; age 78. Dr Rosenberg was a 1942 recipient of the Registrable Qualification Granted by Scottish Conjoint Board, Scotland. He was a Diplomate of the American Board of Anesthesiology and a Fellow of the American College of Anesthesiologists. His memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, Inc, the Medical Society of the County of Queens, and and the Medical Society of the State of New York.

Hyman Rosenfeld, MD, Brooklyn. Died December 17, 1991; age 92. Dr Rosenfeld was a 1924 graduate of Columbia University College of Physicians and Surgeons. He was a Qualified Fellow of the International College of Surgeons. He was a member of the American Society of Abdominal Surgeons, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

Irving Singer, MD, Jamaica. Died December 24, 1991; age 94. Dr Singer was a 1924 graduate of New York Medical College. He was a member of the Medical Society of the County of Queens and the Medical Society of the State of New York.

Beatrice E. Scot Sommer, MD, Massapequa. Died December 8, 1991; age 70. Dr

Sommer was a 1948 graduate of New York University School of Medicine. She was a Diplomate of the American Board of Pediatrics. She was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Morris Springer, MD, New York. Died December 1, 1991; age 83. Dr Springer was a 1935 graduate of George Washington University School of Medicine and Health Science. He was a member of the American Society of Legal & Industrial Medicine, the Medical Society of the County of Kings and the Medical Society of the State of New York.

Oscar Sreter, MD, Long Beach. Died January 23, 1992; age 85. Dr Sreter was a 1934 graduate of Deutsche Univerzita Medizinische Fakulta, Praha, Czechoslovakia. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

H. Harold Taus, MD, San Pedro, California. Died September 24, 1991; age 85. Dr Taus was a 1932 graduate of the University of St Andrews School of Medicine, Dundee, Scotland. Hew as a Diplomate of the American Board of Radiology. He was a member of the Radiological Society of New York State, the Bronx County Medical Society, and the Medical Society of the State of New York.

Annis Eveleen Thomson, MD, Yonkers. Died January 13, 1992; age 102. Dr Thomson was a 1921 graduate of the University of Toronto, Faculty of Medicine, Toronto, Ontario, Canada. Her memberships included the New York Academy of Medicine, the American Medical Women's Association, the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

William F. Treichel, MD, Huntington. Died December 26, 1991; age 76. Dr Treichel was a 1946 graduate of Hahnemann University School of Medicine, Philadelphia, Pennsylvania. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Psychiatric Association, the Suffolk County Medical Society, and the Medical Society of the State of New York.

Joseph J. Tuby, MD, Brooklyn. Died November 13, 1991; age 83. Dr Tuby was a 1935 graduate of the University of Maryland School of Medicine, Baltimore. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

William G. Weare, MD, Youngstown. Died December 27, 1991; age 75. Dr Weare was a 1941 graduate of University of Toronto, Faculty of Medicine, Toronto, Canada. He was a member of the Medical Society of the County of Niagara and the Medical Society of the State of New York.

Daniel H. Welner, MD, Boca Raton, Florida. Died January 2, 1992; age 69. Dr Welner was a 1946 graduate of Cornell University Medical College. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York. New York.

Woon Bok Rhee Yeon, MD, Hopewell Junction. Died December 9, 1991; age 69. Dr Yeon was a 1958 graduate of Korea University Medical College, Seoul, South Korea. She was a Diplomate of the American Board of Psychiatry and Neurology. She was a member of the American Psychiatric Association, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Leonard I. Ehrlich, MD
1914–1992

Dr Leonard I. Ehrlich, a well-known pediatrician who practiced in Great Neck, New York, for half a century, died at home on January 10, 1992, at the age of 77. A graduate of the City College of New York, he received his medical degree from New York University in 1938. He interned at the Beth Moses Hospital (now Maimonides Hospital Medical Center) in Brooklyn and received further post-graduate training at the Willard Parker Hospital and at Cumberland Hospital. In 1943, he established a pediatric practice in Great Neck, which later became a highly respected group known as Great Neck Pediatric Associates.

Dr Ehrlich was a founding physician of the North Shore University Hospital in Manhasset, New York, where he served for many years as an attending physician and director of the Childrens Kidney Disease Clinic. He was also an attending physician in the Department of Pediatrics at Long Island Jewish Medical Center. He helped to establish a pediatric kidney disease clinic at Huntington Hospital and was physician to the Buckley Country Day School in Roslyn, New York.

A botany major at City College, he loved nature walks on Long Island and delighted in the flowering of the trees and flowers in Great Neck in the spring. In addition to being an avid baseball fan, he enjoyed walking on the dunes along Long Island's shore.

Dr Ehrlich was a strong supporter of the *Journal*. His opinions were regularly sought by the editors, and he constantly provided valuable advice about editorial content. We at the *Journal* found him generous, kind, insightful and wise, and will greatly miss him.

Dr Ehrlich's wife, Rosalie, of 46 years, died in 1988. He is survived by three sons, Daniel of Roslyn, David and Paul of Manhattan, a daughter, Julie Ehrlich of Huntington, two brothers, Albert of Great Neck, and Eugene of Mamaroneck and six grandchildren.

Pascal James Imperato, MD
Editor

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1992; 92:122–125). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith^2 reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Samples references:

1. Kepes ER, Thomas P, Vemulapalli K: Methadone and intravenous morphine requirements. *NY State J Med* 1983; 83:925–927.
2. Behrman RE, Vaughn VC: *Nelson Textbook of Pediatrics,* ed 12. Philadelphia, WB Saunders Co, 1983, pp 337–338.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine,* PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

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WHEN DEPRESSION KEEPS YOUR PATIENTS OUT OF THE MAINSTREAM

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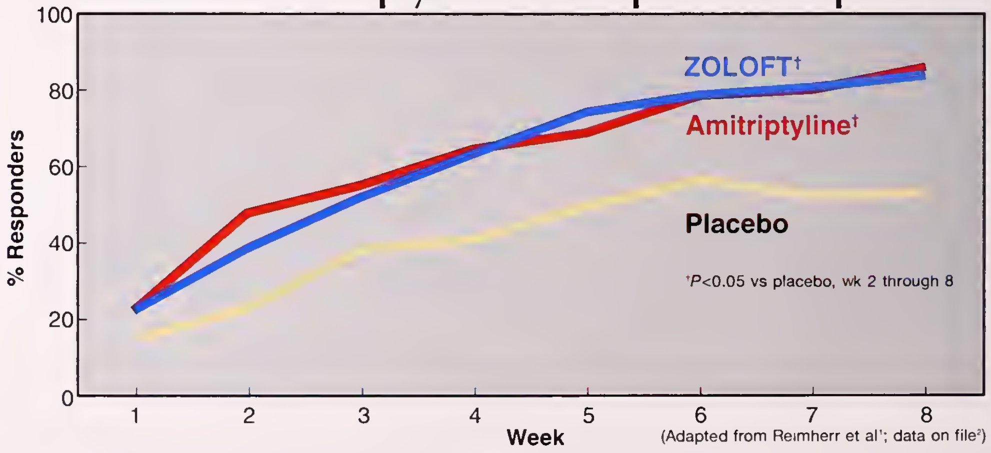
ZOLOFT...Effective in the Treatment of Depression

ZOLOFT vs Amitriptyline: Antidepressant Response*[1,2]



†$P<0.05$ vs placebo, wk 2 through 8

(Adapted from Reimherr et al[1]; data on file[2])

*Observed cases, evaluable patients (ie, patients who took drug on or after double-blind treatment day 11 and who had efficacy evaluations within that same time frame); response is defined as a CGI-Improvement score of 1 (very much improved) or 2 (much improved) at time point. Patient numbers for each regimen: ZOLOFT—wk 1=117, wk 2=120, wk 3=106, wk 4=98, wk 5=93, wk 6=94, wk 7=83, wk 8=80; amitriptyline—wk 1=116, wk 2=113, wk 3=111, wk 4=104, wk 5=102, wk 6=92, wk 7=89, wk 8=76; placebo—wk 1=124, wk 2=120, wk 3=116, wk 4=108, wk 5=97, wk 6=89, wk 7=94, wk 8=85.

Double-blind, multicenter, placebo-controlled, parallel study of antidepressant effects of ZOLOFT vs amitriptyline in 448 subjects who met DSM-III criteria for major depression. Symptoms were assessed after a single-blind, 7-day to 14-day placebo washout. Patients were randomly assigned to ZOLOFT, amitriptyline, or placebo for 8 wk. Daily dosage ranges: ZOLOFT—50-200 mg; amitriptyline—50-150 mg.

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ZOLOFT...Effective Continuation Therapy[2,3]

- Responders to ZOLOFT during an 8-week acute-treatment phase were randomized to continue on ZOLOFT or placebo
- A significantly lower relapse rate* was observed for patients treated with ZOLOFT compared with those on placebo, in an assessment of the first 8 weeks of the continuation period
- The effectiveness of ZOLOFT has not been systematically evaluated in long-term use for more than 16 weeks

ZOLOFT...Physician and Patient Confidence

- Low risk of toxicity in overdose
- ZOLOFT has a lower incidence of tricyclic-like side effects and a low incidence of nervousness, anxiety, and agitation
- The most common side effects of ZOLOFT include nausea, diarrhea or loose stools, tremor, insomnia, somnolence, and dry mouth

ZOLOFT...Once-Daily Dosing May Enhance Patient Compliance

- ZOLOFT is available in 50-mg and 100-mg scored tablets for flexible, once-daily dosing
- ZOLOFT can be taken in the morning or evening, with or without food

*Relapsing patients were those who became ill (CGI-Severity score ≥4 at any visit) during the double-blind continuation phase and who remained ill during the rest of their time in the study.

Responders in an 8-wk, open-label, acute-treatment study entered a double-blind continuation phase and were randomized to ZOLOFT or placebo. ZOLOFT daily dosage range: 50-200 mg.

References: **1.** Reimherr FW, Chouinard G, Cohn CK, et al. Antidepressant efficacy of sertraline: a double-blind, placebo- and amitriptyline-controlled, multicenter comparison study in outpatients with major depression. *J Clin Psychiatry.* 1990;51(12, suppl B):18-27. **2.** Data on file. Pfizer Inc, New York, NY. **3.** Doogan DP, Caillard V. Sertraline in the prevention of depression. *Br J Psychiatry.* 1992;160:217-222.

Please see brief summary of prescribing information on adjacent page.

ONCE-A-DAY

Zoloft™

(sertraline HCl) 50 mg, 100 mg scored tablets

BRIEF SUMMARY

ZOLOFT™ (sertraline HCl)

INDICATIONS AND USAGE: ZOLOFT (sertraline hydrochloride) is indicated for the treatment of depression.

CONTRAINDICATIONS: None known. **WARNINGS: In patients receiving another serotonin reuptake inhibitor drug in combination with a monoamine oxidase inhibitor (MAOI), there have been reports of serious, sometimes fatal, reactions including hyperthermia, rigidity, myoclonus, autonomic instability with possible rapid fluctuations of vital signs, and mental status changes that include extreme agitation progressing to delirium and coma. These reactions have also been reported in patients who have recently discontinued that drug and have been started on an MAOI. Some cases presented with features resembling neuroleptic malignant syndrome. Therefore, it is recommended that ZOLOFT (sertraline hydrochloride) not be used in combination with an MAOI, or within 14 days of discontinuing treatment with an MAOI. Similarly, at least 14 days should be allowed after stopping ZOLOFT before starting an MAOI.**

PRECAUTIONS ***General:*** **Activation of Mania/Hypomania** - During premarketing testing, hypomania or mania occurred in approximately 0.4% of ZOLOFT (sertraline hydrochloride) treated patients. Activation of mania/hypomania has also been reported in a small proportion of patients with Major Affective Disorder treated with other marketed antidepressants. **Weight Loss** - Significant weight loss may be an undesirable result of treatment with sertraline for some patients, but on average, patients in controlled trials had minimal, 1 to 2 pound weight loss, versus smaller changes on placebo. Only rarely have sertraline patients been discontinued for weight loss. **Seizure** - ZOLOFT has not been evaluated in patients with a seizure disorder. These patients were excluded from clinical studies during the product's premarket testing. Accordingly, like other antidepressants, ZOLOFT should be introduced with care in epileptic patients. **Suicide** - The possibility of a suicide attempt is inherent in depression and may persist until significant remission occurs. Close supervision of high risk patients should accompany initial drug therapy. Prescriptions for ZOLOFT should be written for the smallest quantity of tablets consistent with good patient management, in order to reduce the risk of overdose. **Weak Uricosuric Effect** - ZOLOFT is associated with a mean decrease in serum uric acid of approximately 7%. The clinical significance of this weak uricosuric effect is unknown, and there have been no reports of acute renal failure with ZOLOFT. **Use in Patients with Concomitant Illness** - Clinical experience with ZOLOFT in patients with certain concomitant systemic illness is limited. Caution is advisable in using ZOLOFT in patients with diseases or conditions that could affect metabolism or hemodynamic responses. ZOLOFT has not been evaluated or used to any appreciable extent in patients with a recent history of myocardial infarction or unstable heart disease. Patients with these diagnoses were excluded from clinical studies during the product's premarket testing However, the electrocardiograms of 774 patients who received ZOLOFT in double-blind trials were evaluated and the data indicate that ZOLOFT is not associated with the development of significant ECG abnormalities. ZOLOFT is extensively metabolized by the liver. The pharmacokinetics of ZOLOFT have not been studied in patients with significant hepatic dysfunction nor have patients with significant hepatic dysfunction been evaluated during treatment with ZOLOFT. Accordingly, ZOLOFT should be used with caution in such patients. Since ZOLOFT is extensively metabolized, excretion of unchanged drug in urine is a minor route of elimination. However, until the pharmacokinetics of ZOLOFT have been studied in patients with renal impairment and until adequate numbers of patients with severe renal impairment have been evaluated during chronic treatment with ZOLOFT, it should be used with caution in such patients. **Interference with Cognitive and Motor Performance** - In controlled studies, ZOLOFT did not cause sedation and did not interfere with psychomotor performance. ***Information for Patients:*** Physicians are advised to discuss the following issues with patients for whom they prescribe ZOLOFT: Patients should be told that although ZOLOFT has not been shown to impair the ability of normal subjects to perform tasks requiring complex motor and mental skills in laboratory experiments, drugs that act upon the central nervous system may affect some individuals adversely. Patients should be told that although ZOLOFT has not been shown in experiments with normal subjects to increase the mental and motor skill impairments caused by alcohol, the concomitant use of ZOLOFT and alcohol in depressed patients is not advised. Patients should be told that while no adverse interaction of ZOLOFT with over-the-counter (OTC) drug products is known to occur, the potential for interaction exists. Thus, the use of any OTC product should be initiated cautiously according to the directions of use given for the OTC product. Patients should be advised to notify their physician if they become pregnant or intend to become pregnant during therapy. Patients should be advised to notify their physician if they are breast-feeding an infant. ***Laboratory Tests:*** None. ***Drug Interactions:*** **Potential Effects of Coadministration of Drugs Highly Bound to Plasma Proteins** - Because sertraline is tightly bound to plasma protein, the administration of ZOLOFT (sertraline hydrochloride) to a patient taking another drug which is tightly bound to protein (e.g., warfarin, digitoxin) may cause a shift in plasma concentrations potentially resulting in an adverse effect. Conversely, adverse effects may result from displacement of protein-bound ZOLOFT by other tightly bound drugs. In a study comparing prothrombin time AUC (0-120 hr) following dosing with warfarin (0.75 mg/kg) before and after 21 days of dosing with either ZOLOFT (50-200 mg/day) or placebo, there was a mean increase in prothrombin time of 8% relative to baseline for ZOLOFT compared to a 1% decrease for placebo ($p<0.02$). The normalization of prothrombin time for the ZOLOFT group was delayed compared to the placebo group. The clinical significance of this change is unknown. Accordingly, prothrombin time should be carefully monitored when ZOLOFT therapy is initiated or stopped. **CNS Active Drugs** - In a study comparing the disposition of intravenously administered diazepam before and after 21 days of dosing with either ZOLOFT (50 to 200 mg/day escalating dose) or placebo, there was a 32% decrease relative to baseline in diazepam clearance for the ZOLOFT group compared to a 19% decrease relative to baseline for the placebo group ($p<0.03$). There was a 23% increase in T_{max} for desmethyldiazepam in the ZOLOFT group compared to a 20% decrease in the placebo group ($p<0.03$). The clinical significance of these changes is unknown. In a placebo-controlled trial in normal volunteers, the administration of two doses of ZOLOFT did not significantly alter steady-state lithium levels or the renal clearance of lithium. Nonetheless, at this time, it is recommended that plasma lithium levels be monitored following initiation of ZOLOFT therapy with appropriate adjustments to the lithium dose. The risk of using ZOLOFT in combination with other CNS active drugs has not been systematically evaluated. Consequently, caution is advised if the concomitant administration of ZOLOFT and such drugs is required. **Hypoglycemic Drugs** - In a placebo-controlled trial in normal volunteers, administration of ZOLOFT for 22 days (including 200 mg/day for the final 13 days) caused a statistically significant 16% decrease from baseline in the clearance of tolbutamide following an intravenous 1000 mg dose. ZOLOFT administration did not noticeably change either the plasma protein binding or the apparent volume of distribution of tolbutamide, suggesting that the decreased clearance was due to a change in the metabolism of the drug. The clinical significance of this decrease in tolbutamide clearance is unknown. **Atenolol** - ZOLOFT (100 mg) when administered to 10 healthy male subjects had no effect on the beta-adrenergic blocking ability of atenolol. **Microsomal Enzyme Induction** - Preclinical studies have shown ZOLOFT to induce hepatic microsomal enzymes. In clinical studies ZOLOFT was shown to induce hepatic enzymes minimally as determined by a small (5%) but statistically significant decrease in antipyrine half-life following administration of 200 mg/day for 21 days. This small change in antipyrine half-life reflects a clinically insignificant change in hepatic metabolism. **Electroconvulsive Therapy** - There are no clinical studies establishing the risks or benefits of the combined use of electroconvulsive therapy (ECT) and ZOLOFT. **Alcohol** - Although ZOLOFT did not potentiate the cognitive and psychomotor effects of alcohol in experiments with normal subjects, the concomitant use of ZOLOFT and alcohol in depressed patients is not recommended. ***Carcinogenesis, Mutagenesis, Impairment of Fertility:*** Lifetime carcinogenicity studies were carried out in CD-1 mice and Long-Evans rats at doses up to 40 mg/kg in mice (10 times on a mg/kg basis and the same on a mg/m^2 basis as the maximum recommended human dose) and at doses up to 40 mg/kg in rats (10 times on a mg/kg basis and 2 times on a mg/m^2 basis, the maximum recommended human dose). There was a dose-related increase in the incidence of liver adenomas in male mice receiving sertraline at 10-40 mg/kg No increase was seen in female mice or in rats of either sex receiving the same treatments, nor was there an increase in hepatocellular carcinomas. Liver adenomas have a variable rate of spontaneous occurrence in the CD-1 mouse and are of unknown significance to humans. There was an increase in follicular adenomas of the thyroid in female rats receiving sertraline at 40 mg/kg; this was not accompanied by thyroid hyperplasia. While there was an increase in uterine adenocarcinomas in rats receiving sertraline at 10-40 mg/kg compared to placebo controls, this effect was not clearly drug related. Sertraline had no genotoxic effects, with or without metabolic activation, based on the following assays: bacterial mutation assay; mouse lymphoma mutation assay; and tests for cytogenetic aberrations *in vivo* in mouse bone marrow and *in vitro* in human lymphocytes. A decrease in fertility was seen in one of two rat studies at a dose of 80 mg/kg (20 times the maximum human dose on a mg/kg basis and 4 times on a mg/m^2 basis). ***Pregnancy—Pregnancy Category B:*** **Teratogenic Effects** - Reproduction studies have been performed in rats and rabbits at doses up to approximately 20 times and 10 times the maximum daily human mg/kg dose (4 to 4.5 times the mg/m^2 dose), respectively. There was no evidence of teratogenicity at any dose level. At doses approximately 2.5-10 times the maximum daily human mg/kg dose, sertraline was associated with delayed ossification in fetuses, probably secondary to effects on the dams. There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. **Non-teratogenic Effects** - There was also decreased neonatal survival following maternal administration of sertraline at doses as low as approximately 5 times the maximum human mg/kg dose. The decrease in pup survival was shown to be most probably due to *in utero* exposure to sertraline. The clinical significance of these effects is unknown. **Labor and Delivery** - The effect of ZOLOFT on labor and delivery in humans is unknown. **Nursing Mothers** - It is not known whether, and if so in what amount, sertraline or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when ZOLOFT is administered to a nursing woman. **Pediatric Use** - Safety and effectiveness in children have not been established. **Geriatric Use** - Several hundred elderly patients have participated in clinical studies with ZOLOFT. The pattern of adverse reactions in the elderly was similar to that in younger patients. **ADVERSE REACTIONS** ***Commonly Observed:*** The most commonly observed adverse events associated with the use of ZOLOFT (sertraline hydrochloride) and not seen at an equivalent incidence among placebo-treated patients were: gastrointestinal complaints, including nausea, diarrhea/loose stools and dyspepsia; tremor; dizziness; insomnia; somnolence; increased sweating; dry mouth; and male sexual dysfunction (primarily ejaculatory delay). ***Associated with Discontinuation of Treatment:*** Fifteen percent of 2710 subjects who received ZOLOFT in premarketing multiple dose clinical trials discontinued treatment due to an adverse event. The more common events (reported by at least 1% of subjects) associated with discontinuation included agitation, insomnia, male sexual dysfunction (primarily ejaculatory delay), somnolence, dizziness, headache, tremor, anorexia, diarrhea/loose stools, nausea, and fatigue. ***Incidence in Controlled Clinical Trials:*** The table that follows enumerates adverse events that occurred at a frequency of 1% or more among ZOLOFT patients who participated in controlled trials comparing titrated ZOLOFT with placebo. Most patients received doses of 50 to 200 mg per day.

Treatment-Emergent Adverse Experience Incidence in Placebo-Controlled Clinical Trials*

Adverse Experience	ZOLOFT (N=861)	Placebo (N=853)	Adverse Experience	ZOLOFT (N=861)	Placebo (N=853)
	(Percent of Patients Reporting)			(Percent of Patients Reporting)	
Autonomic Nervous System Disorders			Hot Flushes	2.2	0.5
Mouth Dry	16.3	9.3	Fever	1.6	0.6
Sweating Increased	8.4	2.9	Back Pain	1.5	0.9
Cardiovascular			**Metabolic and Nutritional Disorders**		
Palpitations	3.5	1.6	Thirst	1.4	0.9
Chest Pain	1.0	1.6	**Musculoskeletal System Disorders**		
Central and Peripheral Nervous System Disorders			Myalgia	1.7	1.5
			Psychiatric Disorders		
Headache	20.3	19.0	Insomnia	16.4	8.8
Dizziness	11.7	6.7	Sexual Dysfunction-Male (1)	15.5	2.2
Tremor	10.7	2.7	Somnolence	13.4	5.9
Paresthesia	2.0	1.8	Agitation	5.6	4.0
Hypoesthesia	1.7	0.6	Nervousness	3.4	1.9
Twitching	1.4	0.1	Anxiety	2.6	1.3
Hypertonia	1.3	0.4	Yawning	1.9	0.2
Disorders of Skin and Appendages			Sexual Dysfunction-Female (2)	1.7	0.2
Rash	2.1	1.5	Concentration Impaired	1.3	0.5
Gastrointestinal Disorders			**Reproductive**		
Nausea	26.1	11.8	Menstrual Disorder (2)	1.0	0.5
Diarrhea/Loose Stools	17.7	9.3	**Respiratory System Disorders**		
Constipation	8.4	6.3	Rhinitis	2.0	1.5
Dyspepsia	6.0	2.8	Pharyngitis	1.2	0.9
Vomiting	3.8	1.8	**Special Senses**		
Flatulence	3.3	2.5	Vision Abnormal	4.2	2.1
Anorexia	2.8	1.6	Tinnitus	1.4	1.1
Abdominal Pain	2.4	2.2	Taste Perversion	1.2	0.7
Appetite Increased	1.3	0.9	**Urinary System Disorders**		
General			Micturition Frequency	2.0	1.2
Fatigue	10.6	8.1	Micturition Disorder	1.4	0.5

*Events reported by at least 1% of patients treated with ZOLOFT are included.

(1) - % based on male patients only: 271 ZOLOFT (primarily ejaculatory delay) and 271 placebo patients.

(2) - % based on female patients only: 590 ZOLOFT and 582 placebo patients.

Other Events Observed During the Premarketing Evaluation of ZOLOFT (sertraline hydrochloride): During its premarketing assessment, multiple doses of ZOLOFT were administered to approximately 2700 subjects. Events are further categorized by body system and listed in order of decreasing frequency according to the following definitions: frequent adverse events are those occurring on one or more occasions in at least 1/100 patients (only those not already listed in the tabulated results from placebo-controlled trials appear in this listing); infrequent adverse events are those occurring in 1/100 to 1/1000 patients; rare events are those occurring in fewer than 1/1000 patients. Events of major clinical importance are also described in the PRECAUTIONS section. **Autonomic Nervous System Disorders**—*Infrequent:* flushing, mydriasis, increased saliva, cold clammy skin; *Rare:* pallor. **Cardiovascular**—*Infrequent:* postural dizziness, hypertension, hypotension, postural hypotension, edema, dependent edema, periorbital edema, peripheral edema, peripheral ischemia, syncope, tachycardia; *Rare:* precordial chest pain, substernal chest pain, aggravated hypertension, myocardial infarction, varicose veins. **Central and Peripheral Nervous System Disorders**—*Frequent:* confusion; *Infrequent:* ataxia, abnormal coordination, abnormal gait, hyperesthesia, hyperkinesia, hypokinesia, migraine, nystagmus, vertigo; *Rare:* local anesthesia, coma, convulsions, dyskinesia, dysphonia, hyporeflexia, hypotonia, ptosis. **Disorders of Skin and Appendages**—*Infrequent:* acne, alopecia, pruritus, erythematous rash, maculopapular rash, dry skin; *Rare:* bullous eruption, dermatitis, erythema multiforme, abnormal hair texture, hypertrichosis, photosensitivity reaction, follicular rash, skin discoloration, abnormal skin odor, urticaria. **Endocrine Disorders**—*Rare:* exophthalmos, gynecomastia. **Gastrointestinal Disorders**—*Infrequent:* dysphagia, eructation; *Rare:* diverticulitis, fecal incontinence, gastritis, gastroenteritis, glossitis, gum hyperplasia, hemorrhoids, hiccup, melena, hemorrhagic peptic ulcer, proctitis, stomatitis, ulcerative stomatitis, tenesmus, tongue edema, tongue ulceration. **General**—*Frequent:* asthenia; *Infrequent:* malaise, generalized edema, rigors, weight decrease, weight increase; *Rare:* enlarged abdomen, halitosis, otitis media, aphthous stomatitis. **Hematopoietic and Lymphatic**—*Infrequent:* lymphadenopathy, purpura; *Rare:* anemia, anterior chamber eye hemorrhage. **Metabolic and Nutritional Disorders**—*Rare:* dehydration, hypercholesteremia, hypoglycemia. **Musculoskeletal System Disorders**—*Infrequent:* arthralgia, arthrosis, dystonia, muscle cramps, muscle weakness; *Rare:* hernia. **Psychiatric Disorders**—*Infrequent:* abnormal dreams, aggressive reaction, amnesia, apathy, delusion, depersonalization, depression, aggravated depression, emotional lability, euphoria, hallucination, neurosis, paranoid reaction, suicide ideation and attempt, teeth-grinding, abnormal thinking; *Rare:* hysteria, somnambulism, withdrawal syndrome. **Reproductive**—*Infrequent:* dysmenorrhea (2), intermenstrual bleeding (2); *Rare:* amenorrhea (2), balanoposthitis (1), breast enlargement (2), female breast pain (2), leukorrhea (2), menorrhagia (2), atrophic vaginitis (2).

(1) - % based on male subjects only: 1005; (2) - % based on female subjects only: 1705.

Respiratory System Disorders—*Infrequent:* bronchospasm, coughing, dyspnea, epistaxis; *Rare:* bradypnea, hyperventilation, sinusitis, stridor. **Special Senses**—*Infrequent.* abnormal accommodation, conjunctivitis, diplopia, earache, eye pain, xerophthalmia; *Rare:* abnormal lacrimation, photophobia, visual field defect. **Urinary System Disorders**—*Infrequent:* dysuria, face edema, nocturia, polyuria, urinary incontinence; *Rare:* oliguria, renal pain, urinary retention. **Laboratory Tests** In man, asymptomatic elevations in serum transaminases (SGOT [or AST] and SGPT [or ALT]) have been reported infrequently (approximately 0.8%) in association with ZOLOFT administration. These hepatic enzyme elevations usually occurred within the first 1 to 9 weeks of drug treatment and promptly diminished upon drug discontinuation. ZOLOFT therapy was associated with small mean increases in total cholesterol (approximately 3%) and triglycerides (approximately 5%), and a small mean decrease in serum uric acid (approximately 7%) of no apparent clinical importance. **DRUG ABUSE AND DEPENDENCE** ***Controlled Substance Class*** - ZOLOFT (sertraline hydrochloride) is not a controlled substance. ***Physical and Psychological Dependence*** - ZOLOFT has not been systematically studied, in animals or humans, for its potential for abuse, tolerance, or physical dependence. However, the premarketing clinical experience with ZOLOFT did not reveal any tendency for a withdrawal syndrome or any drug-seeking behavior. As with any new CNS active drug, physicians should carefully evaluate patients for history of drug abuse and follow such patients closely, observing them for signs of ZOLOFT misuse or abuse (e.g., development of tolerance, incrementation of dose, drug-seeking behavior). **OVERDOSAGE** ***Human Experience*** - There have been 3 cases of ZOLOFT (sertraline hydrochloride) overdosage (approximately 750-2,100 mg). No specific therapy was required for any of the 3 patients, all of whom recovered completely. ***Management of Overdoses*** - Establish and maintain an airway, insure adequate oxygenation and ventilation. Activated charcoal, which may be used with sorbitol, may be as or more effective than emesis or lavage, and should be considered in treating overdose. Cardiac and vital signs monitoring is recommended along with general symptomatic and supportive measures. There are no specific antidotes for ZOLOFT. Due to the large volume of distribution of ZOLOFT, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. In managing overdosage, consider the possibility of multiple drug involvement. The physician should consider contacting a poison control center on the treatment of any overdose.

Pfizer

Roerig
Pratt
Pharmaceuticals

(*continued from p8A*)

biology and Infectious Diseases. Sheraton Inn. Contact: Office of Continuing Education, SUNY Health Science Center at Syracuse, 750 E Adams St, Syracuse, NY 13210. Tel: (315) 464-4606.

MANHATTAN

May 15–16. **Stimulation of Brain and Spinal Cord.** 10 Cat I Credits. NYU Medical Center Alumni Hall. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295.

May 18–22. **Consultant's Course in Cardiology.** Mt Sinai Medical Center. Contact: Registration Secretary, Extramural Programs Dept, American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814. Tel: 1-800-257-4739.

May 29. **Improving Fitness and Lifestyle in the Physically Disabled With Cardiovascular Disease.** Beth Israel Medical Center. Contact: Medical Education Office, Beth Israel Medical Center, First Ave at 16th St, New York, NY 10003. Tel: (212) 420-2000.

SOUTHAMPTON

May 15. **Approaches to Treatment of Patients with Ventricular Tachycardia/ Automatic Implantable Cardiovertor Defibrillators.** Southampton Hotel. Contact: Medical Staff Office, Southampton Hospital, 240 Meeting House Lane, Southampton, NY 11968. Tel: (516) 726-8305.

AROUND THE NATION

CALIFORNIA

May 4–8. **MRI: A State of the Art Clinical Review.** Ritz-Carlton, San Francisco. *May 8–10.* **Advanced Seminars in Diagnostic Imaging.** Ritz Carlton, Laguna Niguel. *May 21–24.* **6th Annual San Diego Duplex Imaging.** Le Meridien Hotel, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

May 14–16. **25th Annual Advances and Controversies in Clinical Pediatrics.** Fairmont Hotel. *May 21–22.* **Current Issues in Anatomic Pathology: 1991.** Union Square Holiday Inn, San Francisco. Contact: Extended Programs in Medical Education, Room LS-105, University of California School of Medicine,

(*continued on p15A*)

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[1] Data on file, Lilly Research Laboratories.

See accompanying page for prescribing information.

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Indications and Usage: 1. *Active duodenal ulcer*–for up to 8 weeks of treatment at a dosage of 300 mg h.s. or 150 mg b.i.d. Most patients heal within 4 weeks.

2. *Maintenance therapy*–for healed duodenal ulcer patients at a dosage of 150 mg h.s. at bedtime. The consequences of therapy with Axid for longer than 1 year are not known.

3. *Gastroesophageal reflux disease (GERD)*–for up to 12 weeks of treatment of endoscopically diagnosed esophagitis, including erosive and ulcerative esophagitis, and associated heartburn at a dosage of 150 mg b.i.d.

Contraindication: Known hypersensitivity to the drug. Because cross sensitivity in this class of compounds has been observed, H_2-receptor antagonists, including Axid, should not be administered to patients with a history of hypersensitivity to other H_2-receptor antagonists.

Precautions: *General*–1. Symptomatic response to nizatidine therapy does not preclude the presence of gastric malignancy.

2. Dosage should be reduced in patients with moderate to severe renal insufficiency.

3. In patients with normal renal function and uncomplicated hepatic dysfunction, the disposition of nizatidine is similar to that in normal subjects.

Laboratory Tests–False-positive tests for urobilinogen with Multistix® may occur during therapy.

Drug Interactions–No interactions have been observed with theophylline, chlordiazepoxide, lorazepam, lidocaine, phenytoin, and warfarin. Axid does not inhibit the cytochrome P-450 enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur. In patients given very high doses (3,900 mg) of aspirin daily, increased serum salicylate levels were seen when nizatidine, 150 mg b.i.d., was administered concurrently.

Carcinogenesis, Mutagenesis, Impairment of Fertility–A 2-year oral carcinogenicity study in rats with doses as high as 500 mg/kg/day (about 80 times the recommended daily therapeutic dose) showed no evidence of a carcinogenic effect. There was a dose-related increase in the density of enterochromaffin-like (ECL) cells in the gastric oxyntic mucosa. In a 2-year study in mice, there was no evidence of a carcinogenic effect in male mice, although hyperplastic nodules of the liver were increased in the high-dose males as compared with placebo. Female mice given the high dose of Axid (2,000 mg/kg/day, about 330 times the human dose) showed marginally statistically significant increases in hepatic carcinoma and hepatic nodular hyperplasia with no numerical increase seen in any of the other dose groups.The rate of hepatic carcinoma in the high-dose animals was within the historical control limits seen for the strain of mice used. The female mice were given a dose larger than the maximum tolerated dose, as indicated by excessive (30%) weight decrement as compared with concurrent controls and evidence of mild liver injury (transaminase elevations). The occurrence of a marginal finding at high dose only in animals given an excessive and somewhat hepatotoxic dose, with no evidence of a carcinogenic effect in rats, male mice, and female mice (given up to 360 mg/kg/day, about 60 times the human dose), and a negative mutagenicity battery are not considered evidence of a carcinogenic potential for Axid.

Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy–Teratogenic Effects–Pregnancy Category C–Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers–Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use–Safety and effectiveness in children have not been established.

Use in Elderly Patients–Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic–Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular–In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS–Rare cases of reversible mental confusion have been reported.

Endocrine–Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic–Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental–Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity–As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other–Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

PV 2093 AMP [101591]

Additional information available to the profession on request.

Lilly

Eli Lilly and Company
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46285

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(*continued from p13A*)

San Francisco, CA 94143. Tel: (415) 476-4251.

May 30–Jun 3. **27th Annual Meeting of the Association for the Advancement of Medical Instrumentation.** Marriott Hotel, Anaheim. Contact: AAMI, PO Box 2942, Merrifield, VA 22116. Tel: 1-800-332-2264.

CHICAGO

May 7–9. **Second Annual Review and Update Course in Cardiovascular Medicine.** Hotel Inter-Continenal, Chicago. Contact: Conference Administrator, Center for Bio-Medical Communication, Inc, 80 W Madison Ave, Dumont, NJ 07628. Tel: (201) 385-8080.

DISTRICT OF COLUMBIA

May 6–9. **15th Annual National Conference on Rural Health.** Hyatt Regency Crystal City Hotel, Washington. Contact: National Rural Health Association, 301 E Armour Blvd, Suite 420, Kansas City, MO 64111. Tel: (816) 756-3140.

May 13–15. **Clinical Auscultation of the Heart.** Georgetown University Medical Center. Washington. Contact: Registration Secretary, Extramural Programs Dept, American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 10814. Tel: 1-800-257-4739.

May 31–Jun 6. **Intensive Bioethics Course XVIII.** Georgetown University, Washington. Contact: LeRoy Walters, PhD, Director, Center for Bioethics, Kennedy Institute of Ethics, Georgetown University, Washington, DC 20057.

FLORIDA

May 15–17. **Positron Emission Tomography: Applications in Cancer Management.** Hilton at Walt Disney World Village. Lake Buena Vista. Contact: American College of Radiology, Educational Services Division, 1891 Preston White Dr, Reston, VA 22091. Tel: 1-800-ACR-LINE ext 4961.

LOUISIANA

May 6–10. **Spring Symposium on Back Pain.** New Orleans Sheraton Hotel. Contact: American Back Society, 2647 E 14th St, Oakland, CA 94601. Tel: (510) 536-9929.

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May 3. **Eighth Special Regional Symposium on the Modern Treatment of**

(*continued on p20A*)

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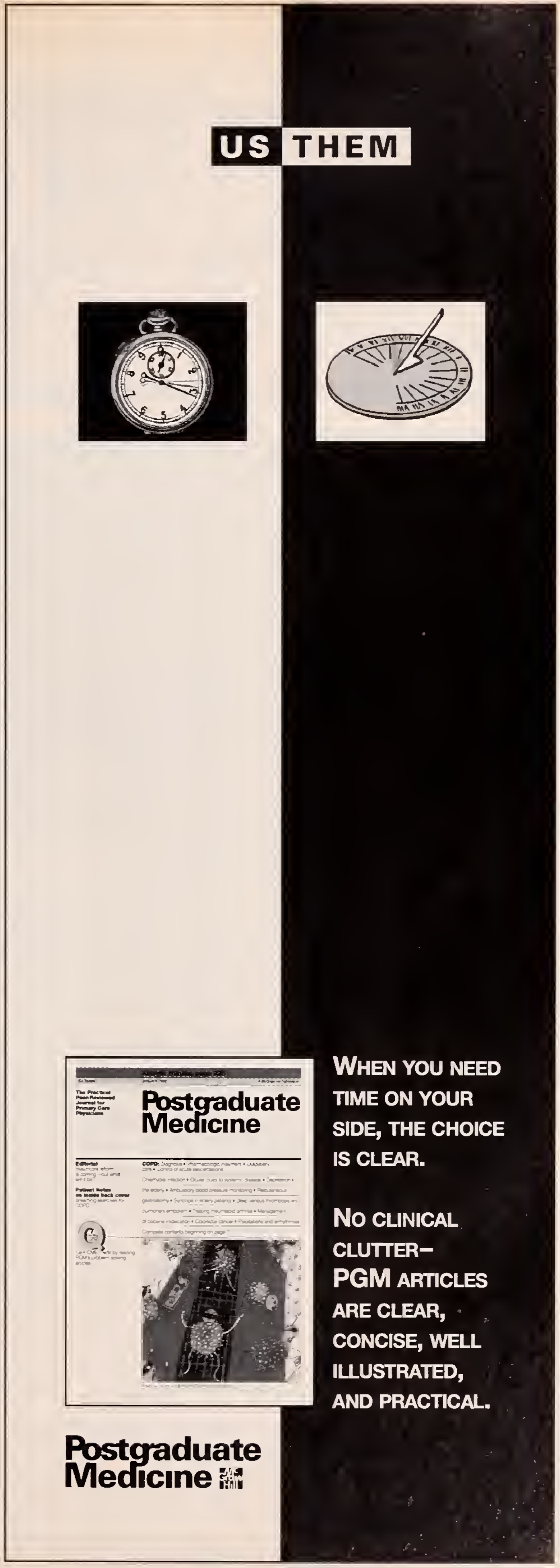

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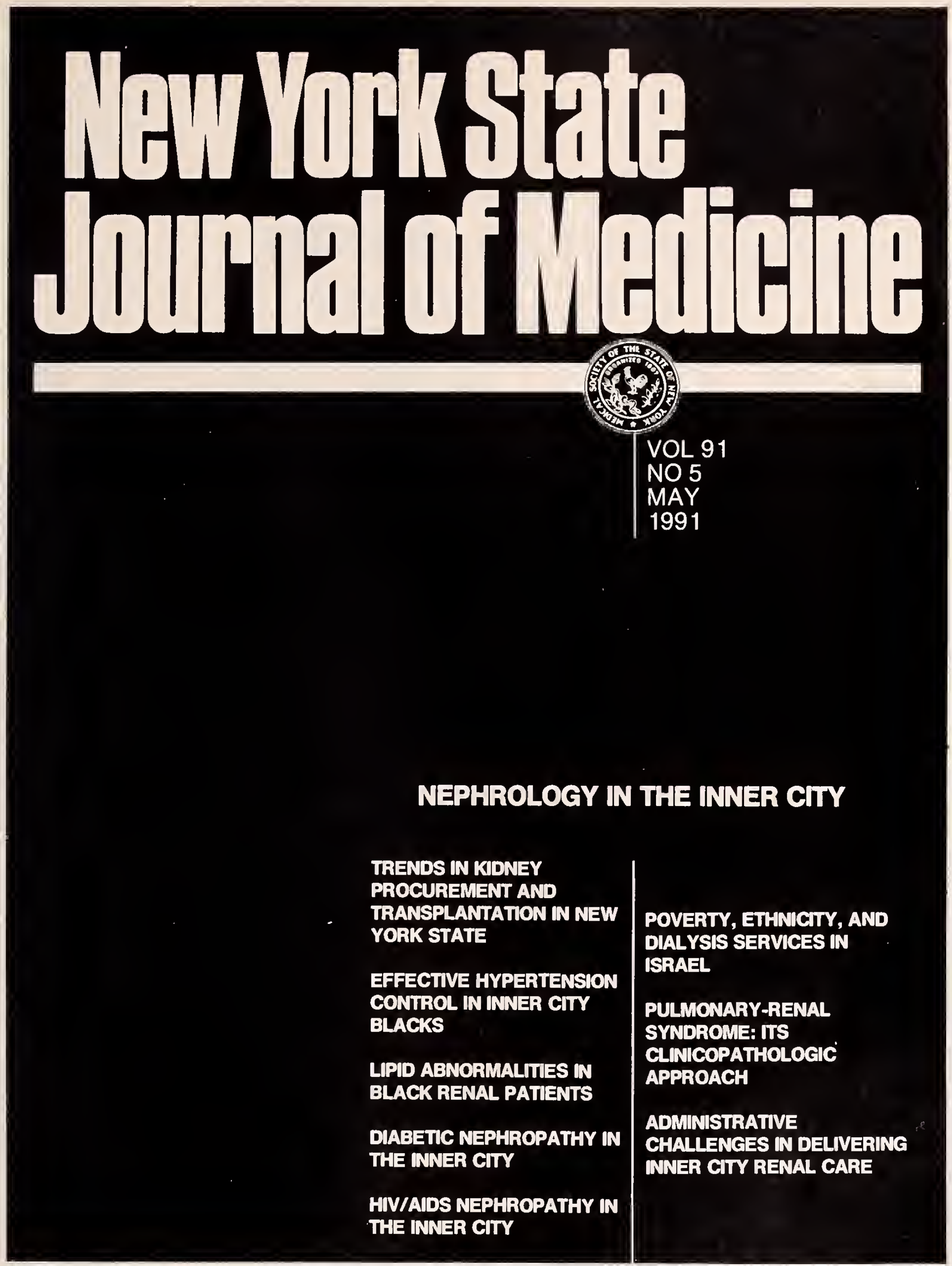

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VOL 91
NO 5
MAY
1991

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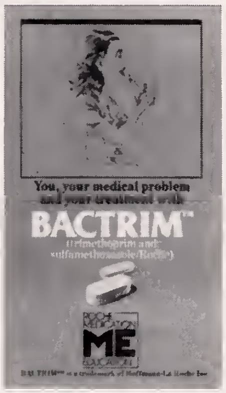
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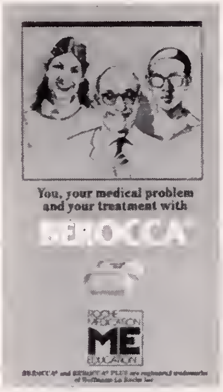
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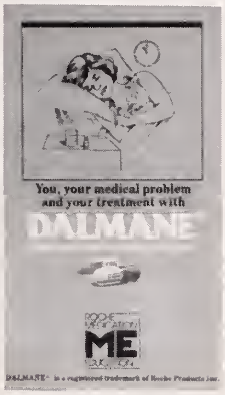
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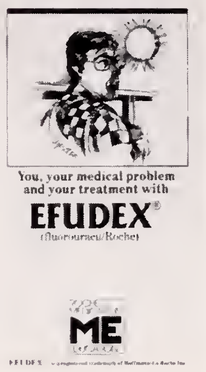

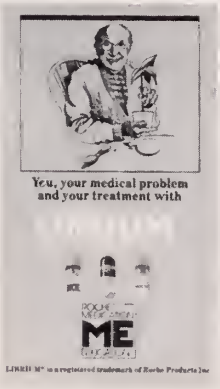
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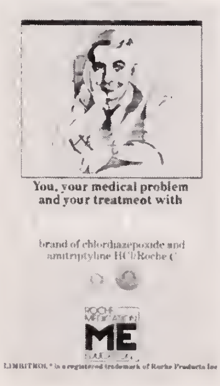
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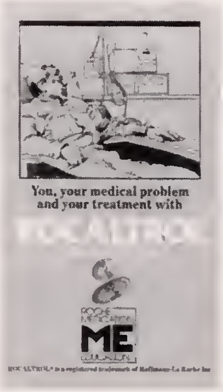
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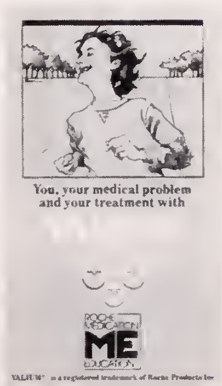
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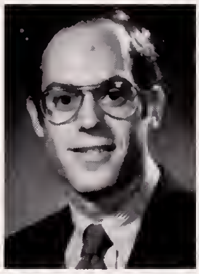
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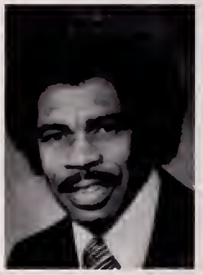
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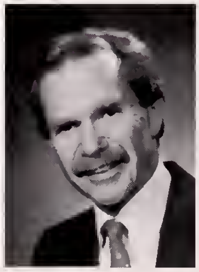
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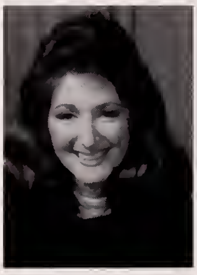
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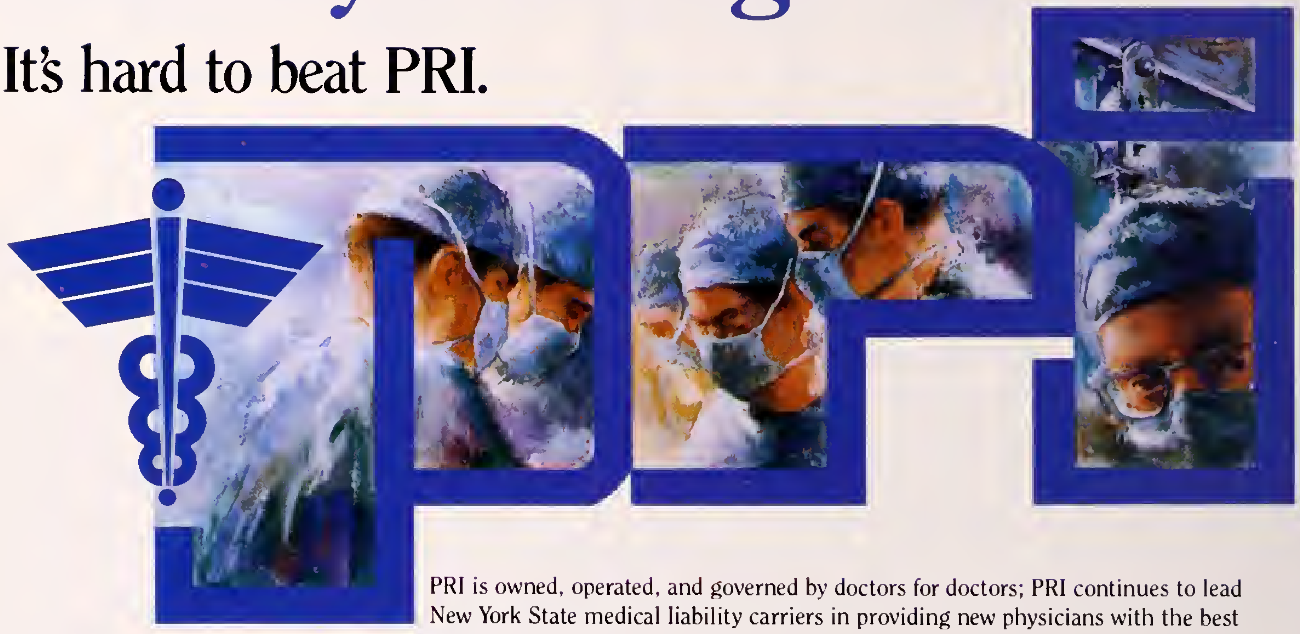

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Axid was not mutagenic in a battery of tests performed to evaluate its potential genetic toxicity, including bacterial mutation tests, unscheduled DNA synthesis, sister chromatid exchange, mouse lymphoma assay, chromosome aberration tests, and a micronucleus test.

In a 2-generation, perinatal and postnatal fertility study in rats, doses of nizatidine up to 650 mg/kg/day produced no adverse effects on the reproductive performance of parental animals or their progeny.

Pregnancy—Teratogenic Effects—Pregnancy Category C—Oral reproduction studies in rats at doses up to 300 times the human dose and in Dutch Belted rabbits at doses up to 55 times the human dose revealed no evidence of impaired fertility or teratogenic effect; but, at a dose equivalent to 300 times the human dose, treated rabbits had abortions, decreased number of live fetuses, and depressed fetal weights. On intravenous administration to pregnant New Zealand White rabbits, nizatidine at 20 mg/kg produced cardiac enlargement, coarctation of the aortic arch, and cutaneous edema in 1 fetus, and at 50 mg/kg, it produced ventricular anomaly, distended abdomen, spina bifida, hydrocephaly, and enlarged heart in 1 fetus. There are, however, no adequate and well-controlled studies in pregnant women. It is also not known whether nizatidine can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Nizatidine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers—Studies in lactating women have shown that 0.1% of an oral dose is secreted in human milk in proportion to plasma concentrations. Because of growth depression in pups reared by treated lactating rats, a decision should be made whether to discontinue nursing or the drug, taking into account the importance of the drug to the mother.

Pediatric Use—Safety and effectiveness in children have not been established.

Use in Elderly Patients—Healing rates in elderly patients were similar to those in younger age groups as were the rates of adverse events and laboratory test abnormalities. Age alone may not be an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function.

Adverse Reactions: Worldwide, controlled clinical trials included over 6,000 patients given nizatidine in studies of varying durations. Placebo-controlled trials in the United States and Canada included over 2,600 patients given nizatidine and over 1,700 given placebo. Among the adverse events in these placebo-controlled trials, only anemia (0.2% vs 0%) and urticaria (0.5% vs 0.1%) were significantly more common in the nizatidine group. Of the adverse events that occurred at a frequency of 1% or more, there was no statistically significant difference between Axid and placebo in the incidence of any of these events (see package insert for complete information).

A variety of less common events were also reported; it was not possible to determine whether these were caused by nizatidine.

Hepatic—Hepatocellular injury (elevated liver enzyme tests or alkaline phosphatase) possibly or probably related to nizatidine occurred in some patients. In some cases, there was marked elevation (>500 IU/L) in SGOT or SGPT and, in a single instance, SGPT was >2,000 IU/L. The incidence of elevated liver enzymes overall and elevations of up to 3 times the upper limit of normal, however, did not significantly differ from that in placebo patients. All abnormalities were reversible after discontinuation of Axid. Since market introduction, hepatitis and jaundice have been reported. Rare cases of cholestatic or mixed hepatocellular and cholestatic injury with jaundice have been reported with reversal of the abnormalities after discontinuation of Axid.

Cardiovascular—In clinical pharmacology studies, short episodes of asymptomatic ventricular tachycardia occurred in 2 individuals administered Axid and in 3 untreated subjects.

CNS—Rare cases of reversible mental confusion have been reported.

Endocrine—Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity due to nizatidine. Impotence and decreased libido were reported with similar frequency by patients on nizatidine and those on placebo. Gynecomastia has been reported rarely.

Hematologic—Anemia was reported significantly more frequently in nizatidine than in placebo-treated patients. Fatal thrombocytopenia was reported in a patient treated with nizatidine and another H_2-receptor antagonist. This patient had previously experienced thrombocytopenia while taking other drugs. Rare cases of thrombocytopenic purpura have been reported.

Integumental—Urticaria was reported significantly more frequently in nizatidine- than in placebo-treated patients. Rash and exfoliative dermatitis were also reported.

Hypersensitivity—As with other H_2-receptor antagonists, rare cases of anaphylaxis following nizatidine administration have been reported. Rare episodes of hypersensitivity reactions (eg, bronchospasm, laryngeal edema, rash, and eosinophilia) have been reported.

Other—Hyperuricemia unassociated with gout or nephrolithiasis was reported. Eosinophilia, fever, and nausea related to nizatidine have been reported.

Overdosage: Overdoses of Axid have been reported rarely. If overdosage occurs, activated charcoal, emesis, or lavage should be considered along with clinical monitoring and supportive therapy. The ability of hemodialysis to remove nizatidine from the body has not been conclusively demonstrated; however, due to its large volume of distribution, nizatidine is not expected to be efficiently removed from the body by this method.

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Jun 8–12. **12th Annual Emergency Medicine Seminar.** *Jun 12–13.* **Diagnostic and Operative Sinus Endoscopy.** *Jun 22–25.* **15th Annual Review: Selected Topics in Pediatrics.** NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295.

SYRACUSE

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Jun 4–7. **Advances in Aesthetic and Reconstructive Breast Surgery.** Ritz-Carlton Hotel, St Louis. Contact: Continuing Medical Education, Washington University School of Medicine, Box 8063, 660 S Euclid, St Louis, MO 63110. Tel: (314) 362-6893. ☐

New York State Journal of Medicine

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

MAY 1992 / Volume 92, Number 5

Contents

COMMENTARIES

175 **Intensive care for the elderly: Should the gates remain open?**
ROBERT D. BRANDSTETTER, MD

177 **Osseous sarcoidosis, radiologic imaging, and physician interaction**
CHARLES S. RESNIK, MD

RESEARCH PAPERS

179 **Serious complications of laparoscopic cholecystectomy in New York State**
RAPHAEL P. NENNER, MD; PASCAL JAMES IMPERATO, MD; CHARLES M. ALCORN, DSc

181 **Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals**
JUDITH C. AHRONHEIM, MD; SATHYA MAHESWARAN, MD; CARL ROSENBERG, PhD

185 **Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987**
ALJE VENNEMA, MD

SPECIAL ARTICLE

189 **Pigeons as a remedy (*segulah*) for jaundice**
FRED ROSNER, MD

CLINICAL CONFERENCE

193 **Pulmonary cavitary disease**

HISTORY

199 **The story of Lassa fever—Part I: Discovering the disease**
JOHN D. FRAME, MD

203 **A history of the Women's Medical Society of New York State**
JEAN M. HOEFER, MS, MA; LESLIE J. KOHMAN, MD

(continued on p 6a)

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New York State
Journal of Medicine

CASE REPORTS

213 **Lytic osseous destruction in vertebral sarcoidosis**
SHAHIN SHAIKH, MD; AYMAN O. SOUBANI, MD; PETER RUMORE, MD; ERIC CANTOS, MD; ZEBA JELVEH, MD

214 **Clavicular resection in the exposure and resection of malignant tumors**
BUHPENDRA K. GUPTA, MD; GEORGE C. ZOGRAFOS, MD; CONSTANTINE KARAKOUSIS, MD, PHD

217 **Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction**
STEVEN A. KAPLAN, MD; IRA KOHN, MD; E. STEPHEN AMIS, JR, MD; KATHLEEN M. O'TOOLE, MD; RONDA BIXON, MD; JOHN GIELLA, MD

219 **Neutropenia induced by low-dose captopril**
ZOBIDATTE MOUSSA, MD; THOMAS SCHIANO, MD; GIUSEPPE SPATOLIATORE, MD; SHAMIN SALMIN, MD

LETTERS TO THE EDITOR

221 **Treatment of human immunodeficiency virus associated with oral apthous ulcers with inhaled steroids**
EDWARD K. CHAPNICK, MD; JEREMY D. GRADON, MB; LARRY I. LUTWICK, MD

221 **Hyperbilirubinemia in the term infant**
PAUL R. JOSEPH, MD

222 **In lieu of a plaque**
SALOMEA KAPE, MD

224 LEADS FROM EPIDEMIOLOGY NOTES

225 BOOK REVIEWS

229 BOOKS RECEIVED

230 OBITUARY LISTINGS

3A MEDICAL MEETINGS AND LECTURES

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New York State
Journal of Medicine

COMMENTARIES

Intensive care for the elderly: Should the gates remain open?

Probably no where else in medicine is the art-of-the-science practiced more than in the intensive care unit (ICU). This is recognizing that in the past twenty years considerable knowledge has been gathered concerning the care of critically ill patients. Critical care units serve as a high-technology resource to monitor patients at risk for the development of life-threatening deterioration and intervene to support ventilation or circulation. The treatment of critically ill patients in ICUs requires a systematic diagnostic and therapeutic approach, because multiple organ systems are often involved in the pathological processes that cause the patient's illness.

ICUs of one type or another (eg, medical, pediatric, surgical) are present in over 90% of hospitals nationwide.[1] Most of these institutions report that up to 20% of the hospital costs are incurred in ICU utilization.[2] Accordingly, even though ICUs are barely 20-years old, the hospital climate is rapidly changing, acknowledging the impact that ICUs have in society. Rising health care costs, prospective payment, appropriate use of limited resources, and quality assurance issues are ever present. Hospital administrators are therefore anxious for tighter management of special care units and have suggested that ICU directors be the gate keepers of critical care.[3,4]

The most appropriate allocation of costly and limited intensive care resources remains a matter of considerable controversy. Such concerns led a National Institutes of Health panel to urge research to define which patients should not be admitted to ICUs.[5] Accordingly, it has been speculated that age may be a criterion influencing treatment decisions and admission requirements within critical care units.[6] Eleven percent of the population comprises those aged 65 years or older and are continuing to grow in numbers.[7] Close to 30% of the health care dollars spent in the United States is for illness in this group of patients.[7] Furthermore, many of ICUs report that 50% of the monitored patients are over the age of 65.[8] With these facts in mind, is there any justification for keeping the elderly out of the ICU?

A defensible approach to support such a position should center on published data concerning the types of illnesses incurred by the elderly, the management of such conditions, prognosis on arrival in ICUs, mortality and the important issue of outcome (quality of life on discharge from the hospital). At present there is little information available to us in the literature, to either medically, legally, or morally deny the elderly admission into an ICU.

A number of studies have looked at predicting the ultimate outcome of patients on admission to a medical intensive care unit. The Acute Physiology and Chronic Health Evaluation (APACHE),[9] the Mortality Prediction Model (MPM),[10] and the Simplified Acute Physiology Score (SAPS)[11] have been shown to be inaccurate for predicting individual outcome in the critically ill. Accordingly, it is recognized that these studies provide little advantage in clinical decision-making and that on the basis of age alone, the individual elderly patient does not have a higher mortality than those who are younger.[12] Other prognostic scoring systems have also been evaluated, and these too fail to demonstrate that the isolated elderly patient is at higher risk for dying in the ICU.[13] Most would therefore agree that age alone is not a statistically significant independent risk factor for predicting mortality in the ICU.[14,15]

Although age alone does not appear to affect mortality, older patients are likely to present with concomitant health problems that do affect their risk of dying in the ICU.[14] It is not uncommon for a patient to be admitted to ICU with one acute problem, and co-existing stable chronic disorders, only to die from an exacerbation of a chronic illness. This domino-like decompensation of other organ systems is a well recognized occurrence in the critically ill elderly patient.[14]

Certain disease processes appear to have a worse prognosis in the elderly when they are hospitalized in the ICU. Patients who suffer from the adult respiratory

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distress syndrome (ARDS) have close to a 70% mortality if over the age of 60, compared with less than 20% mortality if under 45 years of age.[16] Acknowledging that ARDS is an end result of any number of systemic insults and that the elderly display this domino-like decompensation of organs, it is not surprising that the mortality from this syndrome is high. Similarly, the mortality from myocardial infraction, and pneumonia are higher in the elderly when hospitalized in an ICU.[17,18]

Elderly survivors of critical care units appear to have a surprisingly favorable outcome. Several studies have shown that the number who survive to discharge from the hospital is acceptable and that the quality-of-life at home is good.[13,19,20] Employment status, emotional state, and leisure activities have been evaluated. Although decreased function has been noted with age following discharge from an emergency admission to the ICU, no more compromise of psychologic or physical activity than younger patients were reported.[16] Finally, the percentage of survivors fully recovered, freely ambulatory, fully alert, and productive, was the same in the elderly (65) compared with two other age groups (41, 41–65).[20]

From the studies available to us, it appears at present that we cannot predict which elderly patient will do poorly on arrival to ICU, can recognize several disease entities that may result in a higher mortality in the elderly (ARDS, MI, pneumonia), can anticipate a domino-like effect of decompensation of organs in some elderly patients who are admitted to ICU with an acute illness, and can expect many of the elderly who are discharged from ICU to home to experience any great deterioration in the quality-of-life.

Acknowledging that we know about the facts surrounding the elderly and ICU care, under certain circumstances should they be denied admission? The answer appears to be yes, but on an individual basis and in a compassionate manner.[21] It is here that the art-of-medicine takes over, acknowledging that to decline to place a patient in an ICU is good and sound therapy. This is the occasion on which more than a physician's scientific judgement (risks vs benefits) is required. Physicians, patients, and next of kin must bring together their ethical, religious, social, and economic understanding of the issues at hand. If properly done, so not to reduce the chance of survival by procrastination, it is also the point of no return. Those denied passage through the gates of the[22,23] ICU may thus receive every bit of supportive care with dignity. Finally, only with future studies will these gates that swing in both directions, attest to the true outcome of critically ill elderly patients.

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REFERENCES

1. American Hospital Association Hospital Statistics 1990 edition. Chicago, American Hospital Association 1990.
2. Shepard DS, Ghanotakis AJ: Hospital costs in Massachusetts: A report of the Massachusetts Funds Flow Project. Springfield, VA, National Information Service (HRP-0029335), 1979.
3. Ron A, Aronne LJ, Kalb PE, et al: The therapeutic efficacy of critical care units. Identifying sub-groups of patients who benefit. *Arch Intern Med* 1989; 149:338–341.
4. Terres D: Enter: The era of management. *Crit Care Med* 1985; 13:137.
5. NIH Consensus Development Conference on Critical Care Medicine. *Crit Care Med* 1983; 11:466–469.
6. McClish DK, Powell SH, Montenegro H, et al: The impact of age on utilization of intensive care resources. *JAGS* 1987; 35:983–988.
7. Kane R, Solomon D, Beck J, et al: The future need for geriatric manpower in the United States. *N Engl J Med* 1980; 302:1327–1330.
8. Katzman D, Powell SH, Montenegro H, et al: The impact of age on utilization of intensive care resources. *JAGS* 1987; 35:983–988.
9. Knaus WA, Zimmerman JE, Wagner DP, et al: APACHE—acute physiology and chronic health evaluation: A physiologically based classification system. *Crit Care Med* 1981; 9:591–597.
10. Teres D, Lemeshow S, Aurunim JS, et al: Validation of the mortality prediction model for ICU patients. *Crit Care Med* 1987; 15:208–212.
11. Legall JR, Loirat P, Alperovitch, et al: A simplified acute physiology score for ICU patients. *Crit Care Med* 1984; 12:975–977.
12. Schafer JH, Maurer A, Jochimsen F, et al: Outcome prediction models on admission in a medical intensive care unit: Do they predict individual outcome? *Crit Care Med* 1990; 18:1111–1117.
13. Seneff M, Knaus WA: Predicting patient outcome from intensive care: A guide to APACHE, MPM, SAPS, PRISM, and other prognostic scoring systems. *J Intensive Care Med* 1990; 5:33–52.
14. Krieger BP: Intensive care for the elderly. Is it different? Is it worth it? *Pulmonary Perspectives* 1991; 8:1–4.
15. Fedullo AJ, Swinburne AJ: Relationship of patient age to cost and survival in a medical ICU. *Crit Care Med* 1983; 11:155–159.
16. Gee MH, Gottlieb JE, Albertine KH, et al: Physiology of aging related to outcome in the adult respiratory distress syndrome. *J App Physiol* 1990; 69: 822–829.
17. Fedullo AJ, Swinburne AJ: Intensive care for elderly patients with myocardial infarction: Age-related comparison of clinical features and outcomes. *J Intensive Care Med* 1988; 3:265–271.
18. Murphy DJ, Meenay AM, Robinson BE, et al: Outcomes of cardiopulmonary resuscitation in the elderly. *Arch Intern Med* 1989; 111:199–205.
19. Mundt DJ, Gage RW, Lemeshow S, et al: Intensive care unit patient follow-up. Mortality, functional status and return to work at six months. *Arch Intern Med* 1989; 149:68–72.
20. Zaren B, Hedstrand U: Quality of life among long-term survivors of intensive care. *Crit Care Med* 1987; 15:743–747.
21. Strauss MJ, Logerfo JP, Yeltatzie JA, et al: Rationing of intensive care unit services: An every day occurrence. *JAMA* 1986; 255:1143–1146.
22. Sax FL, Charlson ME: Utilization of critical care units. A prospective study of physician triage and patient outcome. *Arch Int Med* 1987; 147:929–934.
23. Richards EP, Rathbun KC: Legal issues in critical care medicine. *J Intensive Care Med* 1986; 1:101–110.

Osseous sarcoidosis, radiologic imaging, and physician interaction

Sarcoidosis is a systemic granulomatous disease that affects multiple organ systems. Although its prevalence varies greatly among different populations, sarcoidosis is not uncommonly encountered in the United States, and it is reported to occur in approximately 40 of every 100,000 inhabitants of New York City.[1] The thoracic manifestations of sarcoidosis are well recognized, including lymphadenopathy and a variety of patterns of pulmonary parenchymal involvement. Musculoskeletal manifestations are also often encountered, occurring in up to 13% of patients.[2] Approximately 80%–90% of patients with osseous sarcoidosis have concomitant radiographically evident pulmonary disease, and most also demonstrate cutaneous manifestations of the disease (lupus pernio). It is particularly important to the clinician to be able to differentiate sarcoidosis, a relatively nonaggressive disorder, from more aggressive infections or malignancies.

The vast majority of osseous lesions in sarcoidosis occur in the hands and feet, although involvement of the the skull, facial bones, long bones, pelvis, ribs, and spine has been recorded. Basic radiologic patterns of bony abnormalities include diffuse marrow infiltration, permeative lesions, "punched-out" lesions, destructive lesions, neuropathic-like lesions, and sclerotic lesions.[3] The most characteristic of these are the permeative lesions, presenting a reticular or "lace-like" appearance. More localized areas of infiltration lead to discrete foci of bone destruction. These can be particularly disturbing if the diagnosis of sarcoidosis is unknown at the time of imaging evaluation.

Several case reports have illustrated lytic osseous destruction in vertebral sarcoidosis.[4,5] The most recent of these, published in this issue of the *Journal*, includes the appearance of destructive sarcoidosis demonstrated by magnetic resonance imaging (MRI).[5] Other reports have illustrated similar findings using computed tomography (CT).[4–7] The importance of these observations lies not only in a greater likelihood of discovery of an osseous abnormality, but also in the potential for misdiagnosis.

Radiologic imaging examinations are requested for innumerable reasons. Plain radiographs of the spine are obtained in the setting of acute trauma and chronic back pain. CT of the spine is also obtained under similar circumstances. MRI has become the imaging modality of choice for the evaluation of disc herniation, degenerative disease, and a variety of neurological abnormalities. In any instance, areas of bone destruction may be discovered incidentally during a search for the source of related or unrelated symptoms.

The differential diagnosis of lytic bone destruction, particularly if multiple foci of involvement are evident in the spine, would include diffuse metastatic disease, multiple myeloma, lymphoma, and infections such as tuberculosis. Obviously, these diseases are more ominous than sarcoidosis. They are also more prevalent than osseous sarcoidosis, so when incidental bone destruction is encountered, metastases or myeloma would be the more likely diagnostic possibilities. Thus, it is critical to combine all clinical and imaging information (including findings on dermatologic examination and on chest radiographs) to arrive at an accurate diagnosis.

If an underlying primary process is not known at the time of imaging, a final diagnosis is often made only after surgical biopsy. However, if such a process is known to be present, a correct diagnosis might more easily be reached. In such circumstances, communication between the radiologist and the referring clinician is always important and often critical to optimal patient care. If the clinician is aware of the presence of an underlying systemic disorder such as sarcoidosis, this information should be included on the requisition form for any imaging examination. If an unexpected finding is encountered by the radiologist, he or she would be able to suggest a possible connection between this finding and the known disorder that might otherwise have gone unrecognized.

The paper by Shaikh et al[5] in this issue of the *Journal* emphasizes the importance of radiologic imaging of osseous sarcoidosis and the critical role physician communication can play in accurate diagnosis. In the case that they report, a previously healthy, 29-year-old patient was evaluated for slowly progressing lower back pain. Plain radiographs of the spine showed multiple osteolytic lesions, and subsequent CT revealed additional extensive paraspinal soft tissue masses. MRI demonstrated diffuse infiltration of bone and soft tissues leading to an open biopsy. During the course of this work-up, a chest radiograph disclosed bilateral hilar adenopathy.

What might the preoperative diagnosis have been? First of all, lower back pain is a very nonspecific symptom that may be due to any number of muscular or skeletal abnormalities. The osseous destruction discovered radiographically is obviously of much concern, and the additional soft-tissue infiltration demonstrated by CT and MRI would certainly suggest a neoplastic process, perhaps metastatic disease or lymphoma in a younger patient. Hilar adenopathy would support these diagnoses but should also suggest the possibility of sarcoidosis in spite of the relative rarity of lytic destruction of the spine in this disorder. Now, the task facing the pathologist of arriving at the correct diagnosis is certainly made simpler when he is armed with all of the imaging data collected. Thus, recognition that sarcoidosis can occasionally present in this unusual manner was certainly aided by the consultative efforts of all physicians involved in this patient's care. Publication of such cases serves to enhance the reader's knowledge of disease processes and might actually lead to a correct diagnosis when a patient with similar clinical and imaging findings is encountered. It is hoped that publication of this commentary might reinforce the importance of active physician interaction, as well.

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1. Sharma OP: Sarcoidosis. *Disease-A-Month* 1990; 36:469–535.
2. Sartoris DJ, Resnick D, Resnik C, et al: Musculoskeletal manifestations of sarcoidosis. *Semin Roentgenol* 1985; 20:376–386.
3. Yaghmai 1: Radiographic, angiographic, and radionuclide manifestations of osseous sarcoidosis. *Radiographics* 1983; 3:375–396.
4. Resnik CS, Young JWR, Aisner SC, et al: Case report 594. Osseous sarcoidosis of lumbar, spine, and pelvis. *Skeletal Radiol* 1990; 19:79–81.
5. Shaikh S, Soubani AO, Rumore P, et al: Lytic osseous destruction in vertebral sarcoidosis. *NY State J Med* 1992; 9:213–214.
6. Bundens DA, Rechtine GR: Sarcoidosis of the spine. Case report and literature review. *Spine* 1986; 11:209–212.
7. Dinerstein SL, Kovarsky J: Vertebral sarcoidosis: demonstration of bone involvement by computerized axial tomography. *South Med J* 1984; 77:1060–1061.

Serious complications of laparoscopic cholecystectomy in New York State

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ABSTRACT. Serious surgical complications following laparoscopic cholecystectomy are described in a series of 17 patients who underwent the procedure in New York State in 1990 and 1991. Among the 17 cases, the most frequent complication (41.1%) was laceration of the common bile duct and/or right hepatic duct. Other serious surgical complications included perforation or laceration of the small bowel, laceration or injury of an intrahepatic bile duct, and post-operative bleeding. Some of the serious complications described here, such as laceration of the aorta and iatrogenic resection of portions of the extrahepatic biliary tree, have not been previously described. The results of this study point out the need for carefully designed retrospective and prospective studies to assess the prevalence of complications from this procedure.

(NY State J Med 1992; 92:179–181)

Laparoscopic cholecystectomy is a relatively new technique, which allows for the removal of the gall bladder through small puncture wounds in the abdominal wall. The procedure was first introduced in France and the United States and was rapidly driven to popularity by both patient preference and economic forces.[1–5] The appeal to patients consists of a very short hospital stay (1–2 days on average), the absence of a permanent sub-costal scar associated with open cholecystectomy, less pain, rapid recovery, and the conviction that the procedure is less invasive and carries fewer risks than the traditional operation.

Patient demand pushed surgeons to learn the new procedure and forced hospitals to purchase appropriate instruments in order to remain competitive. The equipment manufacturers, drawn by a potentially large market, have also popularized the procedure and encouraged surgeons to learn it by sponsoring training courses.[4] These forces created a situation in which this new technology was widely adopted before it had been fully evaluated in large teaching and research medical centers. This direction of new technology adoption represents an exception to the usual pattern of development and perfection in large academic medical centers followed by later implementation in community hospitals.[5]

The rapid adoption of this new technology occurred against a background in which there was little in the way of data gathered from carefully designed scientific studies, training courses of unequal quality, and widespread rumors of a steep learning curve, bile duct injuries, severe hemorrhage, and even deaths.[6] These issues coupled with uneven credentialling and privileging standards in hospitals have engendered both intense interest in, and much debate about, the procedure.

We present in this report a series of 17 patients who underwent laparoscopic cholecystectomy in community hospitals in New York State in 1990 and 1991 and among whom serious surgical complications occurred. The patients ranged in age from 17 to 86 years with a mean of 48 years. Of the patients, 13 were women, and four were men. The information in this study is not intended to convey impressions about prevalence rates for various complications, but rather to inform and educate physicians about the nature of some of the most serious surgical complications associated with this procedure. Prevalence data can only be generated by epidemiological studies.

This is the first of two reports. The second will be more extensive and will present New York State Medicare epidemological data.

PATIENTS AND METHODS

Island Peer Review Organization (IPRO), a New York State peer review organization since 1989, has been under contract to the Health Care Financing Administration and the New York State Department of Health to perform Medicare and Medicaid reviews, respectively.

Currently, IPRO conducts approximately 150,000 Medicare retrospective medical chart reviews annually and conducts over 100,000 Medicaid acute care, ambulatory, and alternate level of care reviews. Cases in which deficiencies are identified are referred for full review by physician specialists with opportunity for comment by the attending physician. During this process, IPRO identified quality of care issues in 17 cases of laparoscopic cholecystectomy, which were brought to its attention from these reviews.

RESULTS

The surgical complications that occurred in a subset of 17 cases with quality of care issues are presented in Table I. All patients underwent laparoscopic cholecystectomy in hospitals. The most frequent complication was laceration of either the common bile duct or the right hepatic duct. This was followed in frequency by post-operative bleeding, laceration or injury of an intra-hepatic bile duct, and perforation/laceration of the small bowel. In three of the 17 patients, the laparoscopic procedure was converted to an open one. Twelve patients were returned to surgery for laparotomy and in seven Roux-en-Y procedures performed. Two of the 17 patients died. Presented below are four cases, illustrating some of the complications observed in this series.

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TABLE I. Surgical Complications of Laparoscopic Cholecystectomy in 17 Patients with Confirmed Quality Problems

Complication	Number-Percentage
Hemoclip placed on common bile duct	1
Post-operative bleeding	2
Laceration of the common bile duct and/or laceration of the right hepatic duct	7
Laceration or injury of an intrahepatic bile duct	2
Iatrogenic resection of the common bile duct and portions of the right and left hepatic ducts	1
Post-operative leakage from the cystic duct	1
Laceration of the aorta	1
Perforation or laceration of the small bowel	2
Total	17

DESCRIPTIONS OF SELECTED CASES

Laceration of the Common Bile Duct. A 30-year-old female presented with epigastric pain radiating to the back. An abdominal sonogram demonstrated multiple echogenic foci in the gall bladder neck and fundus and thickening of the gall bladder wall. Two days after laparoscopic cholecystectomy, the patient developed abdominal pain. Hepatobiliary scintigraphy was performed and demonstrated a subcapsular and intraperitoneal biliary leak. Endoscopic retrograde cholangiopancreatography (ERCP) was performed demonstrating extravasation of contrast into the peritoneal cavity. A laparotomy was immediately performed and a lacerated common bile duct found. A Roux-en-Y was performed.

Perforation or Laceration of the Small Bowel. A 68-year-old man presented with sudden onset of right upper quadrant pain. A sonogram revealed a gall bladder prominent in size and containing multiple small calculi and debris. A diagnosis of cholecystitis, cholelithiasis, and choledocholithiasis was made. The patient's total bilirubin was 7.9 mg/dL, serum glutamic oxaloacetic transaminase was 143 U/L, and the alkaline phosphatase was 239 U/L. An ERCP was performed, revealing cholelithiasis with obstructive jaundice and no evidence of common bile duct stones. Laparoscopic cholecystectomy was performed. The next day, the patient spiked a temperature to 101°F. A large amount of thick, bilious drainage was noted near the umbilical incision. This appeared to be bowel contents. A laparotomy was performed, and a 1-cm perforation to the mid-ileum was found, caused by a suture used in closing the umbilical wound. A week later, the patient was returned to the operating room because of the breakdown of the previously repaired tear. A 60-cm length of ileum was removed during the procedure, and an ileostomy created.

Laceration of the Aorta. A 66-year-old female was admitted for right upper quadrant pain. A laparoscopic cholecystectomy was performed. When placing the trocar, the operating surgeon felt a deep descent. The surgeon converted to an open procedure and found an intraperitoneal hematoma. He proceeded to perform an open cholecystectomy which took an hour and fifteen minutes. A retroperitoneal hematoma was noticed in the area of the aortic bifurcation. It was then discovered that both the anterior and posterior walls of the aorta had been lacerated by the trocar. The surgeon tried to repair the anterior wall but was unable to repair the posterior one. A vascular surgeon was summoned and repaired the posterior aortic wall. However, the patient died.

Iatrogenic Excision of the Common Bile Duct and Portions of the Right and Left Hepatic Ducts. A 31-year-old female underwent laparoscopic cholecystectomy because of recurrent upper right quadrant pain. Post-operatively, the patient's hemoglobin fell to 7.6 mg/dL. An exploratory laparotomy was performed, and 2,000 mL of blood evacuated. The bleeding was found to be coming from a branch of the cystic artery and from the liver bed. Two loose clips were found in the hepatic bed, and the assumption was that these had slipped. The bleeding was controlled and the patient discharged five days later. Ten days later, the patient developed chills and fever. An ERCP revealed a ligated common bile duct, and a large fluid accumulation in the right upper quadrant. The patient was referred to another hospital. There, an extensive workup revealed iatrogenic excision of the extra-hepatic biliary tract during the previous laparoscopic cholecystectomy. A laparotomy was performed, confirming that the common bile duct and portions of the right and left hepatic ducts had been resected. The patient required multiple subsequent procedures and biliary tract reconstruction.

DISCUSSION

Laparoscopic cholecystectomy is a new surgical technology that has been adopted with a speed that has outpaced the ability of carefully designed prospective studies to evaluate its risks and complications. A number of retrospective studies have been published showing complication rates of from 0% to 9%.[2–3,7–9] Overall, this favorably compares with complication rates of 6.0% observed with open cholecystectomy.[11] The mortality rates reported in many published retrospective series have been 0%. A prospective analysis of 1,518 laparoscopic cholecystectomies published by the Southern Surgeons Club for example reported complication rates of 5.1% and a mortality rate of 0.07%. In this large series, four bowel injuries occurred and seven injuries to the common bile duct.[12]

Excellent results and low morbidity and mortality rates reported in published series, however, must be viewed in light of the fact that they often come out of large academic medical centers and are authored by experienced laparoscopists. Careful patient selection also plays a role in reducing the risk and severity of complications in such series. As helpful as such published series are, they do not always provide insights into the frequency and character of complications that arise when the procedure is first undertaken by surgeons who lack extensive experience with it.

Some of the serious surgical complications described in this paper have not been reported, to our knowledge, in previously published series. They reflect the types of complications that may occur when the procedure is undertaken by those who are insufficiently trained and/or lacking in experience. They also support the fact that the anecdotal stories of serious complications in many hospitals are not mere rumor. Although serious surgical complications of this type may reflect the fact that many surgeons are in the early part of the learning curve, they may also indicate that some surgeons are not now, and will not ever be, adept at this procedure.

More and more surgical procedures are being performed through laparoscopy (eg, endoscopic-arthroscopic carpal tunnel release, appendectomy, splenectomy, hysterectomy, herniotomy, hemicolectomy). Laparoscopy requires different types of skills than those needed for open cholecystectomy. The use of a monitor, operating in a two-dimensional field, and dexterity in the use of instruments through tubes may be difficult skills for some surgeons to acquire and master. These and other issues surrounding this procedure can be addressed in part by standardized training programs that incorporate adequate contemporaneous supervision, by appropriate credentialing and privileging standards, and by careful selection of patients for this procedure. Specialty societies can play an important role in setting standards for training programs. However, the hospital is ultimately responsible for appropriate credentialling and privileging of its staff.

Acknowledgments. The authors thank Harvey R. Bernard, MD, Professor of Surgery, Albany Medical College, and Surgical Advisor, New York State Department of Health, for his help and guidance in the preparation of this paper.

REFERENCES

1. Perissat J, Collet DR, Belliard R: Gallstones: laparoscopic treatment, intracorporeal lithotripsy followed by cholecystostomy or cholecystectomy—a personal technique. *Endoscopy* 1989; 21:(Suppl) 373–374.
2. Dubois F, Icard P, Berthelot G, et al: Coelioscopic cholecystectomy: Preliminary report of 36 cases. *Ann Surg* 1990; 211:60–62.
3. Reddick EJ, Olsen DO: Laparoscopic laser cholecystectomy: A comparison with mini-lap cholecystectomy. *Surg Endosc* 1989; 3:131–133.
4. Wolfe, BM, Gardiner B, Frey CF: Laparoscopic cholecystectomy: A remarkable development. *JAMA* 1991; 265:1573–1574.
5. Stoney WS: Laparoscopic cholecystectomy: Problems of rapid growth. *S Med J* 1991; 681–683.
6. Dent TL: Training, credentialling, and granting of clinical privileges for laparoscopic general surgery. *Am J Surg* 1991; 161:399–403.
7. Zucker KA, Bailey RW, Gadacz TR, et al: Laparoscopic guided cholecystectomy. *Am J Surg* 1991; 161:36–44.
8. Peters JH, Ellison EC, Innes JT, et al: Safety and efficacy of laparoscopic cholecystectomy. *Ann Surg* 1991; 213:3–12.
9. Cuschieri A, Dubois F, Mouiel J, et al: The European experience with laparoscopic cholecystectomy. *Am J Surg* 1991; 161:385–387.
10. Wilson P, Leese T, Morgan WP, et al: Elective laparoscopic cholecystectomy for "all comers." *Lancet* 1991; 338:795–797.
11. Pickleman J, Gonzalez RP: The improving results of cholecystectomy. *Arch Surg* 1991;121:930–934.
12. The Southern Surgeons Club: A prospective analysis of 1518 laparoscopic cholecystectomies. *N Engl J Med* 1991; 324:1073–1078.

Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals

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ABSTRACT. Under New York State's unique do-not-resuscitate law, patients admitted to a hospital are assumed to consent to cardiopulmonary resuscitation unless a do-not-resuscitate order has been written in accordance with specific stipulations. This study was undertaken to determine if the law would significantly change the use of cardiopulmonary resuscitation.

The charts of 952 patients who died on the medical services of Bellevue, New York University, and New York Veterans hospitals during the 12-month periods before and after the law went into effect were reviewed. The proportion of patients undergoing cardiopulmonary resuscitation was determined. To take into account the effects of potential confounders (ie, hospital, sex, race or ethnic group, age, diagnosis), a multiple logistic regression analysis was performed.

The relative risk for cardiopulmonary resuscitation was found to be 0.81, (95% confidence interval: 0.59–1.10) after the law as compared with before, which was not statistically significant. Although patients with chronic or potentially reversible illness were much more likely than those with hopeless illness to undergo cardiopulmonary resuscitation (relative risk of 7.08 [3.23–15.54] and 12.89 [8.71–19.07], respectively), the data failed to show that the law had an impact on the use of cardiopulmonary resuscitation in any of the disease categories. Total use of formal do-not-resuscitate orders increased from 32.7% to 83.9% ($p < .0001$), although at Bellevue, where documentation was high before the law, no further increase occurred.

We conclude that since the do-not-resuscitate law, more formal do-not-resuscitate orders are being written; however, there was no significant change in the actual use of do-not-resuscitate or cardiopulmonary resuscitation in our review of nearly 1,000 deaths. Changes might still be detected with a larger study sample.

(NY State J Med 1992; 92:181–185)

In July 1987, New York State enacted a law governing physicians' orders not to resuscitate patients who undergo cardiopulmonary arrest.[1] The law went into effect April 1, 1988. Although the Joint Commission on Accreditation of Health Care Organizations required that all health care facilities have a do-not-resuscitate (DNR) policy in effect as of January 1988,[2] New York is the only state to have enacted a law governing the use of DNR orders with specific stipulations that must be followed. Under the law, it is legal for the attending physician to issue an order not to resuscitate if these stipulations have been followed; however, in the absence of such an order, all persons admitted to a hospital are presumed to consent to CPR. The belief had been expressed by some of our colleagues that the law was leading to a marked increase in the number of resuscitations, many of which might be considered inappropriate because of their futility. This concern has been expressed elsewhere.[3] It is important that such assumptions be subjected to careful study because of their implications and because of the possibility that other states may follow New York's direction.[4]

In a recent retrospective analysis of 245 deaths, Kamer et al[5] demonstrated that New York's legislation had little if any impact on the proportion of patients undergoing CPR in a community tertiary referral hospital. However, that study was small and was conducted in one type of hospital, which might not be representative of other institutions. In addition, the study did not control for confounding vari-

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ables that may have had an impact on the results. We conducted a retrospective analysis of 952 deaths in three demographically distinct teaching hospitals to shed further light on this issue.

TABLE I. Diagnostic Categories
Number (percent)

	Bellevue	UH	VA
Group 1			
Advanced cancer	144	97	20
AIDS	36	246	124
Chronic vegetative[*]	1	2	2
Total	181 (77.4)	345 (69.6)	146 (65.8)
Group 2			
Metabolic[†]	1	2	4
Neurologic[‡]	3	2	6
Respiratory	0	6	5
Total	4 (1.7)	10 (2.0)	15 (6.8)
Group 3			
Cardiac	9	74	5
Collagen vascular	0	13	1
Delirium tremens	0	0	1
Gastrointestinal bleed	2	4	1
Hepatic encephalopathy	6	8	6
Infection	28	31	42
Iatrogenic	0	0	1
Unexpected arrest	1	2	4
Other[§]	3	9	0
Total	49 (20.9)	141 (28.4)	61 (27.5)
All patients	234 (100)	496 (100)	222 (100)

[*] Profound global neurologic impairments of various etiologies.
[†] Almost all chronic renal failure; no cases of diabetic coma or ketoacidosis.
[‡] Neurologic impairments less severe than in group 1 (e.g., hemiparesis).
[§] Includes one case each of hypothermia, anaphylaxis, volvulus; two of hip fracture; four of ruptured aneurysm.

PATIENTS AND METHODS

Charts were reviewed of deaths that occurred on the adult in-patient medical service of Bellevue, New York University, and New York Veterans (VA) Hospitals for one year before implementation of the DNR legislation and for the year following. All three institutions are teaching hospitals of New York University School of Medicine. Bellevue is a 1,242-bed public hospital, which is operated by the New York City Health and Hospitals Corporation. Approximately 320 beds are on the medical service. New York VA Hospital has 1,030 authorized beds of which 151 are on the medical service. The University Hospital (UH; renamed Tisch Hospital in 1989) is a 726-bed private, nonprofit hospital whose attending physicians are on the faculty of the medical school; there are approximately 465 beds on the medical service. Medical house staff are recruited for two separate training programs within the medical school, but all spend at least a portion of their time at each of the three hospitals. During the 24-month period of analysis, 1,680 deaths occurred, for which 1,088 charts were available for analysis.

A patient was considered to have DNR status if there was a progress note indicating that a DNR discussion had taken place with the patient or a surrogate and that a decision not to resuscitate had been made, or if a formal order was written, or both. Eighty-eight patients who had undergone CPR on one or more occasion before being designated DNR, and 48 who had neither been designated DNR nor undergone CPR were excluded from the analysis. Among the latter group, chart notes in most cases indicated that they had been found dead in bed.

The proportion of CPR to deaths was determined and is expressed as a percentage. The proportion occurring before the law went into effect ("pre") was compared with that after ("post"), and this comparison was subjected to statistical analysis. Since we analyzed only those individuals either undergoing CPR or designated DNR, CPR, and DNR are complementary. This analysis was considered preliminary, pending statistical analysis that controlled for confounding variables.

To rule out a gradual change that might have occurred independently of the legislative mandate, we scrutinized the data on a month-by-month basis, and because there was some inconsistency in the proportion of charts available in a given month and because only a few deaths occurred in certain months, we grouped months into quartiles of the year for scrutiny: April–June, July–September, October–December, and January–March, for the 12-month periods before and after the law went into effect.

Other variables that were measured included the hospital where the death occurred, sex of the patient, race or ethnic group, age, and diagnosis. Age was analyzed by decade of life. "Diagnosis" refers to the underlying disease that lead to death, if this differed from the immediate cause of death. Diagnoses were grouped according to the likelihood that they would prompt a DNR order: category 1 being the most likely, category 3 being the least likely, and category 2 intermediate (Table I). Diagnostic groupings were made before the statistical analysis and without the authors' knowledge of the patients' DNR status.

In preliminary analyses, the proportions of patients undergoing CPR in the pre-period and the post-period were compared using Fisher's exact test (two-tailed). To take into account the effects of potential confounders of the relationship between CPR and pre- versus post-period (ie, hospital, sex, race, age, diagnosis), a multiple logistic model was fit to the data.[6] We obtained an adjusted relative risk of CPR in the post-period compared with the pre-period. "Relative risk" is defined here as how many times more likely it is that a patient dying during the post-period would undergo CPR than one dying during the pre-period.

We also calculated the proportion of individuals designated DNR who received a formal DNR order. This is expressed as a percentage, and pre- versus post-period data were compared using Fisher's exact test.

RESULTS

Potentially confounding variables are summarized in Table II. Viewing all three hospitals together, and each hospital individually, there was no significant change in the proportion of patients undergoing CPR, and the analysis by quartiles of the year failed to reveal any consistent or lasting trend, either toward increase or decrease in CPR in the two-year period analyzed (Table III).

Multiple logistic regression analysis revealed a relative risk for CPR of 0.81 (95% confidence interval: 0.59–1.10) after the law as compared with before. The relative risk was adjusted for age, sex, race or ethnic group, diagnosis, and hospital where the death occurred. Whereas this change was not statistically significant, the power was low; with a larger sample size, we might have detected a significant difference.

Implementation of the law was significantly associated with an increase in documentation. Before enactment, DNR status was more often indicated as a progress note without a formal order on the order sheet. The increase in the use of formal orders was significant. There was a marked variability in this pattern among the three hospitals, with little change occurring after the law at Bellevue, a dramatic change at the UH, and an intermediate but significant change at the VA (Table IV).

Disease category was predictive of whether someone might undergo CPR, both before and after the law went into effect.

TABLE II. Demographic Variables
Number (percent)

	Bellevue	UH	VA
Total deaths analyzed	234	496	222
Sex			
Male	185 (79.1)	281 (56.7)	222 (100)
Female	49 (20.9)	215 (43.3)	0
Race/ethnic group			
White	72 (30.8)	456 (91.9)	90 (40.5)
Black	77 (32.9)	13 (2.6)	110 (49.5)
Hispanic	73 (31.2)	14 (2.8)	21 (9.5)
Asian	12 (5.1)	13 (2.6)	1 (0.5)
Age (years)			
20–29	25 (10.7)	14 (2.8)	1 (0.5)
30–39	75 (32.1)	51 (10.3)	14 (6.3)
40–49	37 (15.8)	62 (12.5)	16 (7.2)
50–59	28 (12.0)	49 (9.9)	34 (15.3)
60–69	28 (12.0)	105 (21.2)	82 (36.9)
70–79	19 (8.1)	146 (29.4)	47 (21.2)
80–89	15 (6.4)	63 (12.7)	20 (9.0)
90+	7 (3.0)	6 (1.2)	8 (3.6)

Overall, patients in categories 2 and 3 were far more likely to undergo CPR than those in category 1 (relative risk of 7.31 [3.23–16.55] and 13.59 [9.06–20.40]), respectively. However, within the limits of our sample size, there was insufficient evidence that the law had any impact on the proportion of patients in any of the three disease categories who underwent CPR.

Discussion

This study has failed to demonstrate that the New York State law has been associated with a significant change in the proportion of patients undergoing CPR before death. These data were adjusted for epidemiologic variables that were present in the three hospital populations. That such variables could be of potential importance as confounders is demonstrated by the fact that the diagnostic category was highly predictive of the use of CPR. We analyzed age by decade of life because specific disease categories that might prompt DNR discussions tend to occur in particular age groups, and it is noteworthy that the age distribution of deaths varied among the three hospitals. The distribution of sex and race or ethnic group varied as well (Table II). Type of medical insurance had been recorded for each patient but this potential confounder was not included in the analysis because the VA was distinct from the other two hospitals in this regard. Specifically, we could not ascertain from the chart which VA patients retained insurance outside of veteran's benefits or whether outside agencies had been billed. These data, nonetheless, emphasize the importance of type of hospital as a potential confounder (Table V).

The quartile analysis failed to reveal any gradual change that might have occurred during the study period. The data have not captured a change that might have occurred before the period of study as a result of increasing awareness of DNR or attempts to implement local DNR policies. However, a study in New York State conducted two years before enactment of the law suggested that a DNR policy alone would not have an immediate impact on the use of CPR.[7] In the three study hospitals presented here, policies were instituted and implemented at different times, but these differences are not reflected in differences in the use of CPR or DNR. In all three hospitals, it is likely that implementation of the policy and education of the staff would be an ongoing process and would not occur quickly, so it is possible that changes continue to occur even now. However, a significant change in documentation took place in April 1989 (Table IV), suggesting that if any impact on actual use of CPR or DNR had occurred, that would have been the most likely time.

There was variability in the proportion of charts available at the three hospitals with 78%, 69%, and 45% of patients who died having charts available for analysis at the UH, VA, and Bellevue hospitals, respectively. Factors at the institutions that were responsible for this included removal of charts for a construction project at Bellevue and for microfilming at UH, and misplaced charts, most often at the VA. It was our strong impression that these logistic peculiarities rather than epidemiologic factors accounted for the variability of chart retrieval, although we have no way of ruling out the latter. The degree of availability at a given hospital was a consistent phenomenon for both the "pre" and the "post" charts.

We undertook this study to evaluate the impression on the part of some of our colleagues that the DNR law was leading to an increase in the number of medically inappropriate resuscitations. However, a careful analysis fails to

TABLE III. Effects of the Law on Use of CPR and on DNR Status
Number (percent)

	Bellevue	UH	VA	All Three Hospitals
Before the law				
April–June	1/7[*] (14.3)	31/54 (57.4)	6/28 (21.4)	38/89 (42.7)
July–Sept	12/45 (26.7)	13/34 (38.2)	16/34 (47.1)	41/113 (36.3)
Oct–Dec	26/50 (52.0)	15/44 (34.1)	14/32 (48.3)	55/126 (43.7)
Jan–Mar	14/37 (37.8)	29/72 (40.3)	7/22 (31.8)	50/131 (38.2)
Total	53/139 (38.1)	88/204 (43.1)	43/116 (37.1)	184/459 (40.1)
After the law				
April–June	7/25 (28.0)	29/65 (44.6)	9/26 (34.6)	45/116 (38.8)
July–Sept	6/19 (31.6)	27/74 (36.5)	15/35 (42.9)	48/128 (37.5)
Oct-Dec	9/21 (42.9)	24/80 (30.0)	6/25 (24.0)	38/126 (30.2)
Jan–Mar	10/30 (33.3)	27/70 (38.6)	7/23 (30.4)	45/123 (36.5)
Total	32/95 (33.7)	107/289 (37.0)	37/109 (33.9)	176/493 (35.7)
p[†]	0.58	0.19	0.68	0.18

[*] Patients undergoing CPR/all deaths.
[†] By Fisher's exact test, comparing before versus after the law in each column.

TABLE IV. Formal DNR Orders Written
Number (percent)

	Bellevue	UH	VA	All Three Hospitals
Before the law				
April–June	5/6[*] (83.3)	2/23 (8.7)	0/22 (0)	7/51 (13.7)
July–Sept	25/33 (75.8)	6/21 (28.6)	0/18 (0)	31/72 (43.1)
Oct–Dec	20/24 (83.3)	0/29 (0)	4/18 (22.0)	24/71 (33.8)
Jan–Mar	22/23 (95.7)	3/43 (7.0)	3/15 (20.0)	28/81 (34.6)
Total	72/86 (83.7)	11/116 (9.5)	7/73 (9.6)	90/275 (32.7)
After the law				
April–June	16/18 (88.9)	36/36 (100)	5/17 (29.4)	57/71 (80.3)
July–Sept	10/13 (76.9)	47/47 (100)	3/20 (15.0)	60/80 (75.0)
Oct-Dec	12/12 (100)	56/56 (100)	13/19 (68.4)	81/87 (93.1)
Jan–Mar	18/20 (90)	43/43 (100)	7/16 (43.8)	68/79 (86.1)
Total	56/63 (88.9)	182/182 (100)	28/72 (38.9)	266/317 (83.9)
p[†]	0.48	<.0001	<.0001	<.0001

[*] Patients with formal DNR orders/all DNR patients.
[†] By Fisher's exact test, comparing before versus after the law in each column.

confirm this hypothesis. Although patients in certain disease categories were more likely to undergo CPR than were others, there was no evidence that the law altered the proportion undergoing CPR in any of the three diagnostic groupings used in this study. It is not possible to know if there were an increase in the use of a so-called "slow code," in which a delay occurs before the staff is paged or personnel respond slowly or perform maneuvers unaggressively. In such cases, chart notes presumably would have indicated that such patients had undergone CPR.

One might also argue that, by being restrictive or cumbersome, the DNR law in fact prevented an increase in the use of DNR that might have occurred in its absence. However, our study reveals no trend in this direction during the year preceding the law. Conversely, the law could have facilitated the use of DNR because it provides immunity to health providers who carry out DNR orders in good faith.[1]

Because we analyzed only patients who died, we do not know if the law affected the writing of DNR orders for patients who survived and were later discharged from the hospital. However, we have no reason to believe that the law would have affected the use of DNR differently in such patients in those patients who actually died, in our study of deaths, the use of CPR and DNR did not appear to change regardless of disease severity. We also do not know if the law prevented the expeditious execution of a DNR order for a patient who then went on to be unwillingly resuscitated. Our study would have missed few such patients, since the vast majority of medical patients who undergo CPR in the hospital do not survive to discharge.[8] Only 8% of the original 1088 patients in this study underwent CPR before the execution of a DNR order, and this proportion was essentially the same before and after the law went into effect (Table VI). Further study involving large numbers of patients would be required to measure directly the impact of the law on patients who survive to discharge.

TABLE V. Type of Medical Insurance
Number (percent)

	Bellevue	UH	VA
Insurance			
Medicare	39 (16.7)	263 (53.1)	*
Medicaid	99 (42.3)	15 (3.0)	*
Private	6 (2.6)	218 (44.0)	*
Other	0	0	222 (100)
None	90 (38.5)	0	0
Total	234 (100)	496 (100)	222 (100)

[*] Does not apply (see text).

The DNR law was associated with a change in documentation, with an increase in formal DNR orders being written for patients who had been designated DNR. This change was confined to two of the three hospitals—at Bellevue, a high rate of documentation already existed when the law went into effect. At the VA, documentation increased significantly but remained under 40% during the time of this study. These differences could have been due to local differences in staff education, speed of policy implementation, and the degree of involvement by attending staff in primary care. The long-standing high prevalence of the acquired immunodeficiency syndrome (AIDS) at Bellevue may have created a climate in which house staff felt more comfortable about initiating DNR discussions; 57% of deaths on the medical service at Bellevue during the study period were due to AIDS, compared with 20.6% for the UH and 16.7% at the VA hospitals.

We are unable to assess whether the increase in documentation is a good or a bad outcome. Presumably, a formal DNR order would increase the ability of personnel to react appropriately when a cardiopulmonary arrest occurs. However, there is no evidence that this is an important effect because the degree of increased documentation differed vastly by hospital, but the effect on use of CPR was virtually identical at all three hospitals. Potential negative effects might be the increase in time and cost of meeting the requirements for documentation and the resulting institutional review process.

This study and the smaller study of Kamer et al[5] failed to demonstrate an impact of the New York State law on the actual use of CPR or DNR. These studies do not completely rule out the possibility that a significant change

TABLE VI. Total Number of Patients Undergoing CPR or Designated DNR

	Before the Law	After the Law
Only DNR	275	317
Only CPR	184	176
Both	41	47
Neither	24	24
Total analyzed	524	564

occurred. An even larger sample size than this study would be required to rule out an effect on the numbers; however, this would not resolve the question of whether the law is good or bad. This study does not tell us, for example, whether DNR discussions entered into under pressure of a legal or regulatory mandate are more sensible than they are inhumane. The law clearly allows that if a DNR discussion with a competent patient would cause "severe and immediate injury,"[1] the decision can be made by a surrogate without the patient's participation. However, the "severe and immediate injury" standard is very strict, requiring that two physicians make the determination that a consequence such as a heart attack or suicide would result from such discussions. It seems unreasonable that only such extreme situations should permit the avoidance of such a discussion, since the topic is best approached gradually and with great sensitivity. Regulatory mandates superimpose an additional urgency to sign the DNR order quickly in certain very ill patients, to avoid CPR that would be technically mandated although medically inappropriate. Although the law allows CPR to be withheld at the discretion of the physician in cases of "medical futility," this has been defined as the inability to restore "cardiac and respiratory function," or the likelihood that "the patient will experience repeated arrest in a short time period before death."[1] This has led to a New York State Department of Health regulation requiring that, in such cases, a second physician, appointed by the hospital, must first review, concur with, and then document the determination of medical futility.[9] Although such requirements are almost certain to prevent secret DNR orders, a stated purpose of the law,[10] they are cumbersome and potentially harmful.

When death is imminent and inevitable, a DNR discussion, in effect, makes it appear as if there is a choice in the matter. This can pose an enormous psychological burden on a dying patient. The burden placed on surrogates may also be enormous if they are made to feel that they are in the position of determining whether their loved one will live or die. There should be further in-depth analysis of the psychological impact of DNR mandates on patients and their families and the financial impact of administrative costs resulting from this law.

Acknowledgments. The authors thank Dr Norton Spritz of the New York Veteran Hospital and Dr Arthur Zitrin of Tisch Hospital for their expert advice and assistance.

REFERENCES

1. *Laws of 1987* 818C NY Pub Heal Law Art 29-B §2960-78.
2. Joint Commission on Accreditation of Health Care Organizations. Accreditation Manual for Hospitals 1989: 82.
3. Rules on reviving the dying bring undue suffering, doctors contend. *NY Times* October 4, 1990, pp A1, B20.
4. 1990 Tenn Pub Acts 1082C.
5. Kamer RS, Dieck EM, McClung JA, et al: Effect of New York State's do-not-resuscitate legislation on in-hospital cardiopulmonary resuscitation practice. *Am J Med* 1990; 88:108–111.
6. Breslow NE, Day NE: The analysis of case-control studies. Lyon; International Agency for Research on Cancer, 1980. *IARC Scientific Publications No. 32. Statistical Methods in Cancer Research; vol I.*
7. Quill TE, Stankaitis JA, Krause CR: The effect of a community hospital resuscitation policy in elderly patients. *NY State J Med* 1986; 86:622–25.
8. Blackhall LJ: Must we always use CPR? *N Engl J Med* 1987; 317:1281–1284.
9. *Codes, rules, and regulations of the State of New York* 10C §405.43.
10. The New York State Task Force on Life and the Law. Do not resuscitate orders. The proposed legislation and report of the New York State Task Force on Life and the Law, 2nd ed; 1988:19.

Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987

ALJE VENNEMA, MD

ABSTRACT. The Bureau of Tuberculosis of the New York City Department of Health has carried out investigations of contacts to active cases of tuberculosis for many years. In addition, the Bureau has undertaken tuberculosis screening, with tuberculin testing and by radiography where indicated, of persons for employment purposes, school attendance, and for various other reasons. In 1969, the Bureau began providing isoniazid chemoprophylaxis; since then, more than 150,000 persons have been given isoniazid. Several isoniazid chemoprophylaxis effectiveness trials have been carried out since 1955.[1–3] If the optimum results of chemoprophylaxis are achieved, the frequency of developing tuberculosis in one's lifetime would be reduced by 50%–70% by the time treatment is completed, according to several studies.[4] On the basis of the beneficial results of these trials, the Bureau of Tuberculosis introduced the use of isoniazid chemoprophylaxis. This study was undertaken to assess the acceptability of isoniazid chemoprophylaxis and the success of contact investigation under both regular clinical conditions and uncontrolled conditions.

(NY State J Med 1992; 92:185–188)

Identified close contacts of persons with tuberculosis were referred to Department of Health clinics for examination. These contacts were tested with five tuberculin units of purified protein derivative (PPD); an induration of 5 mm or more was considered positive. Those persons with positive results were offered isoniazid for a period of 12 months. Isoniazid was also given to tuberculin negative contacts (primary chemoprophylaxis) for a period of three months to prevent infection, and the isoniazid was continued in those who converted after a second tuberculin test, regardless of their age, to prevent development of disease. In addition, isoniazid was given to persons under the age of 35 who reacted to tuberculin with a 10 mm or more induration ("reactors" or persons who are not contacts to a case but received a PPD test for reasons such as employment and had a positive result) and to persons with fibrocalcific lesions on chest films for a period of 12 months. Reactors

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TABLE I. Tuberculosis Contacts and Their Follow-up (%), New York City, 1979–1987

	1979	1980	1981	1982	1983	1984	1985	1986	1987	Total
Contacts identified	1,679	2,071	2,827	3,164	3,310	3,152	2,805	4,161	2,827	25,996
Examined	1,374 (82)	1,854 (90)	2,612 (92)	2,915 (92)	2,975 (90)	2,830 (90)	2,405 (86)	3,573 (86)	2,374 (84)	22,912 (88)
Not examined	305 (18)	217 (11)	215 (7)	249 (8)	335 (10)	322 (10)	400 (14)	589 (14)	453 (16)	3,085 (12)
Of those examined										
TB cases	23 (2)	47 (3)	47 (2)	53 (2)	78 (3)	142 (15)	107 (4)	75 (2)	79 (3)	651 (3)
Infected	428 (31)	578 (31)	812 (31)	929 (32)	826 (28)	818 (30)	677 (28)	977 (27)	641 (27)	6,686 (29)
Not infected	923 (67)	1,229 (66)	1,753 (67)	1,896 (65)	2,071 (70)	1,870 (66)	1,621 (67)	2,426 (68)	1,630 (69)	15,419 (67)
Of those infected										
Treated	371 (87)	425 (73)	652 (80)	587 (63)	524 (63)	513	513 (63)	579 (76)	456 (71)	4,620 (70)
Untreated	57 (13)	153 (77)	160 (20)	342 (37)	302 (37)	305 (37)	164 (24)	398 (41)	185 (30)	2,066 (29)
Of those not infected										
Ever treated	106 (12)	144 (12)	150 (9)	252 (13)	238 (11)	204 (11)	315 (19)	265 (11)	190 (22)	1,864 (12)
Treated and seroconverted	165 (18)	196 (16)	675 (38)	233 (12)	306 (15)	265 (14)	228 (14)	354 (14)	265 (16)	2,687 (17)
Untreated	652 (71)	889 (72)	928 (52)	1,411 (74)	1,527 (73)	1,401 (74)	1,078 (66)	1,807 (75)	1,175 (72)	10,868 (70)

with other medical conditions such as diabetes, substance abuse, silicosis, or gastrectomy, and patients who were receiving immunosuppressants or who had other medical conditions were also offered isoniazid. All interview forms in the Department of Health for 1979–1987 that list all close contacts to a newly reported case of active tuberculosis were reviewed. Forms that did not list any close contacts were excluded.

METHODS

The Bureau kept records on all patients tested and placed on therapy in its clinics. Municipal and voluntary hospitals kept their own records. The Bureau, through a review of procedures, became aware in 1978 that the records were unsatisfactory for proper assessment of compliance. It was impossible to know the numbers of patients entering, dropping out, or completing therapy. Hence, in 1978, a new method was devised by which the Department of Health Clinics would fill out a "start and stop form" on all new patients. Municipal and voluntary hospitals were encouraged to use the same forms. Thus, only isoniazid prophylaxis data on close contacts, reactors, and those with fibrocalcific lesions placed on isoniazid by the Department of Health Clinics for the years 1979–1987 were analyzed. For 1979–1987, 1,066 from municipal hospitals and voluntary hospitals were examined for completion but were not included in this study as few charts contained satisfactory information. The "start and stop forms" were reviewed with reference to initiation and completion of preventive therapy; occurrence of side effects; follow-up; and numbers of patients who defaulted, died, moved, developed tuberculosis, or were discharged from therapy. Treatment was initiated by a physician in the Department of Health Clinics. Thereafter, clinic nurses followed-up patients on a monthly basis. At the time of each visit, patients were questioned as to drug ingestion, tolerance of the drug, and its toxic effects. Liver studies were only done when clinically indicated. Completion of treatment percentages were calculated at 3, 6, 9, and 12 months.

RESULTS

Over a period of nine years (1979–1987), 25,996 close contacts were identified (Table I). On average, each case named 3.7 contacts in this cohort. Of these contacts, 22,912 (87.9%) were examined. Of these, 651 (28%) were considered cases of tuberculosis by their respective physicians; however, only 350 of these 651 met the US Centers for Disease Control case definition[5] (ie, either positive culture or positive biopsy or a combination of clinical findings, negative culture, positive PPD, positive chest film, and improvement with treatment) (Fig 1). Twenty-nine percent (6,686) of the contacts examined were infected, and 15,419 (67%) were not infected. Of the infected contacts, 4,620 (70%) were placed on isoniazid. The remainder refused treatment. Of the "not infected" contacts, 2,687 (17.6%) were placed on primary chemoprophylaxis and, on subsequent retesting had sero-converted; 1,864 (12%) received primary prophylaxis and remained negative to PPD on retesting. For the latter group, isoniazid was stopped after three months. Seventy percent (10,868) of all not infected, examined contacts did not receive primary prophylaxis. In addition, 2,066 (30%) infected contacts did not receive treatment. The different categories fluctuated from year to year. Contact examination peaked in the early 1980s, thereafter decreasing slightly. Table II indicates the patients who started and completed therapy by risk category and how many defaulted. Of those in the non-infected category, 36.7% completed the advised three months of treatment. About 60% of the other groups (Figs 2,3) completed three months of treatment. The six-month completion rate for infected contacts, those who seroconverted, and reactors ranged from 48.0% to 49.8% (Table II). Fifty-four percent of patients with fibrocalcific changes completed six months of therapy. Thereafter, the withdrawal rate was about 4% per month. The overall 12-month completion rate was 34.4%.

When looking at the completion rate by age (Table III), it was noted that no statistically significant difference existed

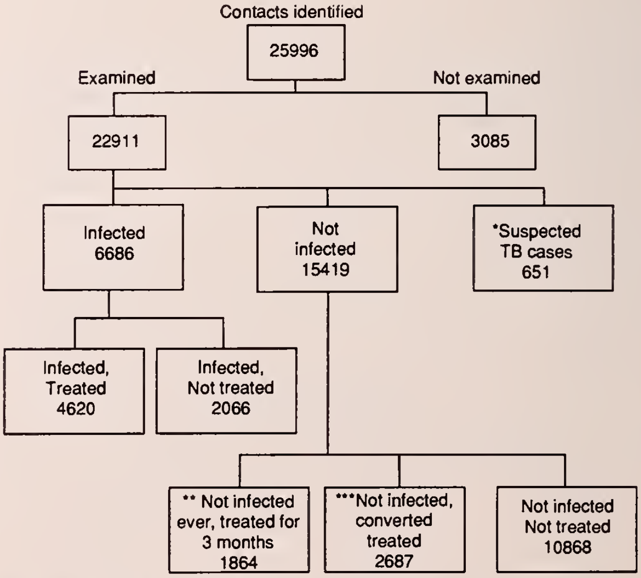

FIGURE 1. Tuberculosis contacts, identified, and treated, 1979–1987.

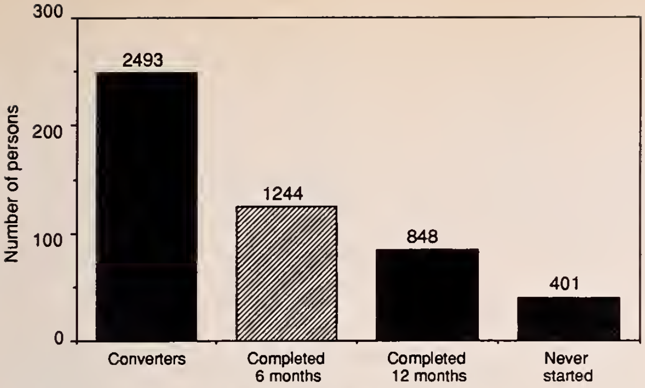

FIGURE 2. Converters placed on treatment, 1979–1987.

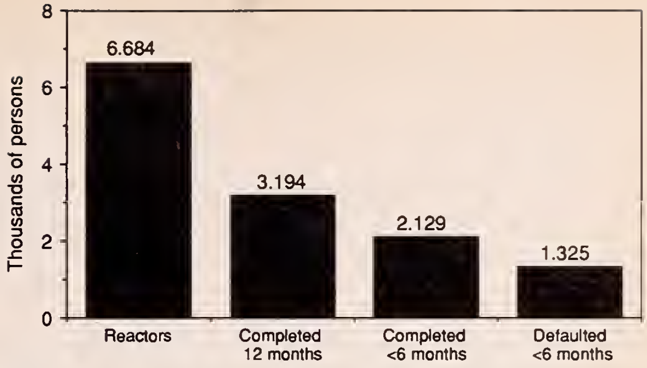

FIGURE 3. Tuberculin reactors placed on treatment, 1987–1987.

between the age groups 0–14 year (46%) and 15–19 year (39%); however, a significant difference did exist between the age groups 15–19 year (39%), 20–34 year (28%), and 35+ year (37%). During treatment, 1.5% of patients had a drug reaction and had medication discontinued, no cases of hepatitis occurred, and there were no new cases of tuberculosis; three patients died from other causes unrelated to isoniazid.

DISCUSSION

That only 34.4% of contacts completed 12 months of treatment is a dismal result. When shortening the time period of prophylaxis to six months, only 48% of contacts completed treatment. Before March 1986, preventive therapy had been prescribed for a period of 12 months. Thereafter a joint statement of the American Thoracic Society and the Centers for Disease Control advocated a change to preventive therapy with isoniazid given for 6-to-12 months.[6] When considering a treatment period of 6-to-9 months, the overall completion rate in this study was only 38.6%. In fact, more than half of the contacts discovered did not show up for examination, and the great majority decided not to complete treatment.

The guidelines, it seems, are only effective under optimum conditions, and they should therefore be modified to take into account patients' needs, public health needs, and resources. Patients may not want to take medication, although they may oblige at first. If patients do not wish to take medication, they should not be placed on medication, and this applies particularly to reactors. Although contacts are at risk of developing tuberculosis, giving a drug to people who are not sick and not motivated, for a period of 6-to-12 months, is impractical. In two recent articles attention has been focused on decision analyses with reference to cost effectiveness of chemoprophylaxis. Tsevat et al[7] concluded that the net benefit from isoniazid is small. Cost effectiveness has been addressed by Moulding[8] and Snider.[9] These latter investigators showed that chemoprophylaxis is very costly, and that a six month course is more cost effective than a 12-month course. The following question arises: Are the expenses incurred by this type of program warranted and will this approach contribute to the elimination of tuberculosis, or is it better to focus only on close contacts under the age of 15? Although efficacy studies have shown that isoniazid chemoprophylaxis is advisable, the major problem is poor compliance.[10] It is doubtful that compliance can be imposed, as compliance is a function of attitude and behavior on the part of the patient.[11,12] Other reasons for non-compliance are moving away, death, and side effects from drugs. Age, sex, level of education and economic status do not affect compliance.[10,11,12] A large number of contacts did not even come for examination; many of the infected contacts defaulted after starting treatment. A study carried out by the International Union Against Tuberculosis showed that 10.7 cases of tuberculosis could be prevented in 1,000 persons with fibrocalcific changes after a 52-week regimen of isoniazid and a five-year follow-up.[13] (Compliance in that study was 100%.)

In the present study, 568 patients had fibrocalcific changes and 48.7% completed treatment; hence, approximately 2.5 cases of tuberculosis could be prevented. This group, with a completion rate of almost 50%, should be persuaded to take isoniazid. The treatment of 2,108 contacts and 2,493 converters with isoniazid, considering a lifetime risk of tuberculosis of 10%, an efficacy rate of 70%, and a six-month completion rate of 49% would only have prevented approximately 15 cases of tuberculosis. Under the present socioeconomic conditions in New York City, it is questionable that compliance can be improved.

Recommendations. Who then should be placed on isoniazid prophylaxis? Close contacts who have been recently infected, whose tuberculin test is strongly positive, and who have not previously received bacillus Calmette-Guerin vaccine are recommended to take isoniazid for a

TABLE II. Patients Compared by Completion of Treatment, 1979–1987

Length of Treatment (%)	Non-infected Contacts	Infected Contacts	Converters	Reactors	Fibrocalcific Lesions	Total
Started	1,106	2,108	2,493	6,648	568	12,923
3 months	406 (37)	1,191 (56)	1,442 (58)	3,752 (56)	408 (72)	7,199 (56)
6 months	NA* (NA)	1,028 (48.7)	1,244 (49.8)	3,194 (48.0)	308 (54.2)	5,774 (48.8)
9 months	NA (NA)	830 (39.3)	986 (39.5)	2,471 (37.1)	279 (49.1)	4,566 (38.6)
12 months	NA (NA)	790 (37.4)	848 (34.0)	2,129 (32.0)	277 (48.7)	4,044 (34.2)
Total completed	406 (36.7)	790 (37.4)	848 (34.0)	2,129 (32.0)	277 (48.7)	4,450 (34.4)

*NA indicates not available.

TABLE III. Completion Rates by Age, All Clinics, 1979–1987

No. (%) Completed Treatment	0–14 yr	15–19 yr	20–34 yr	35+ yr	Total
Total completed	883 (46)	599 (39)	1,911 (28)	1,057 (37)	4,450 (37.5)
Non-infected contacts	177 (35)	39 (35)	99 (35)	91 (43)	406 (37)
Infected contacts	200 (47)	92 (39)	230 (29)	268 (40)	790 (37)
Converters	161 (48)	134 (41)	361 (28)	192 (36)	848 (34)
Reactors	310 (52)	314 (37)	1,157 (27)	348 (41)	2,129 (32)
Fibrocalcific lesions	35 (70)	18 (58)	61 (42)	163 (50)	277 (50)

period of at least six months. Tuberculin negative contacts, including children above the age of five, should not be placed on isoniazid. They should be retested in three months and, if found to have a positive response, should have a chest film taken. If the chest film is normal, these children should be given a six-month course of isoniazid. Children under the age of five who are close contacts with an adult with a positive sputum smear regardless of their tuberculin status should be given six months of isoniazid. Adult contacts who are reluctant to take medication should not be given medication; however, they should be encouraged to return for a follow-up chest film in three, six, and 24 months, or to return if symptoms develop. Close contacts who remain negative on retesting should be discharged from follow-up. Newborn babies born to mothers who are newly diagnosed as having tuberculosis on the basis of a positive sputum smear should be given three months of isoniazid; thereafter, they should be tuberculin tested, if positive, isoniazid should then be continued for a total course of six months. Should the mother's sputum remain positive, isoniazid should be continued until the mother's sputum has converted to negative.

In view of the poor compliance seen in this study, reactors who are screened for reasons of employment or other reasons should not be placed on chemoprophylaxis. Casual contacts need not be examined unless they have a risk factor, such as immunosuppression. HIV-positive, tuberculin-positive contacts or reactors should be strongly urged to take 9-to-12 months of isoniazid chemoprophylaxis.[14] Patients never before treated, with an abnormal chest film showing fibrocalcific lesions should be offered one year of isoniazids. If they do not wish to take treatment, they should not be started on treatment.[6,14] Tuberculin positive contacts or reactors who have a special condition, such as those undergoing therapy with steroids, immunosuppressive agents, chemotherapy, or radiotherapy, and those with diabetes silicosis, gastrectomy, gastrointestinal bypass, and chronic renal failure should be offered six months of isoniazid. If they cannot be persuaded to take medication they should be followed closely.

Isoniazid chemoprophylaxis will have no impact on the eradication of tuberculosis in the United States since most tuberculosis exists in big cities where prophylaxis does not work. Chemoprophylaxis is, in general, wasteful and unproductive. From a public health point of view, only certain high-risk persons should be placed on isoniazid therapy.

REFERENCES

1. American Thoracic Society statement: Chemoprophylaxis for the prevention of TB. *Am Rev Respir Dis* 1967; 96:558–560.
2. Ferebee SH: Controlled chemoprophylaxis trials in tuberculosis: A general review. *Bibl Tuberc* 1969; 17:28–105.
3. American Thoracic Society statement: Preventive treatment of T.B. *Am Rev Respir Dis* 1971; 104:460.
4. Farer L: Preventive treatment of tuberculosis: Basics of Respiratory Disease. 1973; 2:1–4
5. American Thoracic Society: Diagnostic standards and classification of tuberculosis. *Am Rev Respir Dis* 1981; 123:341–358.
6. Treatment of tuberculosis and tuberculosis infection in adults and children. *Am Rev Respir Dis* 1986; 134:355–363.
7. Tsevat J, Taylor WC, Wong JB, et al: Isoniazid for the tuberculin reactor: Take it or leave it. *Am Rev Respir Dis* 1988; 137:215–220.
8. Moulding T: Chemoprophylaxis: When is the benefit worth the risk and cost. *Ann Intern Med* 1971; 74:761–770.
9. Snider DE: Decision analysis for isoniazid preventive therapy: Take it or leave it? *Am Rev Respir Dis* 1988; 137:2–4.
10. Blackwell B: Treatment adherence. *Br J Psychol* 1976; 129:513–531.
11. Fox W: Compliance of patients and physician: Experience and lesions from tuberculosis. *Br Med J* 1983; 287:33–35,101–105.
12. Sbarbaro JA: Public health aspects of tuberculosis: Supervision of therapy. *Clin Chest Med* 1980; 1:253–263.
13. Thompson NJ: Efficacy of various durations of isoniazid preventive therapy for tuberculosis: Five years of follow-up in the I.U.A.T. trial. *Bull World Health Organ* 1982; 60:555–564.
14. Snider DE: Preventive therapy with isoniazid. Cost effectiveness of different durations of therapy. *JAMA* 1986; 225:1579,1583.

Pigeons as a remedy (*segulah*) for jaundice

Fred Rosner, MD

A popular treatment for jaundice in Israel (and elsewhere) is to place a pigeon on the navel of the patient to "transfer" the jaundice to the pigeon, so that the pigeon will die and the patient will recover. Sometimes more than one pigeon is required.

This type of remedy is called a *segulah* or nostrum, a form of medical treatment that has no rational or scientific basis but is nevertheless sometimes efficacious. It is not a folk remedy in the usual sense of that term. A *segulah* is an alternative or unconventional medical therapy, having a place in traditional Jewish practice alongside traditional scientific medicine. Elsewhere, I have written that Judaism sanctions alternative therapies such as prayers, faith healing, amulets, incantations and their like, when used as a supplement to traditional medical therapy.[1]

The present essay probes the source for the pigeon remedy (*segulah*) to treat jaundice, cites other treatments for jaundice in classic Jewish sources, briefly discusses pigeons in the Bible and the Talmud, and expounds on the concept of "transference" of illness and sin from a human being to a non-human species.

Pigeons in the Bible and the Talmud

In this discussion, the term pigeon, dove, and turtledove are used interchangeably. Doves and turtledoves are found in abundance in Israel where many varieties live, especially near Lake Tiberias. In this latter area, they are so abundant that several valleys are known as The Valley of Pigeons.[2] The dove is first mentioned in the Bible in connection with the story of the Flood,[3] where it is considered a symbol of peace because it brought back an olive leaf to Noah's Ark. Abraham used a dove when he made a covenant with the Lord.[4] Doves were used as a general purification offering.[5] The dove was brought as an offering by the poor[6] and by the Nazarite.[7]

Doves are monogamous, the female following the male.[8] At nesting time, the male coos.[9] The dove builds its nest in the clefts of precipitous rocks inaccessible to birds and animals of prey.[10] The Talmud mentions about ten varieties of doves and raising pigeons was probably popular in talmudic times. To race pigeons was forbidden, and bettors on pigeon races were unfit as witnesses in legal proceedings.[11] The Talmud refers to the catching of wild pigeons in nets.[12] The loft dove is a wild variety of pigeon found under the eaves of houses in Jerusalem, whereas the cote dove is bred.[13] The state of young doves' development was considered in intercalating the year.[14]

The Sages consider the dove to be persecuted like the Jews.[15] The dove symbolizes marital felicity,[16] as well as beauty, purity, and innocence.[17]

Pliny reports the use of pigeons by ancient navigators who released them when the ship lost its course. The captain would follow their flight direction to seek land.[2] Medieval Jewish poets such as Moses Ibn Ezra, Abraham Ibn Ezra, and Judah Halevi represent the Jewish people as a lovely turtledove longing for her lover.[18]

Moses Maimonides stated that turtledoves increase memory, improve intellect, and sharpen the senses. Pigeon eggs are good aphrodisiacs, and house pigeons increase natural body heat. On the negative side, young pigeons may produce migraine headaches.[19] (Maimonides also described the therapeutic efficacy of chicken soup.)[20]

Using Pigeons to Treat Jaundice

It is not clear when or where the use of pigeons to treat jaundice originated. The book most people point out when asked for a classic rabbinic source for this remedy is *Taamey Haminhagim* ("Explanation of Customs"). This book is a collection of laws and customs of the Jewish people. The book cites sources from early rabbinic and more recent *Responsa* literatures. The book was edited by Abraham Issac Sperling-Danzig in Lvov, Poland in 1896, reprinted in Lvov in 1906 and again in New York in 1944. The most recent revised edition was published in Israel in 1982. In a paragraph entitled, "Remedy (*Seguleh*) for jaundice which is called *gehl zucht* [yellowness]," the author writes the following:[21]

> Take a male pigeon for a male patient or a female pigeon for a female patient. Place it on the patient's navel. The pigeon will draw all the yellowness out [from the patient] and the pigeon dies. This [therapy] has been tested and is efficacious.

At the end of this brief description of the remedy, the source is cited in parentheses: a work called *Segulot Yisrael* ("Remedies of Israel"). This book, also known as *Sefer Hachayim*, ("The Book of Life") was authored by Shabtai Lifshitz and first published in Munkatch, Hungary in 1905.[22] The title page indicates that it is a collection of medical nostrums or remedies for spiritual and bodily healing based on a variety of books and other sources. The book was reprinted in Israel (undated) and again in Brooklyn, New York (undated) by the author's grandson, Isaiah Karpen. Section 8:2 of this work is entitled, "The Illness of Jaundice," and the following remedy is recommended:[23]

> A tested [and efficacious] therapy and remedy for the illness of jaundice called *gele zucht* [literally: yellowness] is to take a male pigeon for a male patient and a female pigeon for a female patient. Place it on the patient's navel, and the pigeon will draw the

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> yellowness out until it is finished. The pigeon dies. This [therapy] has been investigated and found to be efficacious. Another tested remedy for the aforementioned illness is for the patient to take a slice of bread and eat a little when he is actually moving his bowels. This should be done at the beginning of the illness when he observes the yellowness.

At the end of this paragraph, in parentheses, is the source citation: *Divrei Yitzchak* ("The Words of Isaac"). This work was authored by Yitzchak Isaac Weiss and was published in 1896 in Munkatch, Hungary. Remedy 130 on page 33 of that book states as follows:[24]

> I received word of a tested [and efficacious] remedy for the illness of jaundice called *gele zucht* and that is to take a male pigeon for a male patient and a female pigeon for a female patient. Place it on the patient's navel, and the pigeon will draw out all the yellowness therein until it is finished. The pigeon will die. This [remedy] has been tested and found to be efficacious.

There is no source indicated at the end of the paragraph suggesting that this book is the original rabbinic source for the pigeon therapy to cure jaundice. The title page of this book indicates that the remedies and therapies described therein were gathered from earlier holy and pious rabbis but the latter are not identified.

This pigeon therapy for jaundice is widely used in Israel and to a lesser extent in the United States and elsewhere. It is usually performed by non-physicians. Although most patients with hepatitis and jaundice recover spontaneously over several weeks time, the reported rapid recovery after the pigeon therapy suggests an additional effect. It is unclear why the pigeons die. Perhaps, they are held so tightly to the patient's abdomen that they suffocate. The pigeons do not become jaundiced, although they are supposed to absorb the patient's yellowness by transference. One reliable observer witnessed pigeon treatments applied to six patients with jaundice. All six patients survived, but the pigeons only died in two cases. Autopsies on the pigeons revealed ruptured spleens in both cases (personal communication from Rabbi M. D. Tendler, November 21, 1990). In another instance where a physician tried the pigeon therapy for a patient in hepatic coma, both patient and pigeon died (Personal communication from Rabbi A. Soloveitchik, November 23, 1990). Thus, the pigeon therapy is not always successful.

This unusual treatment relies to a considerable extent on the faith and belief of the patient and the practitioner. The scientific community, including the medical profession, tends to dismiss such healing as quackery. To true believers in nostrums, the explanation is simple—it is a miracle by divine intervention, perhaps associated with fervent prayers by the patient and others for recovery and cure from the illness.

A similar but earlier-described remedy for jaundice is the placement of live fish under the soles of patients suffering from jaundice or similar ailment. This remedy is cited by Shimshon Morpurgo in his book of *Responsa* published in Venice in 1743,[25] although the responsum in question was written in 1716. The question posed to the Rabbi was whether or not a patient with *hydrakon* (dropsy, ascites, or anasarca) is allowed to eat non-kosher snake meat as a remedy. This remedy was recommended by an expert physician because the patient failed to respond to all the standard therapies. Flesh of snakes was supposed to prolong life and was recommended already by Galen.[26] The Rabbi answered that since snake meat consumption is not a scientifically proven medical therapy, it is categorized as a nostrum or *segulah* and is therefore not permissible, since snake meat is not kosher. He defines standard medical treatments as those whose efficacy is known and predictable and are accepted and used by expert physicians. He contrasts such therapies with nostrums and amulets whose efficacy is not scientifically provable. This Rabbi was also a practicing physician and, in his responsum, points out that many remedies are used for many illnesses, but their efficacy depends not only on their scientific rationale, but also on the constitution of the patient and the circumstances of the time and place of the illness. He cites numerous examples of nostrums from classic Jewish sources such as the Talmud and Maimonides' *Mishneh Torah* and repeats the definition of a nostrum several times: a remedy which is sometimes efficacious and which has no scientific basis or rationale.

An even earlier source for the treatment of jaundice using fish to take away the patient's jaundice is found in a 16th or 17th century Hebrew manuscript in the library of the Wellcome Institute for the History of Medicine in London.[27] This manuscript is a medical notebook, including remedies for "urinitis, exhaustion in children and youths, and pain in the ileum." Remedy no 6 in this manuscript is as follows:

> For a person jaundiced because of illness, take a *tinga* fish, fresh and moist, tie it to the chest of the patient between the breasts, and leave it there until the *tinga* dies. Then one observes that the fish is yellow and the patient returns to the normal appearance. This [remedy has been tested (and found to be efficacious]).

Here the death of the fish can be readily explained by the fact that it cannot survive outside water. That the jaundice is transferred from the patient to the fish which then becomes jaundiced cannot be verified.

TRANSFERENCE TREATMENT OF OTHER ILLNESSES

Other transference of disease is mentioned already by Pliny.[26(p 252, note 192)] The Talmud describes a therapy for fever which is based on the transference principle.

> Let one sit at the cross-roads, and when he sees a large ant carrying something, let him take and throw it into a brass tube and close it with lead, and seal it with sixty seals. Let him shake it, lift it up and say to it, "Thy burden be upon me and my burden be upon thee." But if this is impossible, let him take a new pitcher, go to the river and say to it, "O river, O river, lend me a pitcher of water for a journey that had chanced to me." Let him then turn it seven times about his head, throw it behind his back, and say to it, "O river, O river, take back the water thou givest me, for the journey that chanced to me came in its day and departed in its day!"[28]

Zimmels cites Tobias Hacohen's work *Maaseh Tobiah* which mentions remedies called transplantations or transferences, the value of which was greatly exagerated by medicine men.[26(p141)] These remedies are all based on the principle that a body is able to transplant or transfer its nature to another, or that an illness can be transferred by grafting. One example given is that a man looses his illness when he places his excrement on a plant or seed growing in the earth, to which his illness will be transferred.

Zimmels also cites other examples of the transference of illness from the *Responsa* literature from the 17th and 19th centuries. For a child who could not sleep, old women advised the cutting of the nails of its fingers and toes and of a little of its hair which should be put into the shell of an egg and laid on a willow.[29] It was recommended that a man with epilepsy place his hand in the hand of a dead Gentile, saying, "Take this disease from me; it will do you no harm but will do me good."[30] Zimmels also suggests that selling the disease to another person is also a form of transference.[26(p 142)]

TRANSFERENCE OF SIN

The concept of transference of sin from a human being to an animal has been suggested as one of the explanations for the sacrifices offered in the Temple. The sinner is guilty of transgression for which the death penalty should be imposed.[31] However, as part of the repentance and atonement for the transgression, the guilt and hence the death sentence is transferred to the animal, which is then offered to G-d.

This scapegoat principle is illustrated by the law of the two goats on the Day of Atonement. The High Priest placed a lot over the he-goat for a sin offering and said, "For the Lord, a sin offering." The other goat was designated as a scapegoat and sent away. Thus, the High Priest effected atonement for himself, his family, and for all the Israelites.[32]

Nowadays, two annual customs performed by Orthodox Jews may illustrate the principles of transference of sin. These are the *Tashlich* ceremony, usually performed on the first day of Rosh Hashanah, and the *Kaparot* ceremony, usually performed on Yom Kippur eve.

The *Tashlich* (literally: throw away) prayers are customarily recited on the first afternoon of Rosh Hashanah at a body of water, preferably one that contains living fish. The custom began during medieval times. The opening prayer is a scriptural passage,[33] symbolizing hope that the Lord will dismiss the sins of the people on this day of judgment.

At the conclusion of the prayer recitation, some people cast crumbs or empty their pockets into the water perhaps symbolizing the transfer of one's sins to the fish in the water. According to at least one source,[34] there is an allusion to this emptying of one's pockets in the Scriptural phrase, "Also I shook out my lap and said: So G-d shake out every man from his house. . . ."[35]

The *Kaparot* (Atonement) ceremony is an ancient custom of taking a white rooster, for males, and a white hen, for females, on the eve of Yom Kippur and performing the *Kaparot* ritual, as follows. One takes the chicken in one's hand and recites a prayer three times.[36] Then one revolves the chicken around the head three times each time reciting the following phrase:[37]

> This is my exchange, this is my substitute, this is my atonement. This chicken will go to its death while I will enter and go to a long life, and to peace.

Fish or money may be substituted for the fowl. Some people use a different chicken for each person, although others use a single rooster for many men and a single hen for many women. A pregnant women customarily takes both a hen and a rooster: a hen for herself and in case she is carrying a female offspring, and a rooster in case she is carrying a male. This custom was banned by Moses Nachmanides (*Ramban*) and by Solomon ben Abraham Adret (*Rashba*) because it resembled the ways of the Amorites.[34(pp183–184)] Rabbi Joseph Karo, in the first edition of his *Shulchan Aruch*, calls it a foolish custom.[38] However, most rabbinic authorities and Orthodox Jews throughout the world participate in this ritual every Yom Kippur eve.

The custom of *Kaparot* symbolizes the scapegoat which the High Priest used to atone for the sins of the Israelites. The chicken is the symbol of the atonement. Some people use plants for the *Kaparot* ritual based on *Rashi's* interpretation of the word *parpisa* in the Talmud:[39]

> . . . they made baskets of palm leaves and filled them with earth and animal excrement [as fertilizer]. Twenty-two or 15 days before Rosh Hashanah, they designated one for each male and female child in the household and planted therein an Egyptian bean or pulse and they called it *parpisa* and it sprouted. On the eve of Rosh Hashanah, each person took his *parpisa* and revolved it around his head seven times saying: "This is instead of this, and this is my exchange and this is my substitute" and they would cast it into the river.

The casting in the river may again be symbolic of the transference of one's sins into the fish that live in the river, reminiscent of the *Tashlich* ritual described above.

OTHER TREATMENTS FOR JAUNDICE

Numerous therapeutic regimens are recommended in the Talmud for jaundice. Urine from an ass, if imbibed, is good for jaundice.[40] According to Rabbi Matia ben Cheresh, the flesh of a donkey should be eaten by someone suffering from jaundice.[41] Water of palm trees and a potion of roots are said to be efficacious for jaundice.[42] Water of palm trees was thought to "pierce the gall."[43] A useful potion of roots is explained by Rabbi Yochanan as follows:

> The weight of a *zuz* of Alexandrian gum is brought, a *zuz* weight of liquid alum, and a *zuz* weight of garden crocus, and they are powdered together . . . . For jaundice two-thirds thereof are mixed with beets and drunk, and the patient [although cured of his jaundice] becomes impotent.

Other remedies for jaundice are also mentioned in the Talmud:[43]

> . . . let him take the head of a salted *Shibuta* [name of a fish, probably mullet], boil it in beer, and drink it. If not, let him take brine of locusts. If brine of locusts is not available, let him take brine of small birds [alternate translation: clear fish brine], carry it into the baths, and rub himself therewith. If there are no baths, he should be placed between the stove and the wall [to make him perspire].
>
> Rabbi Yochanan said: "If one wishes to make the patient with jaundice warm he should wrap him well [or rub him] in his sheet. Rabbi Acha ben Jacob suffered therewith, so Rabbi Kahana treated him thus and he recovered. But if not, let him take three *kapiza* of Persian dates, three *kapiza* of dripping wax [dripping from an overflowing honeycomb], and three *kapiza* of purple aloes, boil them in beer, and drink it. If not, let him take a young ass; then the patient shaves half his head, draws blood from its forehead, and applies it to his own head, but he must take care of his eyes, lest the blood blind him. If not, let him take a buck's head which has lain in preserves [vinegar], boil it in beer, and drink it. If not let him take a speckled swine, tear it open, and apply it to his heart. If not, let him take porret [leeks] from the wastes of the valley [Rashi: from the middle of the furrow, where the leeks are sharp]."

In the rabbinic *Responsa* literature, remedies for jaundice include the drinking of urine[44] or the swallowing of eight lice taken from one's own head.[45] These folk remedies, like the nostrum of the pigeons on the navel, have no scientific validity but were used, perhaps for their placebo effect. Their efficacy was never in doubt to those who recommended them.

CONCLUSION

Biblical license is given to a human physician to heal, and biblical mandate is given the patient to seek healing from a human healer. Does this mandate also include alternative therapies such as a nostrum or remedy known as a *segulah* as exemplified by the pigeon treatment of jaundice? Leeches have been used for centuries to treat a variety of illnesses. Recently, scientific respectibility has been given to leech treatment as an acceptable medical therapy.[46,47]

In Jewish religious law, there is no objection to the use of amulets for healing purposes. Amulets are deeply rooted in popular belief. There is currently a popular belief, among some Jews, in the therapeutic efficacy of pigeons in the treatment of jaundice. The explanation offered for its alleged efficacy is the transference of illness from the patient to the pigeon. Other examples of disease transference therapies are recorded in Jewish sources. This concept of organic disease transference from the patient to a non-human living species has its parallel in the transference of sins from human to animal, as exemplified by the *Tashlich* and *Kaparot* rituals.

There is no medical or scientific evidence to support the therapeutic efficacy of the aforementioned pigeon remedy for jaundice. Anecdotal reports do not constitute such evidence. The death of pigeons during such attempted treatment can readily be explained by the pigeon suffocating from being held in a vise-like grip. An alternative explanation for the pigeon's death is exsanguination secondary to rupture of the spleen. To my knowledge, no reports have stated that the pigeons become jaundiced and no pre- and post-treatment bilirubin measurements have been carried out on patients or pigeons. Hence, such treatment must be viewed with a great degree of skepticism.

Traditional medical therapy is required because of the biblical mandate for Jewish people to seek healing from a physician when they are ill and not to rely solely on divine intervention or faith healing.

Acknowledgments. The author indebted to Edward Reichman, MD, for calling my attention to the Hebrew manuscript in the Wellcome Institute and to Mrs Annette Carbone for typing the manuscript.

REFERENCES

*ALL TALMUDIC REFERENCES ARE TO THE BABYLONIAN TALMUD

1. Rosner F: Unconventional Therapies and Judaism. *J Halacha and Contemp Soc* Number XIX, Spring 1990, pp 81–99.
2. Landman I: *The Universal Jewish Encyclopedia,* vol 3, New York, 1941, pg 592. Roth C: *The Encyclopedia Judaica,* vol 6, Jerusalem, Keter, 1972, pp 184–185.
3. Genesis 8:8–12.
4. Genesis 15:9–10.
5. Leviticus 12:6 and 15:14; numbers 6:10.
6. Leviticus 5:7.
7. Numbers 6:10.
8. Hosea 7:11.
9. Isaiah 59:11.
10. Jeremiah 48:28.
11. Sanhedrin 25b, Rosh Hashanah 22a.
12. Baba Kamma 7:7.
13. Tosefta, Betzah 1:10.
14. Sanhedrin 11a.
15. Baba Kamma 93a.
16. Erubin 100b.
17. Song of Songs 1:15 and 5:2.
18. Based on Psalms 74:19.
19. Rosner F, transl: *The Medical Aphorisms of Moses Maimonides,* Haifa, Maimonides Research Inst, 1989, pp 304–306.
20. Rosner, F: Therapeutic efficacy of chicken soup. *Chest* 1980; 78:672–674.
21. Sperling AI: *Sefer Taamey Haminhagim Umekorey Hadinim,* Jerusalem, Eshkol, rev ed. 1982, p 574.
22. Lifschitz S: *Sefer Hachayim Hanikra Segulot Yisrael,* Munkatch, Hungary, no publ listed, 1905
23. Karpen I: *Sefer Hachayim Hanikra Segulot Yisrael,* 3rd printing, Brooklyn, New York, Edison, undated, p 88.
24. Weiss I: *Sefer Divrei Yitzchak,* Munkatch, Hungary, Meisels printers, p 33.
25. Morporno S: *Sefer Shemesh Tzedakah,* Venice, 1742, responsum no 29 pp 80–83.
26. Zimmels HJ: *Magicians, Theologians, and Doctors.* London, Goldston and Son, 1952, p 245, notes 51–52.
27. Allan N: Catalogue of Hebrew manuscripts in the Wellcome Institute, London. *J Semitic Studies,* vol 27, Autumn 1982; 193–220.
28. Shabbat 66b.
29. Ashkenazi M: Responsa *Panim Meirot,* pt 1, no 36.
30. Sofer M: Responsa *Chatam Sofer, Yoreh Deah* no 339.
31. Nachmanides M: *Ramban's* Commentary on Leviticus 1: 5–6.
32. Maimonides M: *Mishneh Torah, Avodat Yom Hakippurim* 2: 6 and 3: 4.
33. Micah 7: 18–20.
34. Eisenstein JD: *Otzar Dinim Uminhagim,* New York, Hebrew Publ Co, 1917, p 449.
35. Nehemiah 5: 13.
36. Psalms 107: 10, 14, 17–21 and Job 33: 23–24.
37. Sherman N: *The Complete Artscroll Siddur* (Prayer Book), Brooklyn, New York, Mesorah, 1984, p 775.
38. Karo J: *Shulchan Aruch, Orach Chayim* 605: 1.
39. Shabbat 81b.
40. Bechorot 7b.
41. Yoma 84a.
42. Shabbat 109b.
43. Shabbat 110a.
44. Shapira ZH: *Darchei Teshuvah,* chap 155 no 16.
45. Bachrach Y: Responsa *Chavot Yair* no 105.
46. Bates M, Kneer K, Logan C: Medicinal leech therapy: An overview. *Orthop Nurs* 1989; 8:12–17.
47. Kraemer BA, Korber KE, Aquino TI, et al: Use of leeches in plastic and reconstructive surgery: A review. *J Reconstr Microsurg* 1988; 4:381–386.

Pulmonary cavitary disease

Case Presentation

A 48-year-old man was admitted to the Manhattan Veterans Affairs Medical Center with a dry cough and shortness of breath of two months duration. The cough was most severe when he was in the supine position. It persisted in spite of antitussives prescribed by his private physician and was not associated with fever, chills, night sweats, exertional dyspnea, or hemoptysis. Over several months before admission, the patient had a weight loss of 10 lb.

Two weeks before admission, shortness of breath on exertion developed, forcing him to discontinue his work as a plumber and oil burner mechanic. He denied wheezing, nausea, dysphagia, headache, or myalgias. Several days before admission, he noted blood-streaked sputum; after a chest radiograph, a second private physician recommended admission to the hospital.

The patient reported occasional dizziness during the inhalation of fumes and chemicals in the course of his employment; he denied syncope or loss of consciousness. He experienced mild chest discomfort with occasional radiation to his right scapular region. The patient's medical history was notable only for excision of a pilonidal cyst 25 years previously. He denied allergies, use of medications, cigarette smoking, or alcohol abuse.

The patient was reared in Tennessee; he denied any risk factors for HIV infection. When in his early 20s, during military duty, he was stationed in New Mexico for two years but was never assigned to overseas duty; he remained well during his tour of duty. At age 28, he moved to Brooklyn. His employment recently included conversion of homes from oil to natural gas heating. There was no family history of illnesses; his wife and two children were in good health.

Physical examination disclosed a well developed, thin man in no acute distress. His blood pressure was 110/70 mm Hg; pulse rate was 86/min with a regular rhythm; respiration rate was 22/min; temperature was 98° F. His head, eyes, ears, nose, and throat were unremarkable; the neck was supple; and no carotid bruits were audible. The thyroid was not enlarged. The lungs were clear to percussion and auscultation, without wheezes or rhonchi. The first and second heart sounds were normal; the cardiac rhythm was regular; no abnormal heart sounds, murmurs, or rubs were audible. Percussion outlined a normal-sized liver; the spleen was not palpable. There was no abdominal tenderness or abnormal bowel sounds; the stool guaiac test for blood was negative. Occasional shotty inguinal lymph nodes were palpable, but not in other locations. The extremities demonstrated no cyanosis, clubbing, or edema. The neurologic examination was within normal limits.

The urine was clear, with a pH of 5.0, specific gravity 1.032, and negative tests for glucose, acetone, bile, urobilinogen, and calcium; the test for protein was trace positive; the microscopic examination revealed only 2–3 white blood cells. The hemoglobin count was 13 g/dL; hematocrit was 40%, white blood cell count was 12,000/μL, with 79% mature polymorphonuclear leukocytes, 6% immature poly morphonuclear leukocytes, 6% lymphocytes, 5% monocytes, and 1% eosinophils. The platelet count was 452,000/μL. The prothrombin time was 12.1 seconds, and the partial thromboplastin time was 38 sec. The blood urea nitrogen was 25 mg/dL, and the serum creatinine was 1.2 mg/dL. Serum electrolyte levels were as follows: sodium, 138 mmol/L; potassium, 4.5 mmol/L; chloride, 103 mmol/L; bicarbonate, 27 mmol/L; calcium, 9.5 mg/dL; and phosphate, 3.1 mg/dL. The alkaline phosphatase level was 151 U/L; total serum protein was 7.2 g/dL (albumin was 4.2 g/dL); serum cholesterol was 191 mg/dL; direct serum bilirubin was 0.4 mg/dL; serum aspartate aminotransferase (SGOT/AST) was 18 U/L; and serum amylase was 120 U/L.

Arterial blood gas determinations were reported as follows: pH 7.46; CO_2 33 mm Hg; O_2, 100 mm Hg; bicarbonate, 24 mmol/L Hg; and oxygen saturation, 98%. The purified protein derivative (PPD) skin test for tuberculosis was negative; the anergy panel tests were reported as "positive." The posteroanterior and lateral chest films showed a cavitating mass in the anterior segment of the right upper lobe, together with prominence in the right hilar region (Figs 1,2).

Throughout his hospital stay, the patient remained afebrile; cough and shortness of breath persisted. Pulmonary function tests were reported as normal. A computed tomographic (CT) scan of the lungs confirmed a cavitary lesion in the anterior segment of the right upper lobe; right peritracheal adenopathy was also identified (Fig 3). Cytologic study of sputum and sputum smears for acid-fast bacilli were repeatedly negative.

On the ninth hospital day, bronchoscopy revealed a normal tracheobronchial tree; bronchial washings and brushings were negative on cytologic study. Transbronchial biopsy obtained from the anterior segment of the right upper lobe revealed normal bronchial wall tissue with mild chronic inflammation and normal lung parenchyma. Fungal and bacterial cultures, as well as acid-fast bacillus smears, were negative.

On the 16th hospital day, the patient underwent a second bronchoscopy with transbronchial biopsy, with similar results. On the 25th hospital day, an open lung biopsy was performed.

Aldona Wos, MD (Assistant Attending Physician, Pulmonary Division): In summary, we have a 48-year-old man, who entered the hospital with a two-month history of a dry cough and shortness of breath, followed by hemoptysis during the two weeks before admission. The only significant

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Based on a clinical conference held at New York Infirmary-Beekman Downtown Hospital, New York, NY, on January 29, 1991.

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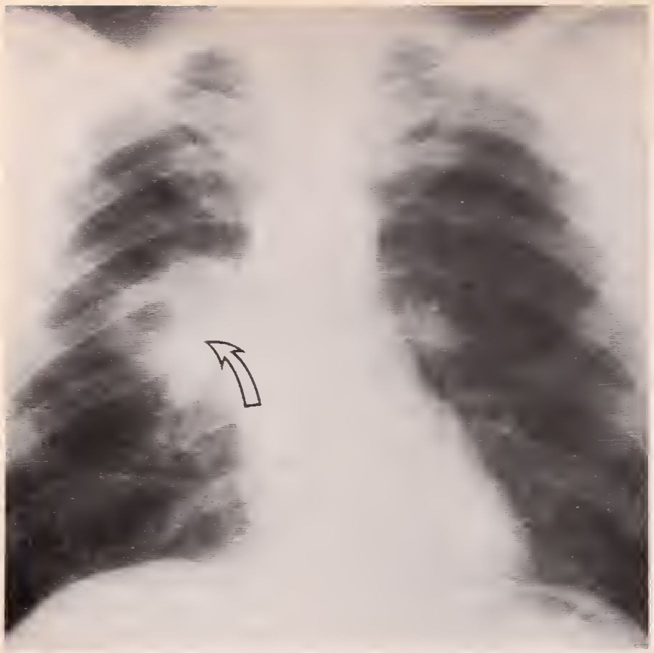

FIGURE 1. Posteroanterior chest film showing cavitation in the right lung (arrow).

history we have is that he worked as a plumber and an oil-burner mechanic for more than 16 years of his professional career. He also had a ten-pound weight loss over several months. Among other important information is the fact that during his military career he was stationed in New Mexico but that was more than 15 years before this admission.

The physical examination on admission to the hospital was unremarkable, and laboratory findings were positive only for a mildly elevated serum alkaline phosphatase level and a mildly elevated serum lactate dehydrogenase count. The complete blood count and the remainder of the laboratory work were within normal limits. His arterial blood gas pressures were within normal limits, although we are not told the ambient ventilatory conditions; a PPD test was negative, although his anergy panel was positive.

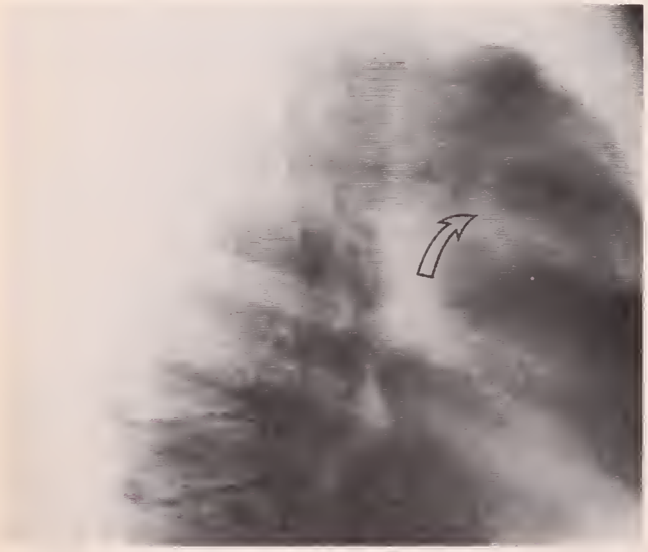

FIGURE 2. Right lateral chest film showing cavity in anterior portion of right upper lobe (arrow).

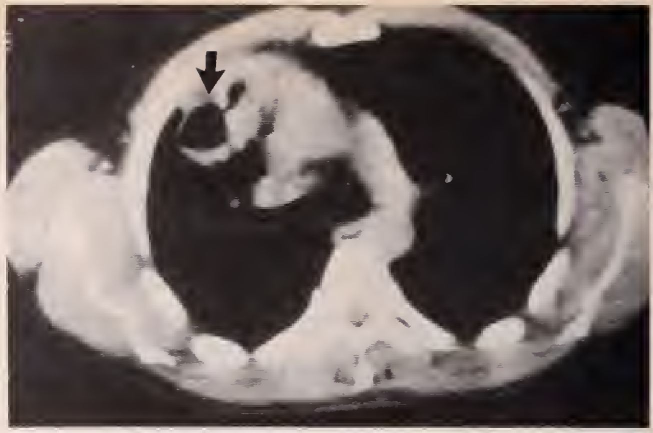

FIGURE 3. CT of chest showing cavity in right upper lobe (arrow).

On hospital admission, he was noted to have abnormal radiographic findings and CT scans. No definite diagnosis was established. A bronchoscopy was performed, and no diagnosis was obtained. Subsequently, on his 25th hospital day, an open lung biopsy was performed. Before I present my differential diagnosis, Dr Lubitz will review the radiographic findings for us.

Alan Lubitz, MD (Attending Radiologist): On the anteroposterior view of the chest, the heart size is normal; in the right parahilar region, there is a cavitating mass, and the right hilar region is enlarged (Fig 1). It is hard to determine if this mass is in the hilum or if there is a separate cavitating mass and a separate right hilar mass because of adenopathy. The lateral chest film again provides us with a view of the cavitating mass lesion in the anterior segment of the right upper lobe, and there is also prominence in the hilar region (Fig 2). An oblique film of the chest again shows the cavitation lesion.

The CT scan of the chest shows a large cavitating lesion in the anterior segment of the right upper lobe (Fig 3). I do not have the entire CT scan of the chest for review. In the radiologic report, however, it is stated that there was right paratracheal adenopathy. There is no mention of the hilum or whether there was hilar adenopathy. The mass is in continuity with the mediastinum on several cuts. The right pulmonary artery is visible, but in the right hilum I cannot tell whether it is enlarged or whether there is adenopathy.

Basically, we are dealing with a cavitating mass in the right upper lobe, supposedly with paratracheal adenopathy. We do not know whether there was any hilar adenopathy, either bilateral or unilateral.

Dr Wos: I will discuss the differential diagnosis of pulmonary cavitary diseases, grouped under nine different causes.

Pulmonary cavitary disease possibly caused by developmental lesions can be confined in this patient to bronchogenic cyst or intralobular bronchopulmonary sequestration. Both of these lesions we can quickly exclude because neither are likely in a 48-year-old man, and they are not associated with progressive symptoms of cough, shortness of breath, weight loss, or hemoptysis. Intralobular bronchopulmonary sequestration is usually located in the left lower lung field, and the lesions may be multiple. Bronchogenic cysts are also usually located in the left lower lung field. Therefore, we can exclude the category of developmental lesions.

The next category is traumatic causes of plumonary cavitary disease. In this patient, pulmonary parenchymal laceration can be excluded because of the absence of a significant history of trauma.

A third category is that of thromboembolic disease, including septic emboli. These emboli commonly originate from vegetations of endocarditis of the tricuspid valve or an interventricular septal defect or from peripheral thrombophlebitis. They occur more commonly in intravenous drug abusers, in patients with tonsillar or pharyngeal infections or with pelvic infections and in patients with arteriovenous shunts or with indwelling catheters. The manifestations in these cases are usually located in the left lower lobe; the cavitary lesions are usually multiple and are commonly associated with pleural effusions. We can safely exclude this type of lesion because of the lack of supportive findings in this patient.

The fourth category is that of airway disease, leading to pulmonary cavitary disease. Here we include two examples. One is cystic bronchiectasis, which is usually seen in the pediatric age group, is bilateral, and includes the lower lung fields. The other example is that of blebs and bullous disease. In primary bullous disease, the patient usually is asymptomatic, but by the time he becomes short of breath, you usually find evidences of obstructive emphysema. On chest films, this disease is usually found in the upper lobes at the apices, and again, usually against a background of emphysema. This patient had normal pulmonary function tests, and no evidence of bleb and bullous disease on chest films.

The fifth category is immunologic disease. The immunologic disorders that cause cavitary disease include Wegener's granulomatosis and rheumatoid nodules. In Wegener's granulomatosis, the onset is usually insidious, commonly in the 50-to-60 year old age group, and is more frequent in the male than in the female. The patients present with nasal, sinus, and chest symptoms, commonly with cough and often with hemoptysis; the majority of patients exhibit renal manifestations. Wegener's granulomatosis affects many organs: the respiratory tract in 100% of patients; the kidneys in 83%; joint involvement in 50%, and muscle, eye, heart, pericardial, and central nervous system involvement in many patients. Except for pulmonary disease, we detected none of these manifestations in the patient we are discussing today, according to the available information.

The radiographic manifestations of Wegener's granulomatosis can include cavitary lesions, usually multiple, one third to one half of which are bilateral when they cavitate. There is no anatomic significance to their location in the lungs, but typically in Wegeners' granulomatosis, the radiographic picture is that of round multiple opacities, with rare involvement of the hilum. On the basis of all these considerations, we can safely exclude this entity.

Another example of immunologic cavitary disease is that of rheumatoid nodules, which are very rare manifestations of pleuropulmonary rheumatoid disease. They are usually associated with advanced rheumatoid arthritis, most commonly in patients with clinically evident subcutaneous nodules. The cavities usually form in the lower lobes, commonly are multiple, and are frequently associated with pleural effusions and pneumothorax. These manifestations of rheumatoid arthritis were not present in this patient, so we can safely exclude that possibility.

The sixth category of pulmonary cavitary disease is that of idiopathic entities. Sarcoidosis is an example of idiopathic pulmonary cavitary disease, although such cavitations in sarcoidosis are very uncommon. In one study of a combined series of 1,254 patients with thoracic involvement, only eight patients with sarcoidosis demonstrated cavitary lesions.[1] When these lesions occur, there is no characteristic location for the cavities on the chest film. They are associated with lymph node enlargement, and usually with bilateral hilar involvement. They are also associated with pleural effusions, atelectasis, and spontaneous pneumothorax, of which we have no evidence in this case.

The clinical manifestations associated with cavitary sarcoidosis in the lung include a dry cough, shortness of breath, and hemoptysis. On pulmonary function tests, there is usually decreased diffusion capacity and a restrictive pattern, neither of which this patient demonstrated. In addition, laboratory findings in cases of sarcoidosis can include an elevated serum calcium level, an elevated serum globulin level, and anemia, none of which were present in this patient. In addition, sarcoidosis can be associated with an abnormal serum protein electrophoresis, a high serum level of angiotensin-converting enzyme, and hypercalciuria, none of which were apparent in this case. Thus, a patient with sarcoid may present with a variety of signs and symptoms, involving many organ systems, but in this patient, we have no clue of such findings.

A seventh category of diseases that cause pulmonary cavitary lesions are secondary to inhalation factors. Two prominent examples include coal workers' pneumoconiosis, which can be excluded on the basis of absence of an appropriate history. In this entity, there is a tendency to form cavities usually in the upper lobes. However, the remaining portions of the lungs will show multiple small opacities. So again, we can exclude this condition. The second is silicosis, in which the cavities are most prominent in the upper lung fields, but again the remaining lung background is abnormal, commonly demonstrating a reticular-nodular pattern. The disease is associated with enlarged hilar lymph nodes, which are usually calcified; that is not the case in this patient. The occupational history usually involves tunneling or mining, exposure to sandstone, stone carving, granite working, or foundry work, so we can safely exclude this possibility.

The eighth category of conditions possibly causing pulmonary cavitary disease is neoplastic disorders. Here we have three examples: Hodgkin's disease, metastatic disease, and bronchogenic carcinoma. Hodgkin's disease can contribute to the roentgenographic evidence of cavitation in the lungs, which is frequently multiple and associated with mediastinal hilar lymph node enlargement. When cavities form, they are usually found in the lower lobes. This patient demonstrates features that could be compatible with Hodgkin's disease in some respects and not in others.

Ordinarily, in 90% of those patients with Hodgkin's disease who seek medical advice, the clinical manifestations include palpable peripheral lymphadenopathy, night sweats, and fever, none of which were reported by this patient. However, he did have other common symptoms of the disease, such as weight loss, cough, and shortness of breath. At this time, we cannot exclude the possibility that the patient had a form of Hodgkin's disease.

The next example of neoplastic cavitation is that caused by metastatic disease, usually associated with multiple pulmonary nodules, several of which cavitate at some point in time. When this occurs, it is usually in the upper lobes. The clinical signs and symptoms are usually related to a primary site of the disease, of which we have no clue in this patient. I believe that metastatic disease can be safely excluded.

The third example of neoplastic disease is bronchogenic carcinoma. In this condition, cavitation occurs in from 2%–16% of patients. Most of the lesions are located in the periphery of the lungs, have a predilection for the upper lobes, and are associated with unilateral hilar enlargement. The majority of such cavitary lesions develop in squamous cell carcinoma. Clinically, the patients tend to be men between 50 and 60 years of age; in 75% of the cases, the patients present with cough and 50% of the time with hemoptysis, as did this patient. The initial symptoms sometimes suggest a block somewhere in the respiratory passages because of a distal partial obstruction of a bronchus by the tumor. This patient's symptoms did not suggest such events.

We know of the close association between cigarette smoking and bronchogenic carcinoma, but this patient was said not to be a smoker. However, as a plumber, he may have had exposure to certain industrial substances; the most noxious and pathogenic of these that are associated with bronchogenic carcinoma are asbestos, uranium, nickel, and toxic gases from coal dust. So at this point, we cannot exclude the possibility that the patient may have had bronchogenic carcinoma.

The ninth category of diseases, causing cavitation in the lung, is infection; here we can divide the entities into bacterial, fungal, and parasitic. We can with reasonable safety exclude the parasitic group, in particular infection with *Endameba histolytica*, because the resulting cavities are almost exclusively located in the right lower lung field and are usually associated with a right pleural effusion. The endameba enters the right hemithorax by extension from a liver abscess. We can safely exclude the likelihood of cavitary disease in this patient being secondary to parasitic disease.

Of the fungal infections that cause cavitary disease in the lung, cryptococcosis usually presents as a single mass, from 2–8 cm in diameter, with only 15% developing cavitary lesions. Usually the lesion is peripheral and located in the lower lobes. In cryptococcosis, it is uncommon for major hilar lymph node enlargement to develop.

Another fungus, *Nocardia asteroides*, usually presents with a patchy nonsegmental infiltrate of the lower lobes; commonly the infection is associated with pleural effusions. The parenchymal infiltrate can progress to cavitation, usually associated with cough, night sweats, and sputum production. This patient had only a dry cough over a two-month period.

Another fungus, *Histoplasma*, is endemic in the central and eastern United States, and in general, has already generated a positive skin test by the time of the patient's initial presentation. This disease can be associated with an elevated white blood cell count, a positive serologic test, and a positive latex fixation test, of which we have no reports for this patient. In primary histoplasmosis, the common finding is that of nodules, usually calcified, in the periphery of the lungs. In chronic histoplasmosis, cavities can develop, are usually multiple, and are usually located in the upper lobes.

Another fungus, *Coccidioides immitis*, causes a lung infection, initially with a parenchymal consolidation, and then usually produces multiple cavities, most often in the upper lung fields. The fungus is endemic in the southwestern United States, where this patient was stationed many years ago. A patient so infected can be asymptomatic for a great many years. The disease can be associated with hemoptysis, cough, and weight loss, as this patient experienced and can be accompanied by greatly enlarged hilar lymph nodes. Reactivation of this fungal infection in a patient usually occurs during periods of immunosuppression, in association with renal transplantation, Hodgkin's disease, or lymphoma. This infection remains within the differential diagnosis here and cannot be excluded, although I doubt its presence very much.

The last category of infectious diseases that cause pulmonary cavitation are bacterial infections. Among these examples are *Staphylococcus aureus* which, when it produces pulmonary cavitation, will do so as a bilateral process, usually associated with pleural effusions, and usually as a complication of pneumonia after a viral infection. It can also be secondary to septic emboli from infected pelvic veins or intravenous catheters.

Other bacterial infections that can cause cavitary lesions are those caused by *Klebsiella, Serratia,* and *Enterobacter.* The usual findings are those of a homogeneous parenchymal consolidation with an air bronchogram and characteristic bulging of a fissure. However, such lesions are usually single, associated with pleural effusions, and most commonly located in the upper lung fields. They commonly occur in men over 60 years of age or in individuals who are debilitated from disease or from alcohol abuse.

Pulmonary infections caused by bacteria, such as *Pseudomonas* and *Eschericia coli,* may undergo central cavitation, usually in the lower lobes, and are frequently associated with empyema, and again appear more commonly in debilitated patients, alcohol abusers, and diabetics. However, the initial chest film findings are not those of a cavity, but rather of bilateral consolidation of the lower lobes. Other bacterial causes include *Bacillus proteus*, which gives rise to a nonsegmental homogeneous consolidation, usually in the posterior segment of the upper lobes, where areas of cavitation may occur.

Streptococcal infection can lead to pulmonary cavitation, most commonly in patients with hepatic cirrhosis, diabetes, renal failure, or after splenectomy. Usually the cavities occur in the lower lobes or in the posterior segments of the upper lobes, as in this patient. Radiography characteristically discloses consolidation with an air bronchogram; cavitation, although it can occur, is very rare.

Infection with anaerobic organisms can frequently lead to pulmonary cavitation, more commonly in the right lung, and usually in the posterior portions of a lung. The diagnosis of anaerobic infection of the lungs is suggested by various characteristics: an insidious infection, in a debilitated patient with poor oral hygiene, cough productive of purulent sputum, and a clinical history that raises the suspicion of pulmonary aspiration. However, many facts suggestive of infections by any of these organisms are not found in this patient's history. Furthermore, when patients present with pulmonary cavitation from bacterial infections of any type, the clinical course is usually abrupt in onset and is usually accompanied by pleuritic chest pain, cough, and purulent sputum production. Therefore, it is unlikely that this patient fits into this category.

With infections caused by mycobacteria, the resulting cavities can be multiple and are more prominent in the atypical mycobacterial diseases than in *Mycobacterium* *tuberculosis* infections. Primary pulmonary tuberculosis is usually seen in the upper lobes, most frequently as a consolidation with ill-defined margins, and rarely with cavitation. In post-primary tuberculosis, there are characteristic changes in the apical and posterior segments of the upper lobes, and sometimes in the apical regions of the lower lobes, as was not true in this patient. The clinical manifestations usually involve a protracted course, persistent pleuritic pain, fever, weight loss, weakness, cough, and a positive 5 tuberculin-unit PPD test, which this patient did not have.

In summary, of the different categories of disease that can present as a cavitary lesion in the lung, the first choice in my differential diagnosis of this patient's disease would be a malignant neoplasm; and among the malignant diseases, the two that I cannot exclude based on the information available are first, a form of Hodgkin's disease or lymphoma, and secondly, a form of bronchogenic carcinoma. Of the fungal infections, it is possible, determined on the basis of the history of the patient's location during military service, that coccidioidomycosis might be the diagnosis, but it is unlikely.

Irwin Sharkey, MD (Chief of Medicine): However, we have the results of two bronchoscopies here, both of which were negative.

Dr Wos: Yes, there were two bronchoscopies that were negative on specimens sent for culture, antibiotic sensitivity, acid-fast bacilli, fungi, and cytologic study. Subsequently, another procedure was performed, which I presume was a thoracotomy with an open lung biopsy, to obtain a pathologic diagnosis.

Dr Sharkey: So what would be your best guess as to the diagnosis?

Dr Wos: As I indicated, my best guess still would favor a malignant lesion, and the differential diagnosis would be between bronchogenic carcinoma and lymphoma. The only substantive information in that regard is the patient's relatively young age, that is, 48 years, and that he had not been a smoker of cigarettes. Both facts would make bronchogenic carcinoma less likely. Then again, in his employment as a plumber he may well have been exposed to asbestos dust for about 16 years, so it is quite possible that he does have bronchogenic carcinoma. On the other hand, the small elevations of the serum alkaline phosphatase and of the serum lactate dehydrogenase, although not specific, would suggest that we not exclude the possibility of lymphoma, on the basis of the data available.

Harold Coppersmith, MD (Attending Physician): Are you surprised at a diagnosis of a malignant lesion in a patient whose course had progressed at a fairly rapid rate, but in whom fever did not occur?

Dr Wos: No, because practically every disease entity that I discussed should be accompanied by fever.

Dora Barnes, MD (Attending Physician): I think the possibility of histoplasmosis should not be overlooked. Here is a man who was raised in Tennessee, and we know that in the Tennessee Valley area histoplasmosis is very common. Could not his clinical presentation be indicative of this disease?

Dr Wos: Histoplasmosis is usually associated with calcified lymph nodes, which we do not see in this patient. He left Tennessee, as I understand, at a young age, before his military service, over 20 or 25 years before his presenting symptoms. Therefore, on the basis of the time sequence, I would assume that histoplasmosis would be unlikely.

Daniel Muppidi, MD (Medical Resident): If this is a malignant lesion, how likely is it that this lesion could be missed on bronchoscopy and cytologic study?

Dr Wos: On the basis of its location, if the hilum is involved it might be easy to miss the lesion on bronchoscopy. The pulmonologist must have been convinced that there was something there, however, because he repeated the procedure in the hope of making the diagnosis. Failure to make the diagnosis on bronchoscopy is quite possible, even though there is an abnormal CT scan.

Dr Sharkey: In a relatively young man, with a thick-walled cavity in the anterior segment of the right upper lobe and with paratracheal node involvement, you should place malignant disease at the top of your list. However, the patient's symptoms seemed relatively mild, and he was not a smoker, so you are faced with the possibility that the answer might be any one of the conditions that you reviewed for us.

CLINICAL DIAGNOSES

Cavitary lesion of the right upper lobe, most probably caused by lymphoma, possibly secondary to bronchogenic carcinoma.

RADIOLOGIC PRESENTATION.

Gurdip Sidhu, MD (Attending Pathologist, Manhattan Veterans Affairs Medical Center, Associate Professor of Pathology, NYU School of Medicine): I would like Dr Lubitz, the radiologist, to express his opinion about the diagnosis, on the basis of the radiographic findings.

Dr Lubitz: I would place carcinoma at the top of my list.

Dr Sidhu: There were no intra-abdominal radiologic findings as far as I know. A thoracotomy was performed, and we felt that a malignant lesion was present on frozen section. A resection of the right upper and middle lobes was then performed.

The mass in the lung was very large, 10 cm in diameter, but with no direct extension into the hilum. Two mediastinal nodes were removed. I will mention more about them later.

The periphery of the mass was yellowish, whereas the center consisted of a cavity containing purulent material, in spite of the fact that the patient never had fever. The central cavitated area contained many polymorphonuclear leukocytes. The infiltrate presented a mixed cellularity, with many neutrophilic leukocytes and some large cells that were probably histiocytic.

In other areas, the infiltrate was organized into nodules with surrounding, thick, dense fibrosis (Fig 4). The interior of the nodules again contained some very large cells, scattered in a background of other cells: polymorphonuclear leukocytes, many small lymphocytes and plasma cells, and a number of other cells with bizarre nuclei, often multinucleated. Some of these bizarre cells had prominent nucleoli and could be immunoblasts, but it is difficult to characterize them. A long, careful search disclosed cells with two nuclei, each of which had a very large prominent nucleolus. A high-power view of such cells identified them further as Reed-Sternberg cells (Fig 5). So this patient's illness was Hodgkin's disease, of the nodular sclerosing type.

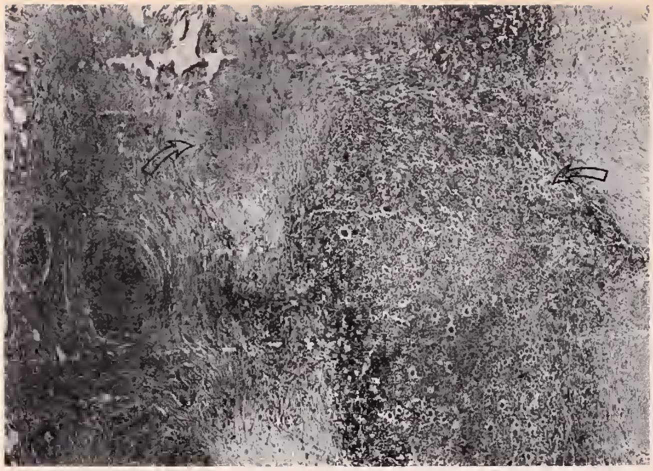

FIGURE 4. Microscopic section of mass in resected right lobe of lung, showing infiltrate of Hodgkin's tumor cells (right arrow) surrounded by densely sclerotic fibrous tissue (left arrow). (Hemotoxin phloxine saffron [HPS] stain; original magnification × 40)

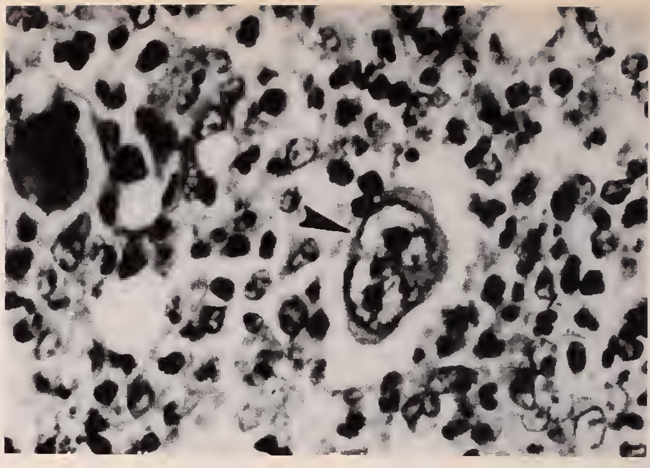

FIGURE 5. Reed-Sternberg cell (at arrow) in background setting of lymphocytes, histiocytes, plasma cells, and neutrophils. (HPS stain; original magnification × 400)

The mediastinal lymph nodes were enlarged and hyperplastic, and one of the nodes had one small focus of neoplastic infiltrate. On the node alone, one could not have stated unequivocally that this was Hodgkin's disease, since there were no Reed-Sternberg cells. However, the node had the same background, with mononuclear variants. Because of this association, we decided that the condition in the lymph node was Hodgkin's disease.

Primary Hodgkin's disease of the lung is very rare;[2] there have been only about 50 cases reported in the medical literature. In such cases, the majority are of the nodular sclerosis type.

Hodgkin's disease in the lung is primary if it presents there with minimal involvement elsewhere. In most such cases, the disease in the lung presents as nodules, either single or multiple; when multiple, the nodules tend to be bilateral. The nodules frequently will cavitate, especially when large and single.

On rare occasions, Hodgkin's disease may present in the lungs with a lymphangitic pattern; it may also be associated with small nodules or a reticulonodular kind of infiltrate. However, probably the most frequent presentation of this rare form of the disease is that demonstrated by this patient. One is more likely to find a lymphangitic spread or a reticulonodular infiltrate in the lung in a patient with non-Hodgkin's lymphoma.

In the lung, Hodgkin's disease generally occurs in a slightly older age group than when it occurs in other organs. The male:female ratio is about 2:1.

This particular patient was given combination chemotherapy, went into complete remission in 1983, and has not had a relapse since then. It appears that he has been cured.

Michael Daniels, MD (Chief Medical Resident): What stage was this patient's Hodgkin's disease?

Dr Sidhu: I am not certain, except that the bone marrow was negative. The CT scan was otherwise negative, including the evaluation of abdominal lymph nodes.

Dr Daniels: Reed-Sternberg cells are not disease-specific for Hodgkin's disease. They can be found in Epstein-Barr virus infections and certain other infectious processes. Was the diagnosis based only on the presence of Reed-Sternberg cells?

Dr Sidhu: That is a problem that nonpathologists frequently have. The diagnosis of Hodgkin's disease is not based on the Reed-Sternberg cells alone. It is based on the background of a mixture of lymphocytes, plasma cells, histiocytes, and polymorphonuclear cells. The background of such a mixture does not occur in the other entities in which you find Reed-Sternberg-like cells. If you had only the results of cytologic studies in which you found Reed-Sternberg cells, you could not be sure that the condition was Hodgkin's disease. However, with the availability of tissue for sectioning and study, Hodgkin's disease will exhibit a background of other cells as well. If Hodgkin's disease is present elsewhere in the body, as evidenced by a fresh biopsy from, for example, the bone marrow, and if you find no Reed-Sternberg cells, yet you have the requisite cellular background in the biopsy, it is considered quite reasonable for the pathologist to make a diagnosis of Hodgkin's disease.

John T. Flynn, MD (Associate Chief of Medicine): Looking back on the case now, would there have been any method, other than the lung biopsy, of making the diagnosis before operation?

Dr Sidhu: One could have done a fine-needle aspiration; but for Hodgkin's disease that is not a good diagnostic procedure. To diagnose Hodgkin's disease, you need to see the overall cellular pattern, as I said. You still need a surgical biopsy. If you are going to do an open lung biopsy, you might as well get consent for a surgical resection of the lesion, which is essentially what the surgeons did in this case. It is quite possible that none of the disease processes remained after surgery. Thus, it is possible that the chemotherapy might have had no role in the cure.

FINAL DIAGNOSIS

Hodgkin's disease apparently confined to the lung, nodular sclerosis type, with cavitation and focal involvement of a mediastinal lymph node.

REFERENCES

1. Mayock RL, Bertrand D, Morrison CE, et al: Manifestations of sarcoidosis. *Amer J Med* 1963; 35:67–89.

2. Yousem SA, Weiss LM, Colby TV: Primary pulmonary Hodgkin's disease: A clinicopathologic study of 15 cases. *Cancer* 1986; 57:1217–1224.

The Story of Lassa Fever

Part I: Discovering the Disease

JOHN D. FRAME, MD

The discovery of the Lassa virus was by serendipity but not fortuitous. It resulted from an attempt to investigate African arbovirus infections using missionaries as "sentinel animals."

HUNTING AFRICAN ARBOVIRUSES

In 1955, eight children of missionaries in Nigeria incurred high fevers and convulsions followed by permanent brain damage of various types. Although various diagnoses, including cerebral malaria, were made by physicians in the field, on their return to the United States I suspected that these individuals had been infected during the course of an unknown arbovirus outbreak in Nigeria. At that time, I was unable to find a laboratory to help elucidate the cause of the children's illnesses.

Ten years later, Dr Harold Brown invited me join the Division of Tropical Medicine at the Columbia University School of Public Health as an Adjunct. When I spoke of this specific research interest among others, Dr Brown introduced me to Dr Roger Williams, medical entomologist in the Division, recently returned from a sabbatical year at the Arbovirus Research Laboratory in Ibadan, Nigeria (Fig 1). Through him, I met Dr Wilbur Downs of the Yale Arbovirus Research Unit (YARU) and was invited to send sera for testing by Dr Jordi Casals who had recently been chosen to classify the arthropod born viruses (Fig 2) being found during the course of investigations throughout the world.

I then attempted to trace the brain-damaged children, to obtain serological specimens. In most instances, the severity of the children's condition had led their families to remain in the United States; in some instances the mission agency had lost track of them. Specimens were finally obtained from four of these children and from four children with similar histories who had become ill in other African countries but with less residual damage than the children in Nigeria. Dr. Casals urged me to obtain specimens from other missionaries, as well. Eventually, the specimens of 65 missionaries who had medical histories of fevers accompanied by serious illness and 100 missionaries light or no with fevers were tested by Dr Casals against a battery of 28 African arboviruses.

One-half the sera from missionaries with "significant" fevers showed antibodies to one or more of the virus antigens in Dr Casals' battery; only 12 of the controls did with a highly significant statistical difference between the two groups. Only four of the eight children with a history of febrile convulsions carried antibodies to the arbovirus antigens in the test battery. The evidence for a variety of infectious agents and the small number of children available for testing left the original problem unsolved.

When we reviewed the list of antigens evoking antibody responses, it became clear that in many instances the clinical characteristics of infections caused by these agents were not yet known. We wondered whether missionaries might not be a population in which arboviral diseases could be better defined. It appeared that enough missionaries with severe or unusual fevers had been infected with arboviruses that attempts to test such patients during their illnesses might result in the diagnosis of viral infections. This in turn might elucidate the syndromes related to specific viral infection.

THE FIRST OUTBREAK

In 1968, as we were completing the statistical observations, I spoke to Dr Jeanette Troup of the Sudan Interior Mission (SIM) in Nigeria. She was returning to be in charge of the Bingham Memorial Nursing Home for missionaries, located in Jos. I suggested that, when a missionary with fever came for treatment, she should obtain and freeze serum early and again two weeks or more afterward and send the specimens to me to be forwarded to Dr Casals. He would then test them for antibodies to his arbovirus antigen battery and also attempt isolation of the infecting agent.

Dr Troup first had occasion to try the plan in January, 1969, when a nurse, Miss Laura Wine of the Church of the Brethern mission in Lassa was brought to the nursing home. She was seriously ill with fever, sore throat, edema of the head and neck and bleeding from body orifices and needle puncture wounds; she died in 30 hours. Dr Troup obtained and froze serum when Miss Wine entered the hospital.

It was initially believed that the disease was likely a fulminant streptococcal infection overwhelming an aged person. Miss Wine, whose wish as a young nurse to enter mission service had been thwarted by the diagnosis of tuberculosis, had become a nursing educator in the Midwest. On her retirement she had decided to fulfill her first ambition and had been accepted as a missionary nurse. She had served for two years in good health until about a week before her hospitalization in Jos.

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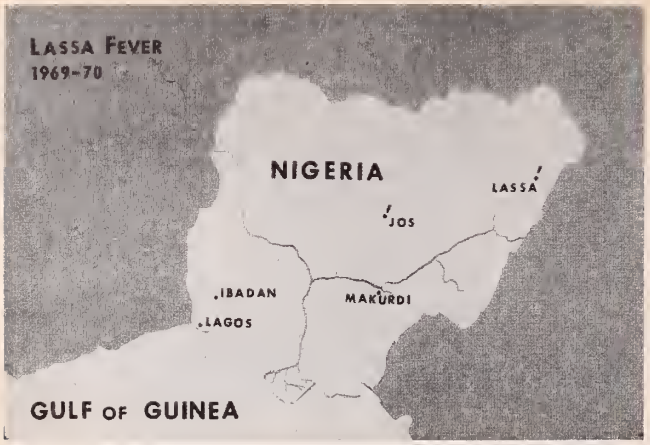

FIGURE 1. Map of Nigeria (John D. Frame, M.D.)

Miss Charlotte Shaw, the duty nurse when Miss Wine was admitted to the nursing home, administered "oral hygiene," that is, she swabbed out crusting sores on the patient's tongue and lips (Fig 3). At the time, she noted a stinging sensation on her finger and realized she had cut herself picking roses for another patient earlier that day. When she completed her initial care of Miss Wine, she treated her finger with antiseptic and a small dressing. Nine days later, she became ill with chills, fever, sore throat, leg and back pains, and eventually a petechial rash and edema; she recognized her symptoms to be those of Miss Wine's and that she had likely caught the infection from her. Miss Shaw died in another 11 days. Dr Troup obtained and froze specimens initially and again shortly before Miss Shaw's death. She also performed an autopsy with the help of the head nurse, Miss Lily Pinneo.

In seven days, Miss Pinneo, too, became ill (Fig 4). When it was evident that the course of her illness was similar to that of the others, Dr Troup determined to evacuate her by air to the United States for diagnosis and treatment. The first leg of her travel, with nurse Deloris Rohe as her companion, was to Lagos in Nigeria where she was detained in a pest-house for four days. With the help of Dr Lyle Conrad of the US Centers for Disease Control (CDC), arrangements were made for transportation home with isolation in the first class cabin. I met her on her arrival in New York City on March 4 and obtained blood for

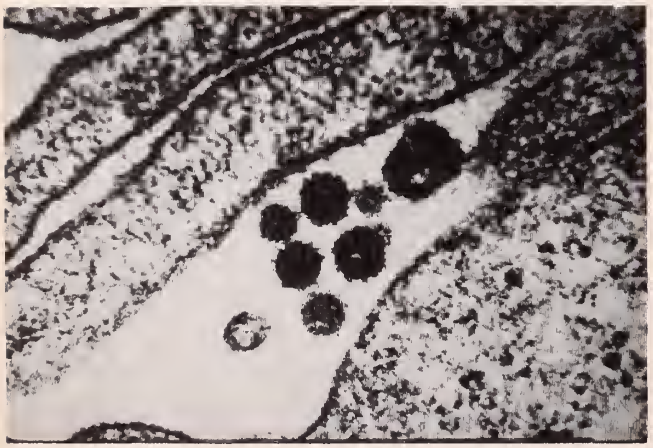

FIGURE 2. Slide of Lassa virus in Vero cell culture, from the Yale Arbovirus Research Unit (Courtesy of Yale University)

FIGURE 3. Charlotte Shaw, RN, who died of Lassa fever in 1969 (courtesy Penny Pineo).

FIGURE 4. Penny Pinneo, RN, the first known patient with Lassa fever treated in the United States (courtesy Penny Pinneo).

virological and serological study. Miss Rohe handed me sera from the other two cases, pathological specimens from the autopsy of Miss Shaw, and the clinical notes of all three patients prepared by Dr Troup. Miss Pinneo was then transported by ambulance to the Columbia Presbyterian Medical Center (CPMC).

Two days previously, a staff member at the SIM office in New York had called to read me excerpts from a brief account by a missionary in Jos regarding the series of cases. It appeared from her notes that we were dealing with a hitherto unknown illness or else that the three nurses had been victims of a bizarre series of coincidences. In any case, Dr John Baldwin, who was to be the attending physician, and I agreed that Miss Pinneo was to be treated in strictest isolation on the infectious disease service. The next morning was my usual day as Adjunct in the Division of Tropical Medicine; I had barely reached my office when I received a call from the resident:

"Dr Frame, what can you tell us about this patient Pinneo?"

"Didn't you get the clinical reports of the three patients, the papers that came with her?" I asked.

"Yes, we did. But, has anyone with this syndrome survived?"

"I'll be right over," I said, hearing panic in his voice.

I discussed the case and reviewed the infectious precautions being taken; I suggested that gloves be added to the cap, mask, and gown technique being followed. The house staff was clearly anxious, and I tried to reassure them regarding the efficacy of infectious precautions.

"It's true, we don't know what we're dealing with. But we must assume that our procedures will protect against the most dangerous of pathogens. What would you do if this were a case of pneumonic plague?"

"I'd die of fright!" was the answer.

"Assume, then, that this is a case of scarlet fever in a surgical ward, and follow what care you would take."

I must add that the nurses had more confidence in their technique than did the house staff, and their confidence was well placed. About 60 members of the staff and Miss Pinneo's sister, who were in contact with Miss Pinneo during her two months in the hospital, were subsequently found negative for Lassa antibodies by serological testing.

Miss Pinneo was critically ill for the next week. Meanwhile, Dr Downs stopped in on his way from his Rockefeller Foundation office in New York and carried specimens to Yale. Tests of numerous pathogens were performed under the direction of Dr David Gocke of the CPMC in the laboratories of the Medical Center, the New York City Department of Health Bureau of Laboratories and the CDC in Atlanta; all tests and cultures were negative. It was nine days before a call from Wilbur Downs informed us that Vero cell cultures by Dr Sonia Buckley showed a cytopathogenic effect. He noted that the changes might be due to some unknown toxin, not a viral agent, and that subcultures were to be performed. In another five days these, too, showed cytopathogenic effects. Cross-testing of the sera corroborated the identity of the agent in all specimens. After testing with over 250 viral antigens a weak reaction to that of lymphocytic choriomeningitis (LCM) was found, and eventually electron microscopy showed that the new agent and the LCM virus were morphologically alike. It was recognized, then, that the syndrome was similar to that of Bolivian hemorrhagic fever, which shares the same viral morphology.

Virus isolation might not have successful except for a fortuitous change in personnel at YARU. About two weeks earlier, Dr Buckley had found that the Vero cultures were no longer effective and suspected that the technician in charge had become careless. She promoted another person, I believe one who normally cleaned glassware, to the responsibility of caring for the cell cultures, and the Vero cells now grew viral agents as expected in time for the receipt of the Nigerian specimens.

While animal experiments, membrane filtration to determine particle size, and other experiments were being conducted at Yale, Miss Pinneo slowly recovered. Residual findings included nystagmus, mild ataxia, and hair loss. After two months in the CPMC, she returned in early May to her Rochester, NY, home for further care by her sister Rose, a nursing instructor.

Dr Jordi Casals Becomes Ill

In early June, Dr Casals felt unwell. Although he attempted to continue working, after five days he was admitted to the isolation ward of the CPMC, and general supportive care was given. His symptoms were similar to those early in the illnesses of the three nurses. Dr Downs called me to ask whether I could find Miss Pinneo with the hope she would donate plasma to treat Dr Casals (Fig 5). Plasma infusions had appeared to be efficacious for treatment of Bolivian hemorrhagic fever, and Dr Downs believed we should consider this treatment for Dr Casals. Miss Pinneo responded to our appeal at once, and by the next afternoon, a Monday, had returned to New York to donate two units of plasma.

On this day began a series of anxious discussions in the Department of Medicine at CPMC regarding the possible administration of Miss Pinneo's plasma. In the first place, the diagnosis had not been established and would not be until virus isolation had been carried out with results to be expected Friday. Furthermore, might Miss Pinneo be a carrier of hepatitis B virus (HBV), or even a chronic carrier

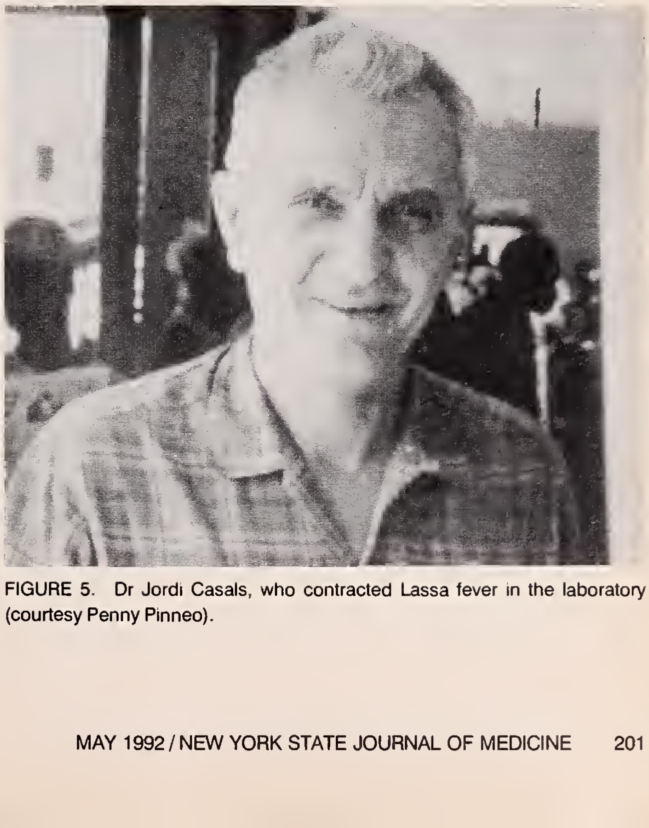

FIGURE 5. Dr Jordi Casals, who contracted Lassa fever in the laboratory (courtesy Penny Pinneo).

of this new virus? Dr Gocke had recently developed counterimmunoelectropheresis as a test for HBV; Miss Pinneo's blood was found non-reactive by this technique. However, it was already evident that the test did not discover all carriers of serum hepatitis; it was thought the test might not be sensitive enough to discover HBV antigen at all times. It was not yet known, of course, that there is at least one other agent, now designated hepatitis C virus, that is a cause of serum hepatitis.

Dr Downs called Dr Karl Johnson, the investigator of the Bolivian disease who had himself recovered from infection; Dr Johnson advised that in Bolivia, if one delayed plasma infusion until the patient was in dire straits, recovery was not likely. Finally, as Dr Casals' status was slowly deteriorating the convalescent plasma was administered Wednesday afternoon.

In 24 hours, Dr Casals became afebrile. Convalescence was slow but steady, marred only by the isolation of virus from his urine, with the test completed only after he had left the hospital.[1]

Naming the Disease

What to call the new disease and its virus? In keeping with the practice of arbovirology at the time, I suggested that a geographic name be assigned and proposed Lassa after the Nigerian community in which the first case had presumably been infected. I initially called the infection "Lassa disease," thinking that what we had seen in our patients might be the extreme of a spectrum of syndromes caused by the new virus. David Gocke believed "Lassa fever" to be more appropriate, and so it became.[2]

When Lassa virus had been identified, the SIM in the United States and Dr Troup in Nigeria were informed. At the time, I asked her to notify the Nigerian health authorities of the finding; I could imagine their chagrin if they should hear of the discovery of the new agent in a roundabout way. It was important, I felt, that we not be guilty of a form of "scientific imperialism," carrying out our investigations with no regard for the public health implications in Nigeria. As a matter of fact, eventually many Nigerians were offended by the name given the agent and illness, suggesting that a small town in their nation had been unfairly stigmatized by what early became the paradigm of a dangerous viral infection.

An Unexplained Case

It was in the early autumn of 1969 that Dr Casals was fit enough to assume his responsibilities. They included participation in various conferences postponed because of his illness; it was not until November that he was able to return to his laboratory. Before his illness, he had inoculated a number of mice intracerebrally, and they had been stored in the freezer; he removed them, prepared some brain specimens in sealed tubes for further work with live virus, and inactivated the others. At this time, an accident occured that could not be explained satisfactorily. A laboratory technician, Juan Roman, began to feel unwell just before Thanksgiving. He and his family went for the holiday to visit relatives in the Chester, Pennsylvania area. On the day after Thanksgiving, he entered a local hospital; the laboratory at Yale was not informed as to his status. After a week, when his condition was deteriorating, his physician called YARU questioning whether he might have been infected with an agent there. Dr Casals immediately visited him in Pennsylvania, felt that he most likely had Lassa fever, drew blood for testing, and returned to Yale to donate plasma. The patient had been ill long enough that plasma was to be administered without waiting for the five days needed to complete virus isolation. However, Roman died before plasma could be given him; the diagnosis was in fact confirmed three days after his death.

Public Reaction

Although knowledge of this new viral agent was spread widely in the virological community, the first public articles were read in January 1970 at a meeting of the New York Society of Tropical Medicine. An account of the new virus, prominent on the front page of the *New York Times*, excited a great deal of interest,[3] and was followed by articles in weekly news magazines and other publications.[4] News that after Roman's death YARU had decided to conduct no more experiments with live Lassa virus gained the agent the sobriquet, "the virus too hot to handle." The fearful reputation of Lassa fever was firmly established in the public mind by coincidental news of another, larger outbreak of Lassa fever in Nigeria. The mission had received cables reporting first that Dr. Troup was ill and then that she, too, had died of the new disease under circumstances that were not yet clear.

It was only during investigations that followed the second nosocomial outbreak that it became apparent that Lassa fever was not new, merely newly discovered, and that it was a significant cause of fever in Africa, previously overlooked by the proclivity of health workers to classify disease in terms of the known rather than the unknown.

References

1. Leifer E, Gocke DJ, Bourne H: Lassa fever, a new virus disease of man from West Africa. II. Report of a laboratory acquired infection treated with plasma from a person recently recovered from the disease. *Am J Trop Med Hyg* 1970; 19:677–679.
2. Frame JD, Baldwin JM, Jr, Gocke DJ, et al: Lassa fever, a new virus disease of man from West Africa. I. Clinical description and pathological findings. *Am J Trop Med Hyg* 1970; 19:670–676.
3. Altman LK: New fever virus so deadly that research halts. *NY Times* February 10, 1970, pp 1, 25.
4. The killer from Lassa. *Time* February 23, 1970, pp 42, 43.

A history of the Women's Medical Society of New York State

JEAN M. HOEFER, MS, MA; LESLIE J. KOHMAN, MD

The Women's Medical Society of New York State (WMS) is the only statewide organization of women physicians ever organized in New York. It was founded in 1907,[1] eight years before the Medical Women's National Association (WMNA) was started. WMS joined the national organization in 1952[2] when it became Branch 18 of the renamed American Medical Women's Association (AMWA).

The organization still maintains its historic identity as WMS since it continues to play its role in the advancement of women in medicine. This paper reviews the founding and history of WMS.

LEGACY OF SARAH ADAMSON DOLLEY

WMS was founded on March 11, 1907, when women physicians from across New York State gathered at the Genesee Valley Club on East Avenue in Rochester to honor Dr Sarah Dolley, one of the first women ever to practice medicine in the United States (Figs 1,2). As a young woman named Sarah Read Adamson, Dr Dolley graduated from the Central Medical College of Syracuse and Rochester in 1851, only three years after the first woman physician in the United States, Elizabeth Blackwell, graduated from the medical college in nearby Geneva. While still a school girl, Sarah had developed a burning interest in physiology, and by the age of 16 had memorized most of the anatomical terms by reading her physician uncle's anatomy book.

When she was 18 years old, Sarah's uncle Hiram Corson started her medical training as his assistant in his practice in Plymouth Meeting, Pennsylvania. After that apprenticeship, she applied for admission to medical colleges, finally being accepted at the coeducational Central Medical College of Syracuse, NY, in 1849,[3,4] the year it was founded. Central Medical College was run by adherents of the Eclectic branch of medicine, which treated diseases by the application of single remedies to known pathologic conditions. Diseases were not classified and special attention was given to developing indigenous plant remedies. Central Medical College was the first of several eclectic medical schools that accepted women students before 1860. Sarah came to Syracuse escorted by another physician uncle who helped her get settled in the city. Early in 1851, Central Medical College moved to Rochester and Sarah graduated there in February.[3]

Sarah was one of the three women to earn the MD in the early years of the school.[3] She spent the year following graduation at Blockley Hospital in Philadelphia and was probably the first women to intern in an American hospital.[4] She finished her internship in June 1852 and returned to Rochester to marry her surgery professor, Dr Lester C. Dolley. The couple eventually settled in Rochester to practice medicine in their home, at 52 East Avenue. Dr Sarah Dolley took postgraduate training in Paris during the winter 1869–1870. After Lester Dolley died in 1872, Sarah Dolley continued to practice in partnership with another women physician, Anna Searing. Sarah Dolley was in demand as a lecturer and teacher, and she extended her professional training by visiting medical clinics in Europe during spring and summer 1875.

In the Quaker tradition, Dr Sarah Dolley was a modest, self-effacing woman with great strength of purpose. In her quiet way, she served as a role model for many women physicians, enhancing their professional status even as she labored to improve the health of the Rochester community. She persuaded the city to let her use a building they owned on Front Street to open a dispensary where poor families could come for free medical care. Her Provident Dispensary was the first dispensary in Rochester. It was supported by $100 a year from the city, and after 10 years it became the outpatient department of the Rochester City Hospital. Dr Dolley was also the moving spirit behind the formation of the Monroe County Red Cross. When the state opened asylums to care for mentally handicapped and insane patients, Dr Dolley worked for the appointment of women physicians to care for women patients. She was instrumental in having women physicians included in city and county lists of medical practitioners. Women physicians in the dispensary formed the Practitioners' Society (Fig 3), which became an important professional organization for women doctors in the Rochester area.[4,5,6]

In 1906, the Practitioners' Society changed its name to the Blackwell Medical Society of Rochester. In her history of the society,[7] Ellen More, PHD, analyzed the social and professional role of women's medical societies around the turn of the century. She concluded that "... women's medical societies were not intended simply to satisfy a hunger for professional success." These women had grown up in the tradition of Victorian social feminism, which "under the banner of female moral superiority, sought to bring about a wide variety of social reforms...." Drs Blackwell and Dolley managed to carry on distinguished medical careers within the context of social feminism, but Sarah Dolley and her female colleagues felt a need for

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Funded by the Women's Medical Society of New York State (American Medical Women's Association Branch 18).

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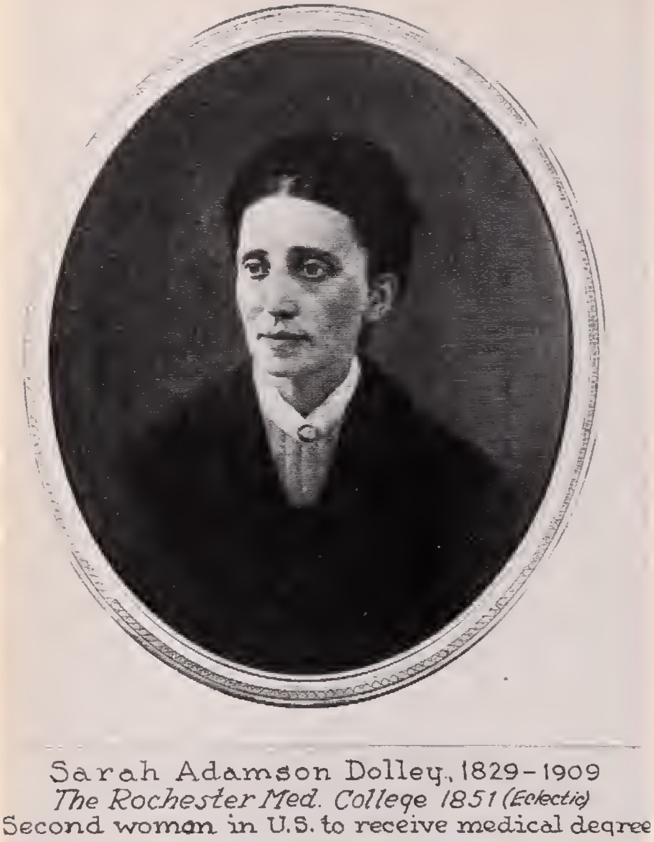

Sarah Adamson Dolley, 1829-1909
The Rochester Med. College 1851 (Eclectic)
Second woman in U.S. to receive medical degree

FIGURE 1. Sarah Adamson Dolley (Courtesy of the Edward G. Miner Library, University of Rochester Medical Center, Rochester, NY).

FIGURE 2. Sarah Dolley in later years (Courtesy of the Edward G. Miner Library, University of Rochester Medical Center, Rochester, NY).

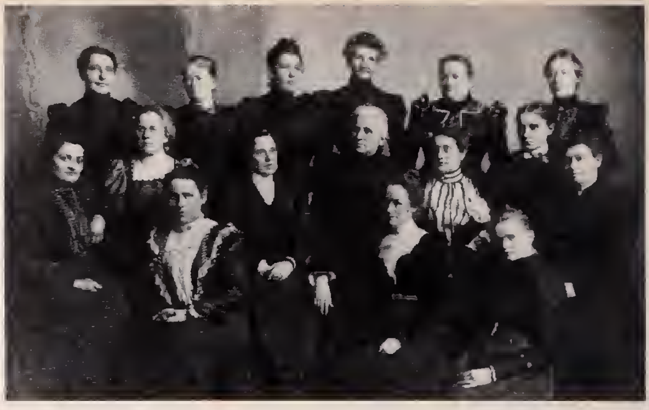


FIGURE 3. The Practitioners' Society of Rochester, NY, forerunner of the Elizabeth Blackwell Society. The date of this photograph is not known, but it was taken between 1887 and 1912. Dr Marion Craig Potter (front row, second from the left) is present, but Dr Dolley is not in this photograph (Courtesy of the Edward G. Minor Library, University of Rochester Medical Center, Rochester, NY).

more organized work to strengthen the precarious professional position of women physicians.

On a winter day in 1907, 45 women physicians gathered at banquet tables in the Genesee Valley Club to celebrate Sarah Adamson Dolley's 78th birthday. However, they also had a long-range objective for their meeting. As representatives of four regional women's medical societies from different areas of New York, they came together in Rochester to organize a statewide Women's Medical Society. For this purpose, The Blackwell Medical Society of Rochester, The Physicians' League of Buffalo, The Women's Medical Association of New York City, and The Dr Cordelia A. Greene Society of Castile together invited all "the Women Physicians of the State to join them at a banquet." Sarah R. Adamson Dolley was unanimously elected the first president of WMS. Names of the officers elected in 1907 are listed in Table I.[1]

The women physicians modeled their organization after the Medical Society of the State of New York (MSSNY) and elected a councilor from each MSSNY geographic district. The councilor's job was to organize a branch of WMS in each district, inform women physicians in every county medical society of branch meetings, and encourage new members to join WMS. The councilors elected in 1907 are listed in Table II.[1]

TABLE I. Officers of the Women's Medical Society of New York State 1907

Honorary President, Elizabeth Blackwell, MD, Hastings, England
President, Sarah R. Adamson Dolley, MD, Rochester
1st Vice President, Electra B. Whipple, MD, Buffalo
2nd Vice President, Mary H. Cotton, MD, New York City
3rd Vice President, Mary Theresa Greene, MD, Castile
Secretary, Evelyn P. Ballintine, MD, Rochester
Treasurer, M. May Allen, MD, Rochester
Toastmistress, Ida C. Bender, MD, Buffalo
Nominations, Helen Kuhlman, MD, Buffalo
Honorary Members: Emily Blackwell, MD
  Hannah T. Croasdale, MD
  Ann Broomall, MD
  Mary Putnam Jacobi, MD
  Elizabeth M. Cushier, MD
  Clara A. Swain, MD
  Sarah J. McNutt, MD
  Gertrude B. Kelly, MD
  Annie S. Daniel, MD
  Helen L. Baldwin, MD
  Anna L. Williams, MD

See reference 1 for publishing information.

TABLE II. District Councilors Women's Medical Society of New York State 1907

First District Branch	Martha Wollstein, MD, New York City
Second District Branch	Anna Craig, MD, Kings Park
Third District Branch	Hortense V. Bruce, MD, Hudson
Fourth District Branch	Caroline S. Pease, MD, Ogdensburg
Fifth District Branch	Angeline Martine, MD, Utica
Sixth District Branch	Anna Bowden White, MD, Norwich
Seventh District Branch	Lettie H. Woodruff, MD, Rochester
Eighth District Branch	Maude J. Freye, MD, Buffalo

See reference 1 for publishing information.

There was a remarkable feeling of solidarity among women physicians in the society at this time. A contemporary newspaper account described the affection and esteem these women physicians expressed for Dr Dolley, their mentor and friend. Headlined "OPENED WAY FOR WOMEN DOCTORS," the story said Dr Dolley was eulogized by her associates for encouraging them in high ideals. In a tribute entitled "Reminiscences," Lettie H. Woodruff, MD, of Rochester spoke of Dr Dolley's "... calmness of spirit, sureness of purpose, sweetness of disposition and presence that seemed to radiate these qualities and inspire them in others. The great courage and usefulness of your life will always be held by us in loving remembrance," Dr Woodruff said.[6]

That evening Dr Dolley did not feel well enough to stand during the whole reading of her inaugural address, so First Vice-President Electra B. Whipple, who sat next to her, picked up the manuscript and finished reading it aloud to the banquet guests.[8] Dr Dolley spoke of her experiences as a women physician and the changes in public attitudes she had noticed during her lifetime. She said: "The women physician has passed that point where everyone she met regarded her in a manner that implied that the great 'head and front of her offending' had been an act of impertinence." She went on to advocate real rather than superficial virtues: generosity, tenderness, sympathy, and most importantly honesty "... in an age that produces splendid falsities."[6]

For its official seal the society adopted a coat of arms from a branch of Sarah's mother's family, the Dickinsons. On this coat of arms a gold cross divides a green shield into four spaces; each space displays a golden hind's head; and the banner beneath bears the Latin motto *Esse Quam Videri* [To Be Rather Than Pretend, or, To Be Rather Than to Seem]. Geneologists trace the name back to 13th century England and speculates that the hinds' heads symbolized a responsibility of the Dickinson ancestors for guarding the king's game preserves. The cross probably means that a family member went on a crusade (Fig 4).[9]

The reverence WMS members had for Dr Dolley is apparent in a booklet, probably published in the 1930s, that contains the constitution, bylaws, 1907 list of officers, and a portrait of their first president and founder. The booklet stated:

> In Western New York, Dr Dolley, as a successful physician, by her dignity and scientific attainments, has made it easier for all women. In her quiet way, she has been an unseen force constantly on the alert to open positions, to effect organization, and to mobilize the forces that act for the helpfulness of the individual and the success of the whole.[1(p2)]

In the new state-wide organization, the four founding societies kept their separate, regional identities while supporting the broader organization for the benefit of all women physicians in the state. At the same time, they were quick to affirm their position in the medical mainstream. Their booklet stated:

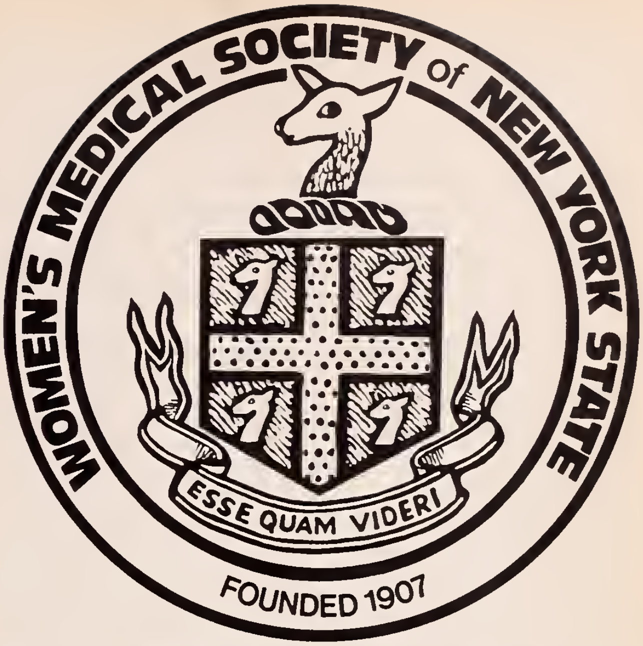

WOMEN'S MEDICAL SOCIETY of NEW YORK STATE
ESSE QUAM VIDERI
FOUNDED 1907

FIGURE 4. The official seal of the Women's Medical Society of New York State.

> This Society is organized with the distinct understanding that it is not to divert interest from the Medical Society of the State of New York, but rather to encourage greater activity in organized work, and to cultivate social relations and mutual helpfulness among women physicians.[1(p3)]

To ensure the professional competence of its membership, the WMS constitution required members of the society to be "women graduates in medicine in good professional standing and who are members of a County Medical Society."[1(p5)] Dues were one dollar a year. Graduates of "recognized Regular Medical colleges" who did not belong to a medical society could be associate, non-voting members after paying a two-dollar fee. Honorary memberships without dues, fees, or voting privileges could be voted for distinguished women physicians and were usually extended to retiring members or those who moved out of state.[1(p6)]

Although Elizabeth Blackwell was retired and living in England in 1907, the founders of WMS identified with her pioneering spirit, and endorsed her philosophy of medicine by printing the following quotation in their booklet:

> Medicine is so broad a field, so closely interwoven with general interests and yet so personal a character in its individual applications, that the cooperation of men and women is needed to fulfill all its requirements.
>
> Elizabeth Blackwell[1(p4)]

Fostering that cooperation while at the same time maintaining a separate organization for women physicians proved to be a difficult balancing act during the subsequent history of WMS.

EARLY YEARS OF THE WOMEN'S MEDICAL SOCIETY

Bylaws of the WMS constitution required annual meetings to be held on Dr Dolley's birthday during her lifetime.[1(p8)] The second annual meeting was held on March 11, 1908, and at the Genesee Valley Club in Rochester. At that meeting, Dr Dolley gave credit to Electra Whipple, who had died in the past year, for the idea of a statewide organization. Drs Whipple and Marion Craig Potter "worked nobly and unceasingly to perfect this organization," she said (Fig 5).[8(p7)] In this same speech, Dr Dolley warned women medical students not to be misled by the relative ease of their admission to medical schools, because the woman physician "has other difficulties to meet."[8(p6)] She counseled them to face continuing opposition and their own limitations with open eyes. She urged her colleagues to be patient with society's natural reluctance to accept innovation. She said, "Our movement, like every other grand one, is not so much endangered by secret antagonism or open opposition, as by a weak compliance with unworthy conventions or conditions among ourselves."[8(p10)] She went on to warn against "that affectation that would imitate the worst failings of men or ignore the highest powers of women," and she invoked, "a spirit of consecration [in which] we will go on from strength to strength, ever animated by *l'esprit scientifique* and *l'esprit morale*."[8(p15)] Sarah Dolley died in December, 1909, and was buried in Mt Hope cemetery,[5] across the street from what is now University of Rochester Medical Center.

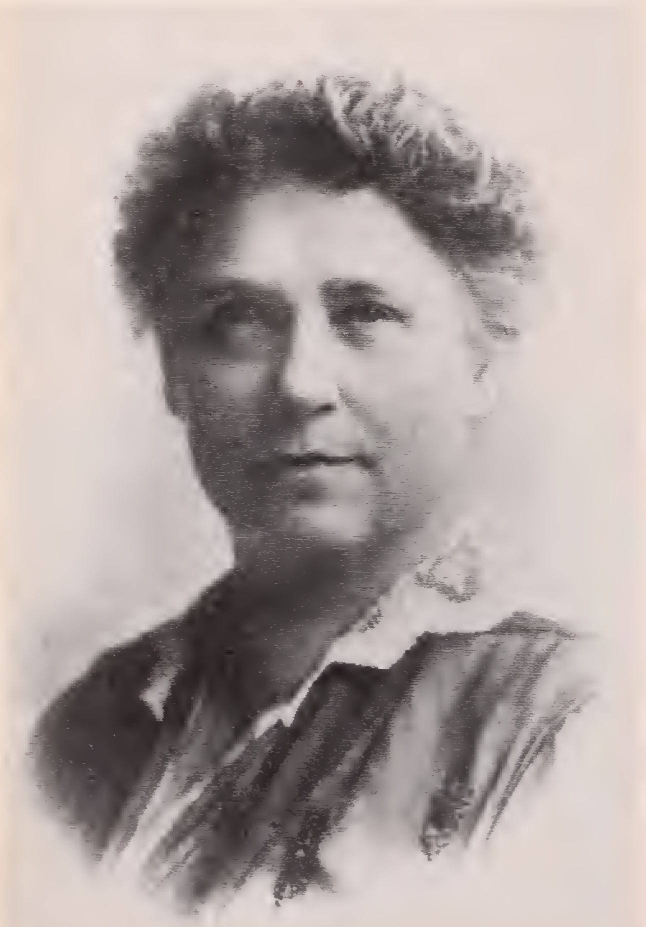


FIGURE 5. Marion Craig Potter (Courtesy of the Edward G. Miner Library, University of Rochester Medical Center, Rochester, NY).

In 1908, WMS was already following *les esprits* mentioned by Sarah Dolley. The Public Health Committee reported to the annual meeting about government efforts to distribute clean milk for babies during warm weather, anti-tuberculosis work, and physical examinations for school children. A speaker discussed the difficulty of teaching physiology and hygiene to students who were "easily offended" by discussions of generative organs "unless the subject was treated in just the right way," avoiding use of the word "sexual." The legislative committee urged their support of legislation to consign alcoholic women and prostitutes arrested for repeated offenses to a state farm because they were "physically diseased and as morally irresponsible as the insane or feeble minded." They believed that physical cure and moral reform should be the objective in dealing with this "idle, dissolute, and dangerous class."[10]

WMS continued to hold its annual meetings on the anniversary of Dr Dolley's birthday. Between annual meetings, the Council, an executive body consisting of the officers and Chairman (sic) of standing committees, met to deal with financial and other business matters. In October, 1910, at a session to plan the 1991 annual meeting, Council members considered scheduling the annual meeting immediately before or after the MSSNY meeting in Albany. After some discussion they decided against it, since Albany hotel accommodations were limited, and the women present at the Council meeting thought it might be undesirable to call attention to the separate WMS.[11] Accordingly, the fifth annual meeting was held on Friday, March 10, 1911 at the New York Academy of Medicine, where members affirmed the Council decision not to schedule their meetings in conjunction with the state medical society gatherings (Fig 6).[12]

This policy was destined to change a year later. In 1913, when MSSNY began its 107th annual meeting on Tuesday, April 29, in Rochester, host Dr Wesley Mulligan officially welcomed WMS along with the rest of the participants. WMS scheduled its seventh annual meeting on April 28 and MSSNY opened its exhibits at Convention Hall a day early "in order that women physicians could visit exhibits and register" for MSSNY convention that officially opened the next day.

A WMS annual meeting was an all-day event. In 1913, for example, the Seventh Annual Meeting[13,14] was called to

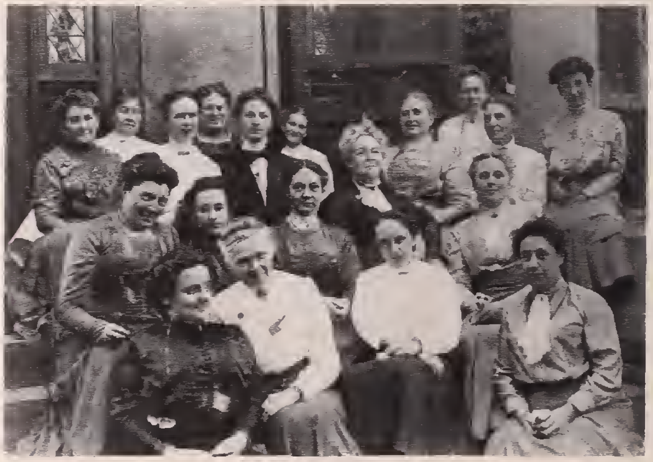


FIGURE 6. Women's Medical Society meeting, September 14, 1911, Castile, NY (Courtesy of the Department of Manuscripts and University Archives, Cornell University Libraries, Cornell University, Ithaca, NY).

order at 10 AM by President Helene J.C. Kuhlman, MD, of Buffalo. After the invocation and welcome, Dr Kuhlman addressed the meeting on the subject, "the Personal Side of Medicine," in which she called for more homes for "aftercare" and day camps for sick mothers. This was followed by a reading of the minutes, committee reports, and discussions of old and new business. The Scientific Program, with an hour break for lunch, consisted of the following papers and discussions: A Plea for Early Recognition and Correction of Faulty Postures; Eugenics in Relation to Sex Hygiene; Report of Case, Meningitis, due to Staphylococcus Infection; Some Advantages in the Caloric Method in Feeding Young Children; Methods of Anaesthesia in Vogue at the Present Time; Further Observations on the Etiology of Incipient Exophthalmic Goiter; The Care of the Normal Case During Pregnancy, Labor, and the Lying in.

After the Scientific Program, the new officers for the coming year were elected, and the meeting adjourned at 4 PM so the members could attend the main lecture, which was open to the public.

The years 1909 to 1913 were the heyday of the public health education movement that was spearheaded by women physicians in the American Medical Association (AMA). Sarah Adamson Dolley, MD, had been named Honorary Chairman of the AMA Committee for Public Education Among Women when it was organized in 1909, with Rosalie Slaughter Morton, MD, of New York City as Chairman. According to an undated pamphlet in the society records, the committee's purpose was to organize public lectures "to disseminate accurate information concerning the nature and prevention of disease and the general hygienic welfare of the people." In three years, the committee reported, it had arranged lectures that were presented before audiences totaling at least 600,000 men and women and a large number of school children "for the purpose of education in the prevention of disease."[15]

At the Rochester meeting, Dr Rosalie Slaughter Morton gave the main afternoon lecture, "Women in Industry," in which she discussed conditions that affected the health of women workers. An interesting sidelight on her lecture is the difference between the report of Marion Craig Potter, MD, who wrote up the minutes of the meeting, and the newspaper story covering the event.

According to Marion Craig Potter, Dr Morton "contended that from experiments [we know that] no work need to be too hard for a woman if she is properly trained to do it, under proper hygienic conditions and with proper rest."[14] A newspaper account of the same lecture reported that Dr Morton said women should not stand for long periods because it would "strain vital parts of their delicate bodies." (Unidentified newspaper clipping, April 28, 1913, "Marion Craig Potter Papers," Record/scrapbook) A subsequent issue of the *Medical Women's Journal* repeated the statement. Beside these clippings pasted in the record/scrapbook are handwritten comments repeating Dr Potter's interpretation of the Morton speech.

After the speech, the program listed the last event scheduled before the evening banquet: an automobile ride.

The evening banquet[16] featured caviar and various dishes rich with French sauces and creams. Each diner paid $2.50, a fairly sumptuous amount compared with the Social Worker's dinner many of them attended the next evening, which cost 75 cents a plate).[14] Entertainment after the banquet consisted of a series of toasts led by Toastmistress Grace Peckham Murray. The first one, "Oh Woman! What of thy hours of ease?" was given by Dr Kuhlman with the following quotation attributed to the 17th century British playwright Thomas Otway:[16]

> O, Woman! lovely woman. Nature made thee
> to temper man; we had been brutes without you;
> There is in you all that we believe of Heaven,—
> Amazing brightness, purity and truth, eternal joy and everlasting love.[16]

In like vein, they continued with toasts presented by six women: Oh Woman! What a ministering angel thou! O, Woman! What of the Past? O, Woman! What of Mere Man, God Bless Him? O, Woman! What of the Household? O, Woman! What of thy Country? O, Woman! What of thy Chosen Profession, Medicine?[16]

According to Rochester newspaper coverage, the WMS annual meeting was an important adjunct of the State Medical Society's activities that week. Having overcome their diffidence about meeting in association with MSSNY, the women physicians continued to hold their annual meetings on the day preceding the annual meetings of MSSNY. Attendance at annual meetings varied from below 50 to more than 100, depending on the size of the city where the meetings were being held. Usually the society tried to reach all women physicians in the state with notices of the meeting. Occasionally, they sent copies of the program to men physicians and sometimes their program notice appeared in the *New York State Journal of Medicine.*[17,18,19] *The Medical Women's Journal* usually published their notices, reported their meetings, and often printed their speeches and scientific papers in entirety.

WMS meetings followed a consistent pattern for many years. Planning and running annual meetings was the work of the officers and the chairman of four standing committees: Scientific Work, Legislation, Public Health, and Arrangements. The by-laws spelled out the order of business beginning with call to order and ending with the banquet. Although "Reading and discussion of papers" was seventh on the agenda, it soon became the biggest item of the meetings. At their March 11, 1910, meeting 15 papers were read in one day in addition to the business meeting with its committee reports and the president's address that opened the afternoon session.[20] The following October, the Council members voted to limit papers to a maximum of ten, with the suggestion that three papers in the morning and four in the afternoon would be optimal.[11] Heather Munro Prescott studied the issues covered in WMS meetings and concluded that they focused more on public health and social hygiene topics than did MSSNY, including more attention to adolescent girls, gynecology, pediatrics, obstetrics, and topics in general medicine, surgery, and psychiatry related to women's and children's health. (Personal Communication H. Munro, Department of History, Cornell University, 1987)

The WMS council of officers met before and after each annual meeting and at other times during the year when it was considered necessary. Getting a quorum at council meetings proved so difficult that in 1922 the membership voted to give past presidents voting privileges since they were already honorary councilors.[21] The annual meetings continued to be well-attended with good participation in both scientific and social programs.

MEDICAL EDUCATION

When Sarah Dolley spoke of "our movement,"[8(p10)] there was no doubt that WMS members considered the status of women in medicine an important concern of the organization. Although the WMS constitution before 1964 excluded physicians who were not members of county medical societies, they encouraged medical graduates to become associate members and they invited women students at local medical schools to attend their meetings and banquets as guests. In 1917, they began to give money as well as moral support to medical education for women. They voted donations from their treasury for Women's Medical College of Pennsylvania[22] and Drs Mosher, Potter, and Greene started the Medical Education Committee to raise and distribute funds for student scholarships.

A New York teacher named Alta Sager and a Korean woman, Miss Yue, were the first medical students helped by WMS,[23] which contributed $800 a year to the American and $150 to the Korean for tuition. Giving by the membership was casual, and for several years the committee reported deficits of $300 to $400 that were covered by personal donations from Dr Greene or Dr Potter, who were then reimbursed by collections at annual meetings.[24,25] After Alta Sager graduated and became the first woman to intern at the Buffalo City Hospital,[26] the committee stopped offering scholarships to New Yorkers but continued to help students in the Far East missionary colleges (Fig 7).

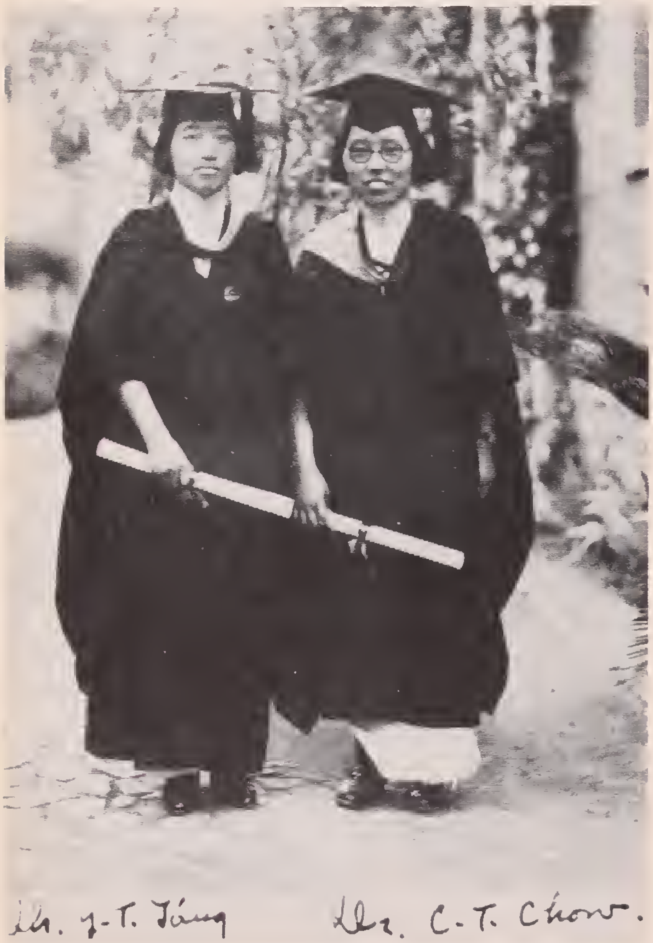


FIGURE 7. Two Chinese medical graduates supported by WMS scholarships, 1935 (Courtesy of the Department of Manuscripts and University Archives, Cornell University Libraries, Cornell University, Ithaca, NY).

For many years Mary Theresa Greene, MD, of Castile, NY, chaired the Committee on Medical Education. Castile is a small town about 80 miles south of Rochester, where in 1849 a farmer named Jabez Greene founded a water cure, a popular type of health facility at that time. Jabez's daughter, Cordelia, became interested in medicine and graduated from Cleveland Medical College in 1856.[27] Cordelia took over her father's sanitarium after his death, making it into a caring and curing place with a large female clientele. In 1905, Cordelia's niece Dr Mary T. Greene succeeded her. Mary T. Greene was an erect, solemn woman who for 50 years managed the sanitarium at Castile.[28] Every year Dr. Greene sent out fund-raising letters to women physicians around the state. WMS furnished the stationery for these mailings, and Dr Greene donated the cost of labor and postage (Fig 8).

Dr Rosetta Sherwood Hall was an early member of WMS although she lived in Korea. An 1889 graduate of Women's Medical College of Pennsylvania, Dr Hall joined the Foreign Missionary Society soon after finishing her internship at Children's Hospital, Staten Island. She opened the Baldwin Dispensary in Seoul in 1892. She practiced for 45 years in the Far East during which time she established four hospitals for women in Korea, pioneered in the education of the blind and deaf, and in 1928 founded the Women's Medical College in Korea. The Korean government presented to her a silver cup and citation for Act of Merit.[29]

For several years, Dr Greene's education committee gave $25 a year to as many Korean women students as they could. The need was very great. Dr Greene reported that the girls at medical school were given rice by their friends. Later, WMS contributions were also sent to Cheeloo Medical School in Shantung, China (Fig 9). Dr Greene's annual fund raising letters went out in the fall and sometimes included society news or announcements. She kept on sending her letters to New York State women physicians

FIGURE 8. A photograph of Chinese physician Lan-huo Lui Yui sent by herself with thanks to Dr Mary T Greene, 1937 (Courtesy of the Department of Manuscripts and University Archives, Cornell University Libraries, Cornell University, Ithaca, NY).

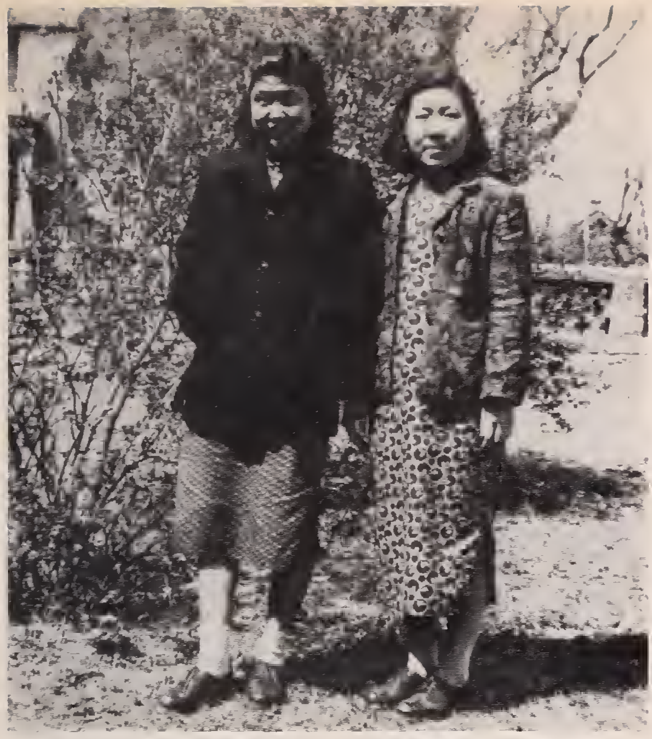
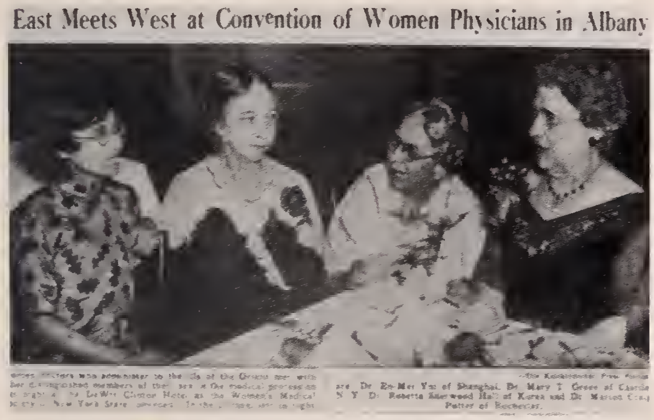


FIGURE 9. Chinese medical students Chang Hsiao Chung, (left) and Li Kwai Ling (right), Cheeloo University, April, 1948 (Courtesy of the Department of Manuscripts and University Archives, Cornell University Libraries, Cornell University, Ithaca, NY).

in spite of the difficulty of keeping an up-to-date list of their names and addresses. In 1947, for example, she sent out 1,000 letters and 300 were returned undelivered. Twenty-four women responded with donations for China. Every year, Dr Greene reported that she collected and sent on $100, $200, or $250 to Chinese students. Whenever $50 accumulated in the committee fund, she sent it along.[30,31] After China expelled foreign missionaries, WMS began a long-standing relationship with the Christian Medical College in Vellore, South India. Letters of thanks from some of the women who received these scholarships, including their photographs and pictures of their schools, are preserved in WMS archives, along with newspaper clippings describing visits and homecomings by the missionary teachers (Fig 10).



East Meets West at Convention of Women Physicians in Albany

FIGURE 10. Newspaper clipping with Drs Greene, Hall, and Potter with a beneficiary of their scholarship activities. Exact year unknown (Courtesy of the Department of Manuscripts and University Archives, Cornell University Libraries, Cornell University, Ithaca, NY).

A scholarship of $100 or $200 a year went a long way at the missionary colleges in Korea, China, and India but was not of significant help with medical tuition in the United States. WMS did not offer financial help to New York students again until the early 1960s when they were able to use the interest from a $15,000 bequest left to them for that purpose by Mathilda K. Wallin, MD. Dr Wallin, an 1893 graduate of the Women's Medical College of the New York Infirmary, had lived in New York City.[32] When enough interest had accumulated, WMS offered $500 loans at three percent interest to New York State women students in medical school. Committee members interviewed applicants and awarded the loans, to be repaid starting three years after graduation.[33]

Not all recipients paid back their loans, but enough money was repaid to keep the fund growing. Dr T. Greene died in 1957, leaving a bequest of $5,000 for the medical education fund.[34] After Dr Greene's death, the Education Fund Committee was chaired by Harriet Hosmer, MD, until the late 1960s, followed by Dr Ruth Knoblock, Dr Jean Smith, and Dr Adelaide Scanlon. One or two loans of $500 each were granted every year, usually to the same woman as she progressed through her medical training. There were few applicants for loans, but the need for money in Vellore was always great. Some beautifully written letters in the archives express the thanks of a number of students to the WMS committee.

In 1970, the annual meeting voted to send $395 to Vellore and, since there were no loan applications, to give the interest from the education fund to AMWA for scholarships.[35]

A $2,000 bequest from the family of Dr Lois Plummer of Buffalo, left to WMS for the "Mary T. Greene Fund," was also sent to Vellore. Dr Plummer had been president of WMS in 1960 and 1961. The annual meeting in February 1972 was told that a plaque in memory of Dr Lois Plummer was to be placed in a room at Vellore Medical College, India. An undated snapshot in the archives shows a plaque in memory of Dr Plummer mounted on a wall above a pair of wash basins.[36]

At the interim meeting in June of 1970 (they stopped calling them council meetings before 1940), WMS members decided to award loans using AMWA application forms. In 1971, federal spending cuts decreased funds available to medical students, and WMS had an unprecendented 14 applicants, many of whom Dr Smith described as desperate for money to continue their education. To meet the crisis the scholarship committee began to liquidate reserve funds and granted 10 loans of $500 each. "Unless we have some substantial contributions or legacies, we will be completing our work in another year or two," Dr Smith wrote.[37] By 1975, medical students were finding other sources of aid. The committee reported that few applications were being received, and that applicants "have more money than the fund has." Repayments and contributions sustained the fund, and in 1978 the members voted to resume contributions to Vellore and to increase the amount of loans to $1,000 each. The year 1980 brought another crunch for student funds and WMS responded by granting loans to eleven students. Poor repayment rates prompted them to authorize the scholarship committee to send out bills by registered mail. In 1987, members voted to distribute the money in the form of grants rather than loans. By 1988, the fund was down to about $4,000, which would have seemed like riches to Dr Greene in the days when medical tuition was $800 a year!

Military Commissions for Women Doctors

At certain times in their history, legislative issues stirred WMS members into action. They held most issues in common with the broad medical establishment, but on matters touching on the status of women physicians, they were likely to take stronger action. The status of women physicians in the armed services was one of these.

WMS annual meeting held on May 3, 1943, included a celebration of victory in the long war for equal status of women and men physicians in the armed forces. A new law requiring the Army and Navy to commission women physicians as officers had recently been signed by President Roosevelt. Passage of this legislation is generally considered a personal triumph for Emily Dunning Barringer, MD, who organized AMWA's publicity and lobbying campaign for the law. A 1902 graduate of Cornell Medical College, Dr Barringer was the first woman to be appointed to the staff of a city hospital, Governour Hospital in New York, where she worked during 1903 and 1904. Her book *Bowery to Bellevue* tells a good story of those years, with considerable evidence of her fighting spirit.[38] She began to fight for commissions for women physicians in 1917 when she was president of WMS. A national effort to have women physicians commissioned in the Army and Navy during World War I was blocked by the surgeon general who refused to consider the idea. Women physicians were allowed to attend patients in the armed forces as contract surgeons, a status that left them with considerably less authority in military facilities than male physicians who were commissioned officers. This policy was still in force in 1939, when World War II started in Europe.

Even before the United States entered the war, AMWA had begun to work for commissions for women physicians. As the nation began to build up its armed forces, Dr Barringer, who was president-elect of AMWA in 1940, started a national committee that registered women physicians and worked to secure commissions for them in the armed services. Dr Barringer had recently retired from her position as Director of Gynecology of the Kingston Avenue Hospital in Brooklyn, New York, giving her time to devote to the difficult struggle for equal recognition in the armed forces. The AMA could not be persuaded to support the effort, but Dr Barringer and her committee found powerful allies in many prominent physicians and state medical societies that sent representatives to testify before Congress. This time public opinion was on their side with national women's clubs and the American Legion helping to lobby members of Congress. Commissions for women physicians became armed service policy in mid-April 1943 when President Roosevelt signed the Sparkman-Johnson Bill for "the appointment of female physicians and surgeons in the Medical Corps of the Army and Navy."[27]

At the 1943 annual meeting of WMS, Dr Barringer moved that resolutions of appreciation be sent to Representatives Celler, Merritt, Sparkman, and Senator Johnson who had introduced the legislation and worked for its passage. Thanks were also sent to the American Legion, Dr J. Stanley Kenney, Dr George W. Cottis, president of MSSNY, and Judge Dorothy Kenyon who had served as legal counsel for the committee. The next year, at the WMS annual meeting of 1944, featured speakers were two Navy Lieutenant Commanders and an Army Captain, all women physicians.

Women Physicians and WMS

Many women physicians who never joined WMS believed that a separate women's organization undetermined the acceptance of women within the profession. Others simply felt no need for a supportive group of women, and the majority were too busy even when they wanted to attend meetings. Statistics are sketchy, but it seems safe to say that at no time were a majority of New York State women physicians enrolled as members of WMS. At the height of its prestige in 1913, WMS records listed 382 women physicians practicing in New York State. Of these, 135 were active members of WMS, 56 were associate members, and 191 were not members.[39]

Lack of participation in WMS by women physicians was not surprising, considering that the state medical society did not attract a majority of male physicians, either, although the county societies' meetings were well attended. The women who organized WMS modeled it after MSSNY, the overall organization for the county medical societies. In a 1941 speech, Marion Craig Potter[40] described how she had joined MSSNY as a representative of the Monroe County Medical Society when she was young: "Delegates were sent to Albany for three years and then became members of the State Society with a certificate of membership," she said.

Joining WMS was much simpler. Members of the four local societies that founded WMS were expected to belong to it, and physicians who practiced where there was no local women's society were welcome members of the state organization. A prospective member completed an application blank, which was endorsed by two (later one) sponsoring members and accepted by vote at the annual meeting. The treasurer kept track of annual dues payments. Announcements of annual meetings were mailed to women members of all the county medical societies, and special membership campaigns solicited applications, usually with very low returns.

In spite of the low level of interest among the majority of women physicians, members of WMS cherished the friendship and collegiality they enjoyed in the Society. At the 1942 annual meeting of WMS, when Dr Potter was in her late 70s, she spoke of the "fellowship and inspiration I have always received from meeting with the women physicians during the past half century."[41] Along with a large practice in obstetrics and gynecology, she had found time to work for the advancement of women physicians on many levels, starting in her home city of Rochester. Marion and her two sisters, Sara and Anna, all earned MD degrees at the University of Michigan. After graduation, Marion practiced with her father, James Craig, MD, in Rochester for two years before she opened her own practice. She had seen how Dr Dolley organized the Provident Dispensary, and she became a character member of the Practitioners'/Blackwell Medical Society in Rochester. She was largely responsible for the organization of WMS in 1907 and was one of the founders of the MWNA, (later AMWA) in 1915.[7]

As a kind of elder statesman among women physicians, Dr Potter spoke at WMS dinners several times before her death in 1943. She told of her world tour in 1891 that started with a visit to her missionary sister Sara Buckley in Japan. She described the elderly Pasteur whom she visited at his institute in Paris and the patients she observed in Charcot's clinics. While in Europe, she met an old friend, Ezra Potter, MD, whom she married two years later. Their son James also became a physician.[42]

During World War I, Dr Potter worked with the MWNA War Service Committee under Rosalie Slaughter Morton. This committee organized the American Women's Hospitals in cooperation with the Red Cross "to meet the demand for the services of women physicians both at home and abroad."[43] Dr Potter took charge of the organization throughout New York State, bringing together willing medical talent and opportunities for hospital service, plus gathering contributions of money for the national committee. She also served on the Committee on Women Physicians of the General Medical Board during World War I and was editor of the committee's national "Census of Women Physicians."[7]

Membership in WMS dropped during and after both World Wars, followed by desperate struggles to keep the organization going. At an interim meeting in 1922 a motion to disband was passed and then tabled. There is no further reference to it in the minutes, but it may have been related to the resolution to join MWNA, which was passed at the annual meeting a few months later.

A similar situation developed after World War II. Early in January, 1950, Helen Walker, MD, of Buffalo wrote to WMS President Adelaide Romaine that during her past three years as president of WMS "an attempt was made to inject life into the organization, but the entire structure was so flimsy that the attempt proved abortive." She mentioned various factions, first in New York City and then upstate, who preferred having separate societies, ". . . and since the national cannot organize along state lines," she wrote, ". . . more effort should be placed on the national group. Therefore," she concluded, "Dr Klein (WMS secretary) and I feel the state society should be put out of its misery painlessly and allowed to lapse."[44]

A resolution to disband WMS was discussed by 22 members at the January, 1950, meeting held at the New York Infirmary. Those present favored continuing the state society. Telegrams and letters from absent members were read urging the group not to disband. Long-time members, Mary T. Greene[45] and Emily Barringer, recalled that WMS had nearly disbanded 25 years before, but "a few loyal women doctors carried on. How easy it is to tear down and how difficult to build up a good structure," Barringer's telegram said. Since then, the group had accomplished some important achievements such as the institution of statewide premarital and prenatal syphilis tests in the 1930s and the commissioning of women physicians in the armed services in the 1940s.[46]

The group agreed women physicians needed a statewide organization, and the resolution to disband was withdrawn. The meeting went on to appoint Past President Marguerite McCarthy-Brough, MD, of Syracuse chair of a committee to study reorganization and ways to increase membership. Of approximately 1,000 women physicians in the state, only 129 were paying members of WMS, average attendance at meetings was about 20, and only one local society, Buffalo, required its members to belong to the statewide group.[47]

In 1951, Dr McCarthy-Brough wrote to 1,500 women physicians in the state urging them to join WMS, and at the annual meeting in Buffalo that April she reported 69 new members.[48] WMS membership reached a high near 300 in 1959 and remained over 200 for most of the 1960s. Annual meetings preceded MSSNY meetings that were held alternately in Buffalo and New York City.

The presidents for the next 20 years alternated between the New York City and upstate areas. Meetings were held in members' homes, and women who lived in the host city brought in "potluck suppers," instead of arranging a formal banquet. Officers held midyear meetings in January or February to plan arrangements and programs for the annual meeting and take care of other business.

WMS Joins AMWA

Minutes of WMS meetings during the 1940s recorded many discussions about the issue of "amalgamation" with AMWA. The national group did not require its members to join county medical societies, and some WMS members feared amalgamation would lower the membership standards of their society. At the midyear meeting in 1945 in New York City, Dr Ruth Ewing presented a report[49] that made a strong case for amalgamation, pointing out that there were too many meetings for women physicians and combining them would strengthen both organizations. The purpose and objectives of WMS and AMWA were similar, she said, and amalgamations of this kind had been arranged in other states. She reported that an official Branch of AMWA required all members of the local group to belong to AMWA and that a Branch may be "part or all of a society existing before 1933." The WMS could retain its own identity, as state and county medical societies do while affiliating with the AMA. President Scanlan appointed a committee "to work out a method of amalgamation including allocation of dues, requirements for membership," and other details. This report was to be ready for the annual meeting in May, but war time restrictions on travel canceled the May meeting, and momentum for amalgamation was stalled for several more years. Dr Adelaide Romaine, who belonged to the New York City branch of AMWA, introduced the motion to make WMS an official branch of the national organization. On May 12, 1952, members at the annual meeting voted to become a branch of AMWA.[50] At their next meeting in January, 1953, they received personal greetings from President E. Stenhouse of AMWA congratulating them on their affiliation as Branch 18 of that organization.[51]

WMS Redefined

In 1966, a revised constitution was approved,[52] codifing changes that had been informally adopted, such as two-year terms of office in effect since 1943. The Council was renamed Board of Directors, with eight elected directors who also served two-year terms. District Councilors now were appointed. By-law changes eased membership requirements, changed the quorum at general meetings from 15 to nine, and increased the number of standing committees to ten, including Medical Economics, Membership, Obituary, Publicity, and Resolutions. Most of these had actually been standing committees since the early 1950s. The new by-laws also raised the dues to $3.00.[53]

Having a statewide organization of their own became even less important for women physicians as they gained professional recognition and acceptance in the various medical specialties and joined those professional societies. WMS membership totals dwindled gradually during the 1970s. Branch 18 of AMWA and WMS, now one organization, gradually shifted to less formal organization.

Another constitutional revision proposed during the presidency of Patricia Numann, MD, of Syracuse in 1980 added a new clause to the statement of purpose: "to be a supportive group for women medical students and those in training."[54] As the women's movement swept the country in the late 1960s and early 1970s, women physicians began to understand their vulnerable position as a small minority in a male-dominated profession. Books and journal articles addressed the loneliness of the women medical student. AMWA offered leadership training courses to raise the consciousness of members about the need for mutual support among women physicians. Networking became an important part of membership meetings, and scientific programs offered continuing education credits for members who attended. Women physicians worked with student chapters of AMWA to help the younger women choose careers and apply for hospital positions.

Adapting to the increasing specialization of their profession, women physicians organized their own associations within traditionally male specialties such as surgery and radiology. They worked for and gained office within the county and state medical societies and professional specialty groups. They established an AMA Task Force on the Status of Women to take political action for or against proposed laws and regulations.

In the 1990s, with women making up an average one-third of medical school graduates, loneliness is less of a problem, but the members of WMS have not forgotten how it was when women were only a peripheral presence in the medical profession. Leslie Kohman, MD, of Syracuse who became president of WMS in 1986 combined a membership drive with an informal meeting style to bring together old and new members and students and keep them aware of continuing efforts for advancement of women in medicine. WMS Branch 18 plans programs with other local organizations such as the Women's Bar Association and the Faculty Women's Caucus. Statewide, WMS members hold official meetings at the time of MSSNY meetings and alternate state society program presentations with AMWA Branch 14, the New York City wing of WMS.

Conclusion

The 75th anniversary of AMWA motivated this look backward at 83 years of WMS history. If Sarah Adamson Dolley were to observe her profession today, she would be gratified by the vision of thousands of capable women physicians confidently filling positions in all ranks of the medical profession. She would applaud their determination to share both responsibilities and rewards equally with men physicians, and she would be especially proud of the few dedicated women who have led WMS. They have followed her in organized effort "to cultivate social relations and mutual helpfulness among women physicians."

References

1. Women's Medical Society of New York State: Constitution and Bylaws, undated, Box 2, Society Archives. (Archives of WMS, Department of Manuscripts and University Archives, Olin Library, Cornell University, Ithaca, NY).
2. Minutes of WMS annual meeting, May 12, 1952, Society Archives.
3. Waite FC: Dr Lydia Folger Fowler: The second women to receive the degree of doctor of medicine in the United States. *Annals of Med Hist* 1932; 4:290–297. (Collections of the Onondaga Historical Association)
4. Index for the correspondence of Sarah Read Adamson Dolley, MD (1829–1909), (History of Medicine Department, Edward G. Miner Library, University of Rochester Medical Center, Rochester, NY).
5. Obituary: Sarah Dolley, MD, *Post Express*, Dec. 27, 1909, Marion Craig Potter papers, (record/scrapbook, Edward G. Miner Library, University of Rochester Medical Center, Rochester, NY)
6. Unidentified newspaper clipping, Marion Craig Potter papers, Record/scrapbook.
7. More E: The Blackwell Medical Society and the Professionalization of Women Physicians. *Bull Hist Med* 1987; 61:603–628.
8. Dolley SA: Address. *Women's Medical Society of New York State: Constitution and By-Laws*. Society Archives, 1908.
9. Smith, Elinor: *Descendants of Nathaniel Dickinson*, Dickinson Family Association, 1978.
10. Minutes of WMS annual meeting, March 11, 1908 (Marion Craig Potter papers, Record/scrapbook).
11. Minutes of WMS Council meeting, October 6, 1910 (Marion Craig Potter papers, Record/scrapbook).
12. Minutes of WMS annual meeting, March 10, 1911 (Marion Craig Potter papers, Record/scrapbook).
13. Program seventh annual meeting of WMS, Society Archives, Box 1, folder 32.
14. Minutes of WMS annual meeting, April 28, 1913 (Marion Craig Potter papers, Record/scrapbook.
15. AMA Council on Health and Public Instruction: Committee for public education among women, undated (Marion Craig Potter papers, Record/scrapbook).
16. Program, WMS banquet, 7:30 pm, Hotel Seneca, April 28, 1913 (Marion Craig Potter papers, Record/scrapbook).
17. Women's Medical Society of New York State annual meeting *NY State J Med* 1917; 17:189.
18. Women's Medical Society of New York State annual meeting *NY State J Med* 1918; 18:159.
19. Women's Medical Society of New York State annual meeting *NY State J Med* 1919; 19:105.
20. Minutes of WMS annual meeting, March 11, 1910 (Marion Craig Potter papers, Record/scrapbook).
21. Minutes of WMS annual meeting, April 17, 1922 (Marion Craig Potter papers, Record/scrapbook).
22. Minutes of WMS council meeting, March 22, 1917 (Marion Craig Potter papers, Record/scrapbook).
23. Letter to "Dear Doctor" from Eliza Mosher, Marion Craig Potter, and Mary T. Greene, November 11, 1919 (Marion Craig Potter papers, Box 1, folder 11).
24. Minutes of WMS annual meeting, May 2, 1921 (Marion Craig Potter papers, Box 1, folder 11).
25. Minutes of AMS annual meeting, March 29, 1926 and council meeting (undated, 1925) (Marion Craig Potter papers, Box 1, folder 11).
26. Minutes WMS annual meeting, May 21, 1923 (Marion Craig Potter papers, Record/scrapbook).
27. Morantz-Sanchez RM: *Sympathy and Science: Women Physicians in American Medicine*. New York, Oxford University Press, 1985.
28. Greene Sanitarium. Undated. Society Archives, Box 1, folder 32.
29. Obituary, Rosetta Sherwood Hall, *JAMWA*, August 1951, p 323.
30. Greene MT: Report, WMS annual meeting minutes, April 29, 1946, Society Archives.
31. Greene MT: Report, WMS meeting minutes, May 5, 1947, Society Archives.
32. Membership list, WMS, April 28, 1913, Society Archives.
33. Matilda Wallin Medical Education Loan Fund, September 23, 1962, amended May 13, 1963, Society Archives.
34. Minutes of WMS annual meeting, May 11, 1958, Society Archives.
35. Minutes of WMS annual meeting, Feb 8, 1970, Society Archives.
36. Minutes of WMS annual meeting, Feb 12, 1972, Society Archives.
37. Smith J: Report, undated, minutes of annual meeting, February 13, 1971, Society Archives.
38. Barringer ED: *Bowery to Bellevue: The Story of New York's First Woman Ambulance Surgeon*. New York, WW Norton, 1950.
39. Membership List, 1913 (Marion Craig Potter papers, Record/scrapbook).
40. Potter MC: Speech, March 28, 1941, Society Archives, Box 1, folder 2.
41. Potter MC: Response to toast, Jan 24, 1942 (Marion Craig Potter papers, Box 1, folder 11).
42. Index for Marion Craig Potter, MD (1863–1943) papers (Edward G. Miner Library, University of Rochester Medical Center).
43. *Association of Women Physicians*, undated (Marion Craig Potter papers, Record/scrapbook).
44. Letter from Helen G. Walker, MD, to Adelaide, January 8, 1950, Society Archives.
45. Letter from Mary T. Greene, MD, Society Archives, Box 1, folder 30.
46. Minutes of WMS midyear meeting, Jan 28, 1950, Society Archives.
47. Letter from Adelaide Romaine, MD, to WMS members, April 28, 1950, Society Archives.
48. Minutes of WMS annual meeting, April 30, 1951, Society Archives.
49. Minutes of WMS midyear meeting, February 3, 1945, Society Archives.
50. Minutes of WMS annual meeting, May 12, 1952, Society Archives.
51. Minutes of WMS midyear meeting, January 24, 1953, Society Archives.
52. Minutes of WMS meeting, February 13, 1966, Society Archives.
53. Proposed constitution and bylaws for voting, February, 1966, Society Archives.
54. Proposed constitution and bylaws, 1980 (WMS Secretary's records).

Lytic osseous destruction in vertebral sarcoidosis

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Zeba Jelveh, MD

Sarcoidosis is a chronic granulomatous disease that most frequently affects the lungs; however, almost any organ system can be involved. Vertebral involvement is quite rare and to date only nineteen cases of vertebral sarcoidosis have been described in the English language literature. Our case is unusual in the type and extent of vertebral involvement and represents the first documented demonstration of magnetic resonance imaging (MRI) findings in vertebral sarcoidosis.

Case Report

A previously healthy, 29-year-old black male complained of slowly progressing lower back pain for one year before admission. The pain was present at rest and exaggerated by movement. The patient admitted to a 20-pound weight loss in the previous year and a history of intermittent fever and night sweats. There was no cough or shortness of breath. There was no history of tuberculosis in the family, and he denied risk factors for human immunodeficiency virus (HIV) infection.

Physical examination demonstrated a fairly well-nourished male with normal vital signs. There were bilateral, small, and non-tender cervical, axillary, and inguinal lymph nodes. There was mild tenderness over the lumbar spine. The motor and sensory examination of extremities was normal with normal deep tendon reflexes. Findings of ophthalmic examination were normal.

Initial investigations revealed the following: Mild anemia of chronic illness, sedimentation rate of 22 mm/hr; serum alkaline phosphate of 96 (normal range 30–115); serum protein electrophoresis was consistent with polyclonal gammopathy. Serum and urine calcium and phosphate levels were normal. Angiotensin converting enzyme level was 65 U/L (normal 10–50 U/L).

The purified protein derivative (PPD) test was negative (not anergic). The sputum was negative on smear and culture for acid fast bacilli. A chest radiograph disclosed bilateral hilar adenopathy. Plain radiographs of the spine showed multiple osteolytic lesions involving the lower cervical, thoracic, and lumbar vertebral bodies and pedicles (Fig 1).

Further work-up included computed tomography (CT) of the spine, showing multiple osteolytic lesions of the dorsal and lumbar vertebrae with extensive paraspinal soft tissue masses (Fig 2). An MRI of the spine revealed numerous infiltrative areas of abnormal signal altering the normal marrow. Almost all vertebral bodies and disc spaces were involved with paraspinal and epidural extension (Figs 3,4). The patient had a bone marrow aspiration which showed mild panhyperplasia, no granulomas, and negative stains for AFB and fungi. A biopsy of axillary lymph node revealed numerous non-caseating granulomas. The patient underwent open biopsy of the right iliac crest which showed non-caseating granulomas involving the bone marrow and periosteal soft tissue consistent with sarcoidosis (Fig 5).

Multiple special stains for micro-organisms (including methenamine silver, periodic acid-Schiff, and carbol fucsin acid fast) on all three biopsy specimens were negative, as were cultures for bacteria, fungi, and acid-fast bacilli.

The patient was started on prednisone 60 mg daily. He started to show evidence of improvement in the form of disappearance of fever, weight gain, and less back pain. He was discharged on tapering dose of prednisone. However, he was lost to follow-up and stopped taking the treatment. Three months later, he was seen in the outpatient clinic. He was symptomatically much better; however, he

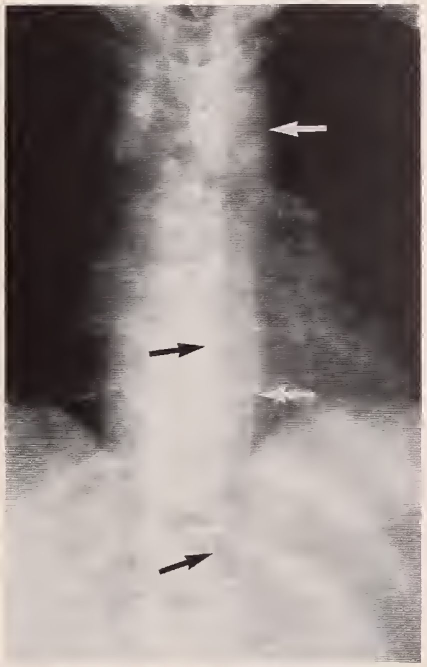

FIGURE 1. Anteroposterior radiograph of thoracic spine demonstrating mixed lytic and sclerotic areas of pedicle and vertebral body destruction (black arrows) and paraspinal soft tissue mass (white arrows).

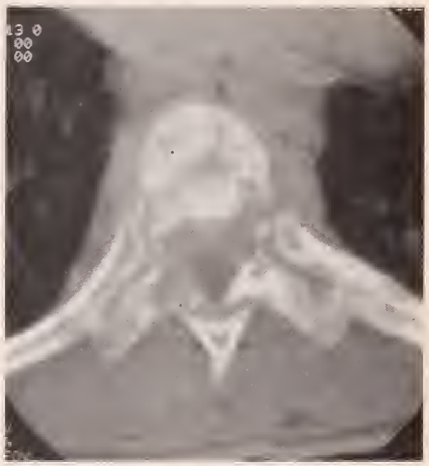

FIGURE 2. Axial CT image of thoracic spine demonstrating the diffuse osseous destruction with epidural and paraspinal extension.

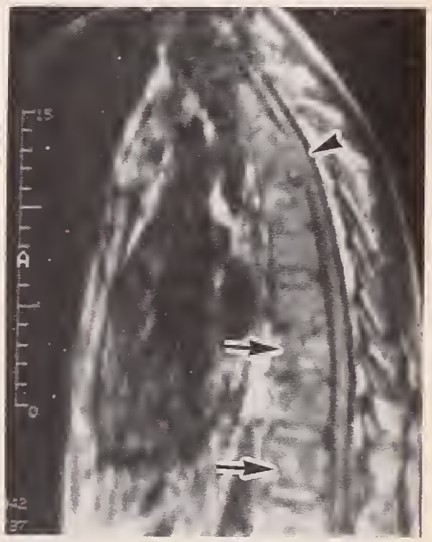

FIGURE 3. MRI study of midsagittal thoracic spine with T1 weighted image demonstrating diffuse multifocal hypointensity of vertebral bodies (arrows) with epidural effacement of upper thoracic cord (arrowhead).

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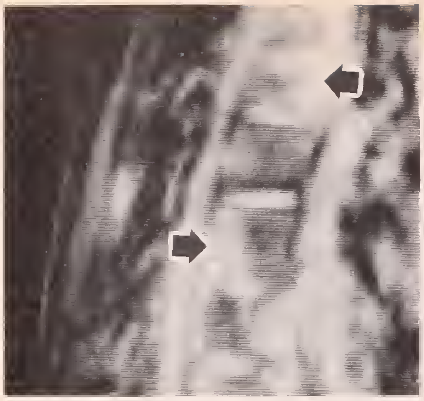

FIGURE 4. MRI study of thoracic spine using gradient echo (T2 weighted) technique: the granulomatous lesions become diffusely hyperintense (arrows). Epidural involvement is again demonstrated.

was found to have new periorbital skin lesions consistent with lupus pernio. Two years after the onset of symptoms the patient is alive and well.

Discussion

Sarcoidosis is a multisystem disorder of unknown etiology characterized by the presence of noncaseating granulomas in two or more organs. Sarcoidosis has world-wide distribution with significant variation in incidence in different countries. Although the prevalence rate in United States is not known, the prevalence in New York City approaches 40 per 100,000 population.[1]

Sarcoidosis has a greater incidence and severity in blacks, especially those born in southeastern states. Black females are afflicted approximately twice as often as males. Comparisons show an earlier age of onset and milder disease in Caucasians.[2]

Skeletal involvement is an uncommon manifestation of sarcoidosis. The radiographically apparent incidence ranges from 1% to 13% in different series. The true incidence may be higher since most of the lesions are asymptomatic.[3,4] Skeletal involvement is rarely encountered without significant pulmonary and cutaneous disease. The association between bone lesions and chronic skin lesions, particularly lupus pernio, is well recognized.[5] Although hilar adenopathy were noted on chest radiograph, our patient had no clinically evident pulmonary involvement, and skin lesions developed only after the bone lesions were noted and biopsied, which is unusual.

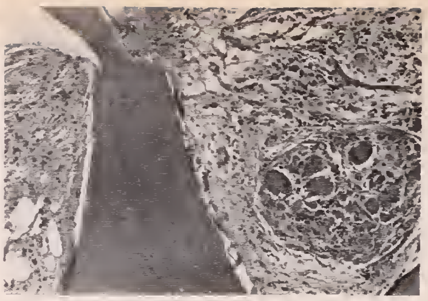

FIGURE 5. Bone marrow replaced by multiple non- caseating granulomata with destruction of bony spicule. (Hematoxylin-Eosin stain; original magnification 250.)

The vertebral lesions of sarcoidosis radiographically appear lytic with sclerotic margins, or purely lytic or sclerotic,[6] of the nineteen reported cases of vertebral sarcoidosis, only seven have been purely lytic, and none showed involvement of more than two vertebrae.[3] The case reported by Resnik et al was first to demonstrate multiple small lytic lesions in the spine- and pelvis-simulating metastatic disease. Involvement is most common in the lower thoracic and upper lumbar spine. Pedicle involvement has been previously described in only one case of vertebral sarcoidosis involving the lumbar spine.[7] Four cases of sarcoidosis with paraspinal masses and four cases with cervical spinal involvement have been described previously.[6] Our patient had both these unusual features. Thus, our patient, the twentieth reported case of vertebral sarcoidosis, was unique with respect to the extent of his disease and type of involvement.

The basic pattern of bony abnormalities include diffuse marrow infiltration, punched-out lesions, destructive, or neuropathic- like lesions and sclerotic lesions.[3] The permeative pattern, as seen in our patient, is associated with granulomatous infiltration of the haversian system.[3] On the MRI images, the granulomatous infiltration results in alteration of the normal marrow T1 and T2 characteristics. Inflammatory tissue has a prolonged T1 and T2 relaxation resulting in excellent contrast differentiation from normal. Gradient echo techniques of MRI may result in greater detection.

Symptoms of vertebral sarcoid are usually limited to pain. Rarely, vertebral collapse and neurologic impairment may ensue. If neurologic involvement occurs secondary to vertebral destruction, it may require stabilization and decompression.[8]

In summary, we have presented a pathologically proven case of vertebral sarcoidosis detected by plain films, CT, and MRI. The radiographic appearance of this entity is not specific and a tissue biopsy is always necessary to exclude neoplasm of infection.[9] The clinical course is variable. Spontaneous improvement both clinically and radiographically has been noted on several occasions in the literature.[10] From the cases reviewed, it is difficult to ascertain the role of steroids in controlling the progression of bone disease. MRI is an excellent tool for detection of vertebral collapse and cord compression in these cases.

References

1. Sharma OP: Sarcoidosis: *Disease-A-Month* 1990; 36:469–535.
2. James DG, Neville E, Carstairs LS: Bone and joint sarcoidosis. *Semin Arthritis Rheum* 1976; 6: 53–58.
3. Resnik CS, Young YW, Aisner SC, et al: Case Report 594: Osseous sarcoidosis of lumbar spine and pelvis. *Skeletal Radiol* 1990; 19:79–81.
4. Sartoris DJ, Resnik C, Resnick D, et al: Musculoskeletal manifestations of sarcoidosis. *Semin Roentgenol* 1985; 20:376–386.
5. Oven TJ, Sones M, Morrissey WL: Lytic lesion of the sternum. Rare manifestations of sarcoidosis. *Am J Med* 1986; 80:285–287.
6. Cutler SS, Sankaranarayanan G: Vertebral sarcoidosis. *JAMA* 1978; 240:557–558.
7. Rockoff DS, Rohatgi PK: Unusual manifestation of thoracic sarcoidosis. *AJR* 1985; 144:513–528.
8. Budens DA, Rechtine GR: Sarcoidosis of the spine: Case report and literature review. *Spine* 1986; 11:209–212.
9. Rodman R, Funderburk EE, Myerson RM: Sarcoidosis with vertebral involvement. *Ann Intern Med* 1959; 50:213–218.
10. Perlman S, Damergis J, Witorsch P, et al: Vertebral sarcoidosis with paravertebral ossification. *Arthritis Rheum* 1978; 21:271–276.

Clavicular resection in the exposure and resection of malignant tumors

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Total or partial clavicular resection has been done in the past for a variety of indications, such as comminuted fractures, dislocations, or non-union,[1–3] the need to lengthen a pectoralis major myocutaneous flap,[4] surgical exposure of neurovascular structures[5] or the upper thoracic vertebrae,[6,7] osteomyelitis,[8] and primary or metastatic tumors of the clavicle.[1,2,9,10–12] Partial or total removal of the clavicle did not result in functional limitations; in fact, one investigator considers the clavicle a surplus part of the skeleton.[13] In all of these cases, the clavicle was removed without substantial dissection and resection of surrounding soft tissues, which could further destabilize the local anatomy.

To our knowledge, there has been no report in the literature of clavicular resection performed to provide the necessary

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exposure and make feasible the enbloc resection of an underlying tumor extending to both the supraclavicular and infraclavicular areas. Described here are 11 patients seen on the Soft Tissue-Melanoma & Bone Service (STM/B) at this hopsital in whom clavicular resection was used (seven for metastatic melanoma to supraclavicular and axillary lymph nodes, and four for adjacent soft tissue tumor). In all these patients, the metastatic work-up revealed no evidence of disease elsewhere. A brief summary of the clinical course of these patients is given below.

CASE REPORTS

Case 1. A 66-year-old man, with a history of malignant melanoma of the skin of the right shoulder that was diagnosed and treated in 1976, was referred to the STM/B Service in eight months with a right supraclavicular mass extending behind the clavicle. Needle biopsy confirmed this to be malignant melanoma. At the time, a right supraclavicular, infraclavicular, and axillary lymph node dissection with incontinuity resection of the right clavicle was performed. The patient did well and was discharged from the hospital on the ninth day after surgery. He had no difficulty in shoulder and arm movements, although moderate postoperative edema of the right arm developed. He died due to progression of his disease 45 months after the operation.

Case 2. A 44-year-old man had had a malignant melanoma of the skin of the right back, which had been excised at another institution. He was referred to the STM/B Service 14 months later, with large palpable nodes in the right axilla and supraclavicular area. A right axillary node dissection with incontinuity claviculectomy and right supraclavicular node dissection was performed. His postoperative recovery was uneventful.

On follow-up in the clinic, the patient complained of pain in the right neck radiating to the arm. There was no swelling in the right arm. Roentgenograms of the cervical spine revealed osteoarthritis, and the pain was controlled with simple analgesics. The patient experienced recurrence of his disease 21 months later and succumbed to the illness 29 months after his operation.

Case 3. A 62-year-old man had wide excision of malignant melanoma of the skin of the left scapular region and left therapeutic axillary node dissection late in 1988. Two months later, he was found to have left supraclavicular and infraclavicular masses. He was referred to STM/B Service, underwent left axillary lymph node dissection and incontinuity resection of clavicular and supraclavicular lymph nodes. Seven of 19 supraclavicular lymph nodes and 12 of 12 lymph nodes from the axilla were positive for metastatic melanoma. He was discharged from the hospital on the 11th day after surgery. He regained full range of motion without any swelling of the left arm on follow-up. Two months after his operation, a repeat computed axial tomographic (CAT) scan revealed metastases to the liver and lungs. He died from progression of the disease three months later.

Case 4. A 75-year-old man had had wide excision of malignant melanoma (Clark's level III, 2.1-mm thick) of the right forearm early in 1984. He also underwent right axillary lymph node dissection. He was found to have enlarged lymph nodes in the right supraclavicular area and right axilla and was referred to STM/B Service. He underwent right supraclavicular lymph node dissection with incontinuity resection of the right clavicle, axillary lymph nodes, and part of the pectoralis major and minor muscles at that time. He was discharged from the hospital 13 days later. The pathology report revealed malignant melanoma of soft tissue adjacent to the pectoralis major muscle and in the supraclavicular lymph nodes. On follow-up, the patient regained, with the help of physical therapy, excellent range of motion with no obvious deformity and no arm swelling. Three months later, left cervical node metastases were discovered, and he underwent left modified neck dissection. He was treated with chemotherapy. Osseous and other metastases, later developed, and he expired 13 months after surgery.

Case 5. A 57-year-old woman had wide excision of malignant melanoma (Clark's level III, 2.4-mm thick)in early 1978. Ten months later, an enlarged, hard, lymph node in the left supraclavicular area and axilla was found. A biopsy revealed metastatic melanoma. She was then referred to the STM/B Service and, one month later, a left axillary lymph node dissection with incontinuity dissection of the supraclavicular lymph nodes and claviculectomy was performed. The pathology report revealed that 7 of 14 axillary and 2 of 11 supraclavicular lymph nodes were positive. Her postoperative course was unremarkable, and she was discharged home nine days later. The patient has remained well without evidence of recurrence and no physical deformity, swelling, or limitation of movement 23 months later.

Case 6. A 55-year-old man with malignant melanoma of the trunk (Clark's level IV, 3.3-mm thick) underwent wide-excised and left axillary node dissection in the summer of 1983. Five of 35 lymph nodes were positive on histologic evaluation. In 1987, the patient was treated for two brain metastases on the left side and underwent craniotomy and excision of these masses late in 1987, followed by postoperative radiation therapy. He did well without any symptoms, until he was found to have a 7-cm mass in his left supraclavicular region 26 months later. There was no evidence of other active disease. One month later, he underwent resection of the clavicle, left radical neck dissection, and superior mediastinal lymph node dissection. Twelve of 30 posterior neck nodes were positive for melanoma. The patient recovered well and was discharged from the hospital on the 12th day after surgery. No swelling of the left arm occurred in the follow-up period. Tumor recurrence was noted five months later, and the patient expired due to progression of the disease two months after that.

Case 7. This 77-year-old man had had malignant melanoma of the skin of the right back (Clark's level III, 10-mm thick) and palpable adenopathy in the right axilla, for which wide excision of the primary lesions and right axillary node dissection was performed in mid-June 1988 at another hospital. He was referred to the STM/B Service nine months later with masses in the right supraclavicular area and in the infraclavicular area deep to the pectoralis major muscle. An en bloc resection of the supraclavicular and infraclavicular masses with the clavicle was performed. On histologic evaluation, the matted mass of nodes above and below the clavicle was positive for melanoma, as well as the marrow of the removed clavicle. Moderate swelling of the right arm developed after surgery. Nineteen months after this operation, the patient remained disease-free.

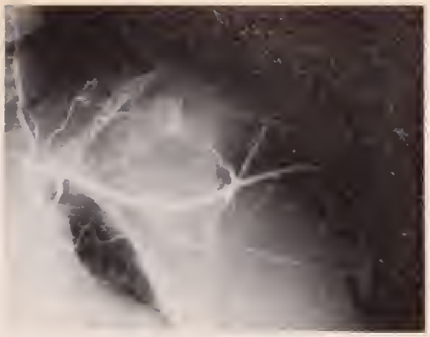

FIGURE 1. Caudal displacement of the axillary artery by the tumor and neovascularity on the arteriogram.

Case 8. This 55-year-old woman presented with a mass in the left infraclavicular region in mid-1985. A biopsy revealed desmoid tumor, and forequarter amputation was offered, which the patient refused. She was referred to the STM/B Service 9 months later. The tumor was located deep to the pectoralis major muscle and histologically was shown to be a desmoid. An arteriogram demonstrated caudal displacement of the left axillary artery and tumor neovascularity (Fig 1), and a CAT scan delineated a large mass in the left axilla (Fig 2). One month later, en bloc resection of the tumor with the left clavicle, pectoralis major and minor, and subscapularis muscles was performed. Portions of the deltoid, teres major and minor, serratus anterior, coracobrachialis, and short head of the biceps were also removed. Intraoperative radiation to 1,000 rad was given. The patient recovered well until the seventh postoperative day, when the left arm became swollen. A venogram demonstrated thrombosis of the axillary vein. This was treated with heparinization and elevation of the arm.

This patient has had a residual, moderate swelling of the left arm for which she elevates the arm at night and occasionally uses an elastic stockinet. She has been able to use this

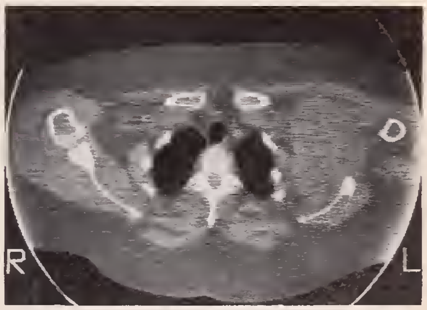

FIGURE 2. Outline of the mass in left axilla on CT scan.

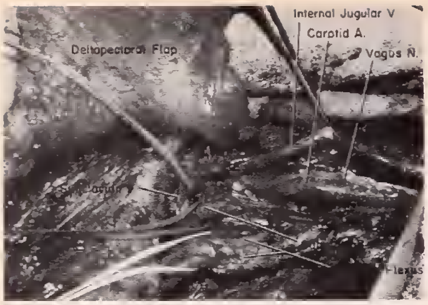

FIGURE 3. Following resection of a desmoid tumor in the lower neck, en bloc with claviculectomy. The two arrows on the medial and lateral side have been placed to indicate the position of the medial and lateral ends of the clavicle, had this bone not been removed.

arm well, and at 66 months she is free of recurrence.

Case 9. A 34-year-old woman was found to have a right, lateral neck mass in late 1986, and biopsy revealed a Grade 2 fibrosarcoma. On CAT scan, the mass extended behind the clavicle. A forequarter amputation was advised, which the patient declined. She was referred to the STM/B Service, and four months after diagnosis, excision of the supraclavicular sarcoma with en bloc resection of the clavicle and resection of the right subclavian artery with Gore-tex graft replacement was performed. She was discharged from the hospital on the eight postoperative day. Because of a question of residual tumor, a second procedure two years later was performed, revealing only scar tissue. She also had adjuvant radiation at the operative site. The patient has done well with full range of motion for her left arm, no swelling, and no evidence of local or systemic recurrence 57 months since her operation.

Case 10. A 54-year-old man with a history of von Recklingenhausen's disease had local excision of a neurofibrosarcoma of the left neck and postoperative radiation in 1974. He was referred to the STM/B Service with a locally recurrent mass 14 years later. A large mass was palpated in the left supraclavicular region; it was 8 × 8 cm in diameter and extended behind the left clavicle. Biopsy revealed a Grade I neurofibrosarcoma. The next day, wide excision of the tumor with incontinuity resection of left clavicle, wedge excision of a portion of the left lung adherent to the tumor, and resection of a portion of the left subclavian vein was performed. A Gore-tex graft was used to restore the continuity of the subclavian vein, and a deltopectoral flap was used to cover the skin defect created in the neck. The patient regained full range of motion at the left shoulder joint after surgery and had no swelling in the left arm. Seven weeks later, sudden thrombosis of the subclavian vein graft developed and was confirmed on venogram; the thrombosis produced swelling of the left upper extremity. This was treated with intravenous heparinization followed by oral coumadin and elevation of the arm. The swelling of the arm receded completely with this treatment. One year after surgery, a local recurrence was resected en bloc with the manubrium, and the defect was closed with a pectoralis major myocutaneous flap. He did well and was discharged from the hospital on the sixth day after surgery. The tumor recurred in the upper neck and paravertebral area 18 months after the last operation, and at present, he is undergoing treatment with radiation and chemotherapy. His left arm is of normal size.

Case 11. A 75-year-old woman had a desmoid tumor in the left lower neck, which had been removed in Greece in April 1990. A year later, she presented with local recurrence at another institution and a forequarter amputation was considered. She was referred to the STM/B Service. On physical examination, there was a large fixed mass in the lower neck.

Surgery performed in mid-1991 was initiated with an elliptical, low-transverse incision in the lower neck, paralleling the clavicle. A large portion of the skin directly over the tumor was left attached to it. The superior flap was made, and the carotid artery and internal jugular vein were dissected off the tumor overlying the brachial plexus so that the roots of the plexus could not be exposed through this approach. The inferior flap was then made to the surface of the clavicle, making plain that the tumor was extending below this bone. The clavicular origin of the pectoralis major muscle was then detached, and the clavicle was divided medially and laterally with a Gigli saw. The brachial plexus and the subclavian artery and vein were thus exposed below the clavicle in an area well below the tumor, making it possible to dissect safely on the surface of these structures in a cephalad direction and remove the tumor without injury to the nerve roots. The skin defect was closed with a deltopectoral flap (Fig 3). The patient was discharged from the hospital seven days later with good wound healing and no swelling in the left upper extremity. She was advised to have adjuvant radiation in Greece and, on the basis of the latest communication, she remains well four months after her procedure.

Data on the above patients are summarized in Table I.

DISCUSSION

The clavicle provides the site of insertion or origin for several muscles. At the superior border the attachments of the sternomastoid and trapezius muscles involve the medial and lateral ends of this bone. In claviculectomy, for exposure of the underlying lymph nodes and incontinuity resection of the supra- and infra-clavicular nodes, only the central portion of the bone need be removed, leaving in place about 1 cm of the bone from each end. The sternomastoid originates in the manubrium sterni, whereas the trapezius muscle has a wide insertion continuing to the acromion and the spine of the scapula.

At the inferior border of the clavicle there is the clavicular origin of the pectoralis major muscle. However, this muscle also has an extensive origin from the side of the sternum and the adjacent ribs. The deltoid muscle originates from the lateral third of the clavicle, as well as from the acromion and the spine of the scapula. Posteriorly, the subclavius, a small muscle, stretches between the clavicle and the first rib. Other than the ligaments fastening the two ends of the clavicle to the adjacent bones, the coracoclavicular ligament, near the acromioclavicular articulation, connects the clavicle to the coracoid process of the scapula.

From the foregoing discussion, it is apparent that claviculectomy does not defunctionalize any of the large muscles attached to this bone, as their attachment also involves other adjacent bones. With claviculectomy, the skeletal continuity of the upper extremity to the trunk is lost, but this does not appear to impair substantially the function of the arm. The connection of the extremity to the arm is maintained, in addition to the connections via the skin and neurovascular structures, with powerful muscles: posteriorly, the levator scapulae, trapezius, latissimus dorsi and rhomboids; anteriorly, the pectoral muscles; medially, the serratus anterior; and laterally, the deltoid muscle.

One theoretical concern is whether removal of the clavicle may result in a gradual drop of the shoulder point and traction on the brachial plexus with attendant neurologic symptoms. Of 11 patients who underwent claviculectomy, one patient, early in our experience, complained later of pain extending from the neck to the ipsilateral arm. This patient also had evidence of

TABLE I. Claviculectomy for En-Bloc Resection of Adjacent Malignant Tumors

No.	Sex (Age)	Prior Disease-free Interval (mo)	Survival after Procedure (mo)	Arm Pain/Swelling		Present Status*
1	M(66)	19	45	No	Yes	DWD
2	M(44)	14	29	Yes	No	DWD
3	M(62)	2	3	No	No	DWD
4	M(75)	60	13	No	No	DWD
5	F(57)	142	23	No	No	AND
6	M(55)	26	7	No	No	DWD
7	M(77)	21	19	No	Yes	AND
8	F(55)	—	66	No	Yes	AND
9	F(34)	—	57	No	No	AND
10	M(54)	—	35	No	No	AWD
11	F(75)	—	4	No	No	AND

*DWD: Dead with disease; AND: Alive, no disease; AWD: Alive with disease.

cervical osteoarthritis with narrowed vertebral interspaces. Because of this initial experience, in all subsequent patients we have used a sling applied so as to prevent caudal migration of the shoulder. The sling is used for three to four weeks, although not constantly, and is removed temporarily for physical therapy or in the execution of other daily routines. No subsequent patient has complained of pain radiating to the arm. The use of a sling is definitely helpful in providing a more comfortable posture and support until healing is completed; whether, it prevents to some extent caudal migration of the shoulder is more doubtful.

Claviculectomy in itself would not be expected to cause lymphedema, and it does not. It is the combination of claviculectomy with supraclavicular and incontinuity axillary dissection, or the wide resection of adjacent soft tissues, that raises this concern. Of 11 patients who underwent this procedure, three patients have had permanent edema of the arm of moderate severity. In one of the three patients, thrombosis of the axillary vein developed postoperatively. The extent of the incisions, excessive thinness of the flaps interrupting subcutaneous lymphatics, concomitant resection of adjacent soft tissues, and wide excisions of the skin of the arm for malignant melanoma, together with the extensive dissection, bring about lymphedema of the extremity.

Claviculectomy en bloc with an underlying tumor often provides the necessary exposure for incontinuity removal of involved cervical and axillary nodes or underlying soft tissue tumor. Other than the completeness of resection, the result depends on the biological behavior and stage of the tumor being removed. Of the seven patients with melanoma on whom this procedure was used, five had relapses and died 3, 7, 13, 29, and 45 months after the procedure; two of the patients are alive and well 19 and 23 months after their operations. Therefore, it appears that this procedure is not useful for the majority of patients with melanoma and extensive regional involvement, although selected patients with long disease-free intervals before recurrence may experience worthwhile palliation and occasionally long-term survival.

Of the four patients with soft tissue tumors (one each with fibrosarcoma and neurofibrosarcoma, and two with desmoid), three are alive and well at 57, 66, and 4 months follow-up, whereas the patient with neurofibrosarcoma has an extensive local recurrence at the upper neck and paravertebral area being treated currently with radiation and chemotherapy, 34 months after the procedure of claviculectomy with en bloc resection.

The technique for incontinuity resection of large axillary and supraclavicular nodes with en bloc claviculectomy usually involves a T-incision with a transverse portion above and parallel to the clavicle and a vertical portion from the middle of the transverse incision, which is curved inferiorly and laterally to the medial wall of the axilla about 3–4 cm below the axillary crease. The clavicle may be divided near its medial and lateral ends with a Gigli saw. When necessary, for greater exposure, either or both of the clavicular ends may be disarticulated from the respective joints.

The above series of 11 patients indicates that claviculectomy en bloc with resection of underlying tumor is tolerated well by the majority of patients. This procedure should be considered after full evaluation of the patient to rule out disease elsewhere, and when the known biologic behavior of the tumor suggests a reasonable survival after complete resection. For soft tissue tumors subjacent to the clavicle, en bloc claviculectomy provides a method for their exposure and resection, and in some cases, may provide a method of limb salvage in contrast to a forequarter amputation.

REFERENCES

1. Gurd FB: The treatment of complete dislocation of the outer end of the clavicle. *Ann Surg* 1941; 113:1094–1098.
2. Wood VE: The results of total claviculectomy. *Clin Orthop* 1986; 207:186–190.
3. Copland SM: Total resection of the clavicle. *Am J Surg* 1972; 72:280–281.
4. Freeman MS, Thomas JR, Zippor JA: Clavicular division technique. *Arch Otolaryngol Head Neck Surg* 1989; 115:224–227.
5. MacCarty CS: Surgical exposure of the brachial plexus. *Surg Neurol* 1984; 21:593–597.
6. Lore JM, Szymula NJ: Superior mediastinal exposure. *Arch Otolaryngol* 1980; 106:6–7.
7. Sundaresan N. Shah J, Foley K, et al: An anterior surgical approach to the upper thoracic vertebrae. *J Neurosurg* 1984; 61:686–690.
8. Kochhar VL, Sirvastava KK: Anatomical and functional considerations in total claviculectomy. *Clin Orthop* 1976; 118:199–201.
9. Abbott LC, Lucas DB: The function of the clavicle. *Ann Surg* 1954; 140:583–599.
10. Lewis MM, Ballet FL, Droll PG, et al: En-bloc clavicular resection: Operative procedure and postoperative testing of function. *Clin Orthop* 1985; 193:214–220.
11. Turra S. Gisante C: Primary clavicular localization of Ewing's tumor treated by total cleidectomy. *Ital J Orthop Traumatol* 1988; 14:389–393.
12. Devaraj VS, Kay SP, Batchelor AG: Vascularized reconstruction of the clavicle. *Br J Plast Surg* 1990; 43:625–627.
13. Gurd FB: Surplus parts of the skeleton. A recommendation for the excision of certain portions as a means of shortening the period of disability following trauma. *Am J Surg* 1947; 74:705–719.

Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction

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A variety of hemorrhagic complications after thrombolytic therapy for acute myocardial infarction has been described previously in the medical literature.[1,2] Concerns regarding these complications have led to the establishment of relative and absolute contraindications for the use of thrombolytic therapy. This paper documents the first reported case of a renal angiomyolipoma diagnosed after retroperitoneal hemorrhage in a patient with acute myocardial infarction treated with tissue plasminogen activator. On the basis of this case, we recommend that the presence of a renal angiomyolipoma be included as one of the risk factors when using thrombolytic therapy.

CASE REPORT

A 63-year-old patient with hypertension had had an anterior wall myocardial infarction that was treated with recombinant tissue plasminogen activator (rt-PA) nine months before presentation. However, her hematocrit level fell immediately after treatment with rt-PA, and she required eight units of whole blood. In addition, urinalysis at that time revealed the presence of 25–30 red blood cells per high-power field. A contrast-enhanced computed tomography (CT) scan re-

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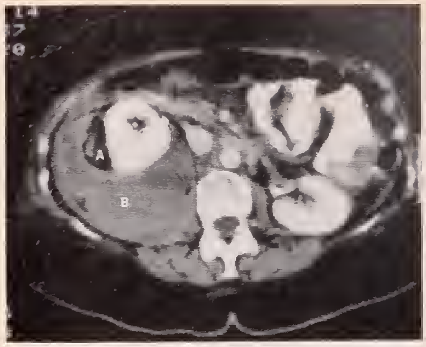

FIGURE 1. Initial CT scan after retroperitoneal hemorrhage. There is a mass extending from the mid-lateral portion of the right kidney (A) with a fat density. There is a large collection (B) around the right kidney consistent with hemorrhage.

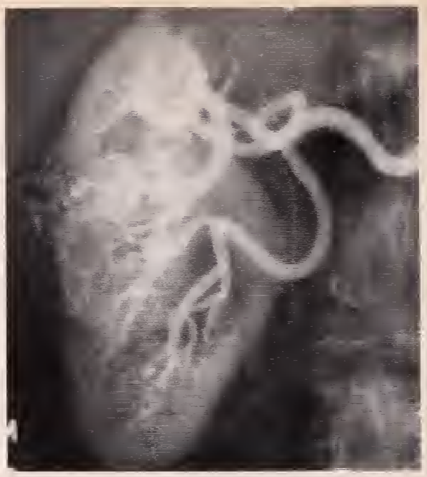

FIGURE 3. An angiogram demonstrating a hypervascular lesion at the lateral aspect of the kidney.

vealed both kidneys to be functioning normally. An ellipsoid, fat-density lesion, approximately 3 ×4 × 5 cm, was seen at the lateral margin of the right kidney. The right kidney was surrounded by a large collection consistent with acute hemorrhage (54 Hounsfield units) extending from the suprarenal area to deep into the pelvis. This probably represented several units of blood (Fig 1). She recovered without cardiac complications and was discharged from the hospital approximately one month later.

The patient was well after her discharge from the hospital. She had maintained left ventricular function and had no recurrence of hematuria. A follow-up abdominal CT scan two months later revealed a persistent fat-density lesion at the lateral margin of the right kidney. The perinephric hemorrhage had significantly resolved and was encapsulated in the perinephric space immediately behind the right kidney. Renal function was normal bilaterally.

Two months before this presentation, the patient experienced painless, gross hematuria for 12 hours, which ended spontaneously. A current urine culture and cytologic evaluations were negative. A current abdominal CT scan revealed almost complete resolution of the perinephric hemorrhage. The only remaining evidence was a 2-cm thick, rimmed, low-attenuation mass immediately adjacent to the right kidney. The previously described fat density remained unchanged (Fig 2).

Since she was 20 years old, hypertension and hypothyroidism were a part of the patient's medical history. Her current medications include the following: metoprolol, 25 mg twice a day; hydrochlorthiazide, 25 mg daily; triamterene, 50 mg daily; and levothyroxine, 0.1 mg daily.

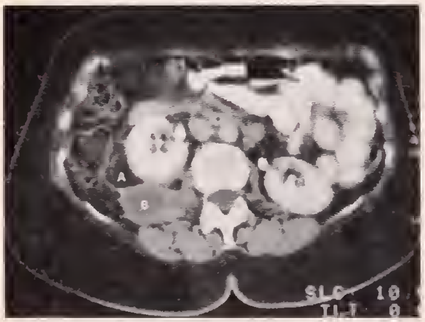

FIGURE 2. Follow-up CT scan six months later revealed a persistent mass lesion (A) and diminution in the size of the retroperitoneal hemorrhage (B).

On physical examination, we found a well-developed, well-nourished woman in no acute distress. Her pulse was 80 beats/min and regular, and her blood pressure was 130/90 mm Hg. Her abdomen was soft with no palpable masses. Her hemoglobin was 14.9 g/dL; the prothrombin time was 11.5 seconds, and the partial thromboplastin time was 29.9 seconds. She had no hematuria or pyuria, and urine cultures revealed no growth. Urine cytology revealed no malignant cells, and cystoscopy was normal.

Her chest film was unremarkable, with no evidence of pleural effusions. A selective right renal arteriogram revealed a hypervascular lesion, extending exophytically from the mid-lateral margin of the right kidney (Fig 3). This study revealed no evidence of acute hemorrhage. The remainder of the renal parenchyma was fed by the vessel catheterized (lower two-thirds of the kidney) and was unremarkable.

Renal exploration was performed via a thoraco-abdominal approach. We obtained vascular control of the right renal artery and vein. Attempts at wedge resection were hampered by the extensive fibrosis and scarring surrounding the kidney; therefore, we performed a right nephrectomy.

The specimen consisted of a right kidney and perinephric fat. On cut surface, areas of organizing subcapsular and perinephric hematoma were noted. A well-circumcised, 4.5-cm in diameter, soft, yellow mass surrounded by hematoma bulged into the perinephric fat at one pole. Most of the mass was extrarenal, although superficial interdigitation with the cortex was seen over a 0.5-cm length. Histologic findings disclosed a mass consisting mainly of mature fat. Scattered throughout were several large, thick-walled blood vessels and a few bundles of fibrillar spindle cells of a myoid appearance (Fig 4). The lesion merged with renal cortex over a small area with no intervening capsule. We confirmed the areas of hematoma noted on gross examination as well as numerous small foci of extravasated blood sprinkled within the lesion. No ruptured vessels were identified. These findings were consistent with a renal angiomyolipoma.

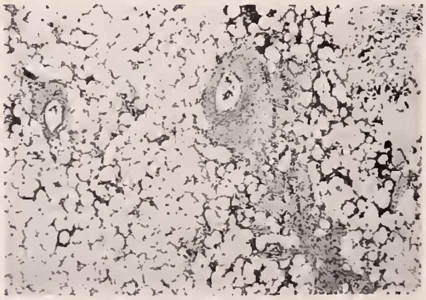

FIGURE 4. Histologic examination of the specimen demonstrated mature fat and thick walled blood vessels consistent with an angiomyolipoma.

DISCUSSION

The goal of thrombolytic therapy is the lysis of intravascular thrombi, causing tissue ischemia, without induction of a bleeding diathesis. Attempting intracoronary thrombus lysis led to intravenous use of converting plasminogen-to-plasmin thrombolytic agents. There is a limited window of time after the onset of acute myocardial ischemia during which reperfusion can salvage susceptible tissue and reduce the infarct size. Therefore, prompt treatment is imperative. Earlier studies reported improvement of ventricular function, with a significant decrease in mortality after thrombolytic therapy.[1,2]

The most common complication associated with thrombolytic therapy is bleeding.[1–3] Berridge et al[4] reported that major hemorrhage (defined as bleeding resulting in hypotension or requiring transfusion) occurred in 5.1% of patients, whereas the incidence of all other bleeding was 14.8%. Bleeding associated with thrombolytic therapy usually results in a small or moderate-sized hematoma at the skin site of placement of catheters. By avoiding nonessential invasive procedures, the incidence of post-thrombolytic therapy hemorrhage is approximately 5%.[5]

The mechanisms of hemorrhage are unclear; however, investigators believe that there are multiple causes, and they have discovered no reliable screening parameters to predict severe hemorrhage.[3,6] In addition, there is no difference in coagulation status between patients who have had major hemorrhage and patients who have not. Thrombolytic therapy is contraindicated in patients who have a history of cerebrovascular accidents, arteriovenous malformations, intracranial neoplasms, or a known bleeding diathesis.[1] However, bleeding has been reported in patients with no associated risk factors and a normal coagulation profile.[3,6]

Because of the concern of thrombolytic-induced hemorrhage, various alternative agents have been investigated. The use of fibrin-selective agents, such as rt-PA, was thought to be potentially safer than less selective agents, such as streptokinase or urokinase.[6,7] However, studies have shown that rt-PA is as equally associated with hemorrhagic complications as other therapeutic regimes that are less fibrin selective. Although rt-PA depletes serum fibrinogen levels to a lesser degree than conventional agents, no thrombolytic agent can discriminate between a pathologic thrombus or a protective hemostatic "plug" at another site. The recommended course of action after thrombolytic-induced hemorrhage is cessation of therapy, direct pressure to a puncture site, if applicable, and transfusion, if necessary.[1]

Renal angiomyolipomas fall into the general category of hamartomas. These are uncommon benign tumors sharply demarcated from adjacent renal parenchyma. They are composed of an admixture of fat, smooth muscle, and blood vessels, and they range from predominantly lipomatous to predominantly leiomyomatous.[8]

Most renal angiomyolipomas can be definitively diagnosed on the basis of a CT scan,[9,10] with the most common radiographic findings being a low-density, fat-containing, intrarenal tumor (Fig 2). Hemorrhage of excessive myomatous tissue may rarely obscure the fatty nature of the mass. In this patient, the radiographic findings were most consistent with an angiomyolipoma that had hemorrhaged into the perinephric space with subsequent, almost complete, resolution of the perinephric hemorrhage.

As more patients are treated with thrombolytic therapy, the number of cases of retroperitoneal hemorrhage secondary to a urologic cause will increase. This case is the first to our knowledge of a hemorrhage from an angiomyolipoma after institution of rt-PA. Patients who suffer acute hemorrhage after intravenous thrombolytic therapy should be examined for a retroperitoneal source. A CT scan is usually appropriate for an initial evaluation. The advantage of a CT scan is that, in addition to demonstrating retroperitoneal bleeding, possible urologic etiologies, including benign and malignant lesions of the kidney may be diagnosed. Finally, the presence of a renal angiomyolipoma should be included as one of the risk factors when using thrombolytic therapy.

REFERENCES

1. Loscalzo J, Braunwald E: Tissue plasminogen activator. *New Engl J Med* 1988; 319:925–929.
2. Topol EJ: Recombinant tissue plasminogen activator: Implications in therapy. *Sem Hemat* 1989; 26:25–35.
3. Conrad J, Samama MM: Theoretic and practical considerations on laboratory monitoring of thrombolytic therapy. *Semin Thromb Hemost* 1987; 13:212–219.
4. Berridge DC, Makin GS, Hopkinson BR: Local, low dose, intra-arterial thrombolytic therapy: The risk of stroke or major hemorrhage. *Br J Surg* 1989; 76:1230–1238.
5. Verstraete M, Collen D: Thrombolytic therapy in the eighties. *Blood* 1986; 67:1529–1535.
6. Rao AK, Pratt C, Berke A et al.: Thrombolysis in myocardial infarction (TIMI) trial—Phase 1: Hemorrhagic manifestations and changes in plasma fibrinogen and the fibrinolytic system in patients treated with recombinant tissue plasminogen activator and streptokinase. *J Am Coll Cardiol* 1987; 10: 970–975.
7. TIMI Study Group: The thrombolysis in myocardial infarction: Phase 1 findings. *New Engl J Med* 1985; 312:932–937.
8. Drago JR, Nesbitt JA, Leb R: Renal angiomyolipomas. *J Surg Oncol* 1988, 39:64–68.
9. Oesterling JE, Fishman EK, Goldman SM, et al: The management of renal angiomyolipoma. *J Urol* 1986; 135:1121–1126.
10. Zenman RK, Cronan JJ, Rosenfield AT, et al: Imaging approach to the suspected renal mass. *Radiol Clin Nor Amer* 1985; 23:503–519.

Neutropenia induced by low-dose captopril

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Captopril, the most frequently prescribed angiotensin converting enzyme inhibitor (ACE) for treating patients with hypertension and congestive heart failure, is known to cause significant side effects, which on occasion can be severe.[1,2] Neutropenia, a serious side effect of captopril use, can lead to significant morbidity and mortality[3,4,5] Captopril-induced neutropenia has previously been reported in the presence of renal dysfunction, collagen vascular disease, or at elevated dosages (>150 mg/day)[3,6,7,8]

At present, there is limited data on the relationship between low-dose captopril and neutropenia. However, there are anecdotal reports (20 cases) of neutropenia caused by low-dose captopril (75 mg/day or less) (personal communication with Bristol-Myers-Squibb, manufacturer of captopril). Therefore, we report here a documented case of neutropenia associated with the use of a relatively low dose of the medication in the absence of any preexisting renal or collagen vascular disease.

CASE REPORT

A 64-year-old woman was admitted to the hospital with fever and progressive weakness of one week duration. The patient was known to have hypertension, non-insulin-dependent diabetes mellitus, coronary artery disease, and cerebrovascular disease. There was no history of collagen vascular disease, renal dysfunction, previous blood dyscrasia, administration of cytotoxic agents, or exposure to radiation. Six weeks before admission she had been started on captopril, 75 mg/day, for hypertension. Her other medications included the following: nitrates, 5 mg patch daily; glyburide, 5 mg day; dipyridamole, 75 mg every eight hours; and aspirin, 325 mg/day. Three weeks previously she had had elective femoral-popliteal bypass for peripheral vascular disease. At that time, her white blood cell count was normal (9,300/mm^3).

The physical examination during the present admission revealed a febrile dehydrated patient with oral candidiasis. No lymphadenopathy or hepatosplenomegaly was noted. Laboratory tests revealed the following: sodium, 122 mmol/L; potassium, 5.0 mmol/L; chloride, 92 mmol/L; CO_2, 22 mmol/L; glucose, 721 mg/dL (40 mmol/L); blood urea nitrogen, 42 mg/dL (15 mmol/L); creatinine, 1.4 mg/dL (123 µmol/L). The leukocyte count was 500/mm^3 with a differential of 4% neutrophils, 88% lymphocytes and 8% monocytes. The hematocrit was 26%, and the platelet count was 171,000/mm^3. A bone marrow aspirate and biopsy revealed decreased myeloid elements with a few promyelocytes and normal erythroid and megakaryocytic elements (Fig 1).

All medications including captopril were stopped, and nafcillin, mezlocillin, and gentamicin were given intravenously. Parenteral hydration and insulin treatment were given as well. Twenty-four hours later, the leukocyte count was 700/mm^3 with 4% neutrophils and 96% lymphocytes. Glyburide was resumed two days later.

Blood cultures grew group A streptococcus. Twelve days after admission, the leukocyte count had gradually risen to 8,000/mm^3, with a differential of 19% lymphocytes, 6% monocytes, and 75% granulocytes. Follow-up electrolyte studies showed a sodium count of 133 mmol/L; potassium, 5.0 mmol/L; chloride, 94 mmol/L; CO_2, 30 mmol/L; glucose, 237 mg/dL (13 mmol/L); blood urea nitrogen, 14 mg/dL (5 mmol/L); and creatinine, 0.7 mg/dL (61 mmol/L). The patient gradually became afebrile and recovered. A normal bone marrow aspirate and biopsy were obtained three weeks after admission (Fig 2).

DISCUSSION

Neutropenia induced by low-dose captopril (<150 mg/day), in the absence of collagen vascular or renal disease, has not

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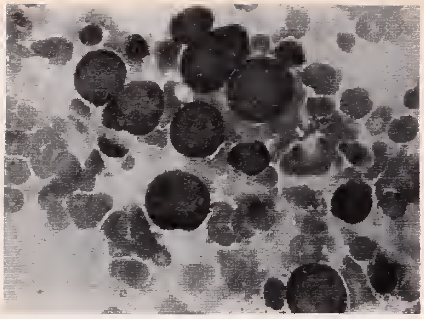

FIGURE 1. Bone marrow aspirate on admission showing early granulocytes (myeloblasts, promyelocytes, and myelocytes). The more mature granulocytes are absent. (Wright-Giemsa stain, original magnification × 100.)

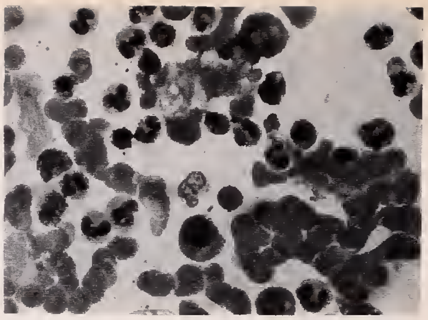

FIGURE 2. Bone marrow aspirate showing return of mature granulocytes (metamyelotes, band forms, and segmented neutrophils) 21 days after cessation of captopril. (Wright-Giemsa stain; original magnification × 100.)

to our knowledge been previously reported in the literature.

In defining captopril-induced neutropenia, we used accepted criteria.[2,6] In this case, there were two consecutive, absolute neutrophil counts less than $1,000/\mathrm{mm}^3$ with evidence of decreased myelopoiesis on bone marrow examination. There was a dramatic downward trend of the white blood cell count during the preceding three weeks of captopril usage. There was also no evidence of chronic leukopenia.

Captopril is an ACE inhibitor that possesses an active sulfhydryl group. There are two categories of captopril-induced side effects: those related to sulfhydryl moiety such as skin rash, dysgeusia, proteinuria, and neutropenia; and those related to its ACE inhibiting activity such as cough, azotemia and angioedema.[5,7,8]

Neutropenia has been noted to occur with high doses of captopril and in the setting of renal disease or collagen vascular disease.[2,3,5,6,7,9] Frohlich et al,[2] in their extensive review of the use of captopril in hypertension, found neutropenia to occur in 0.02% of all patients with intact renal function and no pre-existing collagen vascular disease. However, when these criteria were adjusted to dose, no evidence of neutropenia was seen in the group treated with 150 mg or less of captopril per day.

Glyburide belongs to the family of sulfonylurea oral hypoglycemic agents rarely known to cause neutropenia, especially when used in conjunction with the biguanide group[10] (personal communication with Upjohn Company, manufacturer of glyburide (Micronase). The continued increase of the white blood cell count in this patient in spite of resumption of glyburide was confirmed captopril as the causative agent for the neutropenia. Furthermore, the eight day period required for the white blood cell count to return to normal levels was similar to findings of previous reports of captopril-induced neutropenia.[2] Captopril was not resumed for ethical reasons.

This case report demonstrates the possibility of neutropenia caused by low doses of captopril, even without apparent risk factors. It is likely that captopril induces neutropenia in an idiosyncratic fashion, as occurs with the use of penicillamine, which also possesses a sulfhydryl moiety. The neutropenia is usually seen within the first year of treatment. Its occurrence is independent of dosage and its onset is acute.[7]

In previously reported cases of captopril-induced neutropenia, renal dysfunction, marked by a serum creatinine level of 1.5 mg/dL (133 μmol/L) or more, was a major risk factor.[2,6] In one study involving 1,720 patients in which the captopril dose was 150 mg/day or less, and the serum creatinine level was 1.5 mg/dL (133 μmol/L) or less, there was not one incident of neutropenia.[2]

Although the serum creatinine level was normal in this patient, there was an element of dehydration and prerenal azotemia, which may have contributed to the development of neutropenia by decreasing the renal clearance of captopril.

This case report demonstrates that neutropenia can occur with any dose of captopril, and suggests that pre-renal azotemia may be a predisposing factor in inducing neutropenia caused by captopril usage. We recommend frequent monitoring of blood counts and serum electrolytes levels in all patients using captopril, especially during the first 90 days of treatment.[2,6]

Acknowledgment. The authors thank Ms Patricia Murphy for her patience and support.

REFERENCES

1. Levine TB, Olivari MT, Cohn JN: Angiotensin converting enzyme inhibitors in congestive heart failure: Overview in comparison of captopril and enalapril. *Am J Med* 1986; 81:36–39.
2. Frohlich ED, Cooper RA, Lewis EJ: Review of the overall experience of captopril in hypertension. *Arch Intern Med* 1984; 144:1441–1444.
3. Suarez M, Ho PW, Johnson ES, et al: Angioneurotic edema, agranulocytosis, and fatal septicemia following captopril therapy. *Am J Med* 1986; 81:336–338.
4. Gavras I, Graff LG, Rose BD, et al: Fatal pancytopenia associated with the use of captopril. *Ann Intern Med* 1981; 94:58–59.
5. Safar ME: Fatal bone marrow suppression with captopril. *Br Med J* 1981; 283:791–792.
6. Cooper RA: Captopril-associated neutropenia: Who is at Risk? *Arch Inten Med* 1983; 143:659–660.
7. Jaffe IA: Adverse effects profile of sulfhydryl compounds in man. *Am J Med* 1986; 80:471–476.
8. Irvin JD, Viau JM: Safety profiles of the angiotensin converting enzyme inhibitors captopril and enalapril. *Am J Med* 1986; 81:46–50.
9. Staessen J, Fagard R, Lijnen P, et al: Captopril and agranulocytosis. *Lancet* 1980; 1:926–927.
10. Muller AJ: Lactic acidosis and bone marrow suppression due to oral hypoglycemic agents. *J Am Osteopath Assoc* 1972; 71:1093–1097.

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids

TO THE EDITOR: Aphthous ulcerations of the mouth are a recognized manifestation of the acquired immunodeficiency syndrome (AIDS). We report two cases of oral aphthous ulcerations in patients seropositive for the human immunodeficiency virus (HIV), who were successfully treated with metered-dose-inhaled steroids.

Case Report. Patient 1 was a 37-year-old man seropositive for HIV antibody who had been asymptomatic. His T4 cell count was $120/mm^3$; and he was being treated with zidovudine (100 mg five times daily) and trimethoprim-sulfamethoxazole (one double strength tablet twice daily). The patient had done well until he developed scattered, painful, oral ulcers. Viral and fungal cultures of superficial swabs of the lesions were negative, as were acid-fast stains and mycobacterial cultures. He was treated with oral prednisone 60 mg daily, with prompt relief of pain and resolution of the ulcers. Subsequently, the patient had several recurrences of oral ulcers, resolving with tapering doses of oral steroids. Because of concern about potential immunosuppressive effects of long-term intermittent systemic steroids, the next recurrence was treated with inhaled beclomethasone dipropionate (two puffs three times daily). Pain relief occurred within 24 hours, and the lesions resolved several days later. The patient reported that the relief was as rapid and as complete as had been obtained with prednisone.

Patient 2 was a 32-year-old hemophiliac who is seropositive for antibodies to HIV. He has a history of *Pneumocystis carinii* pneumonia diagnosed by bronchoalveolar lavage, and has been treated with zidovudine, 100 mg five times daily and trimethoprim-sulfamethoxazole (one double strength tablet twice daily). The patient developed scattered, painful, oral ulcers treated with beclomethasone dipropionate (two puffs three times daily). The pain resolved in approximately 24 hours, and the lesions were healed one week later.

Discussion. Oral aphthous ulcers are a common occurrence in both immunocompetent and immunocompromised patients. In immunocompetent patients, the lesions tend to be self-limited and benign.[1] In patients with AIDS, the lesions may be more severe and progressive. Bach et al[2,3] reported a total of nine patients with AIDS who developed severe aphthous ulcerations involving the mouth, hypopharynx, esophagus, and colon. Eight of the nine responded promptly to prednisone therapy.

Phelan et al[4] reported nine patients with AIDS who had persistent, painful, oral ulcers. The patients were successfully treated with topical tetracycline and steroids or topical steroids alone (one patient).

We have reported two HIV-seropositive patients with oral ulcers who were successfully treated with inhaled steroids. Although systemic therapy may be needed for diseases involving the lower gastrointestinal tract,[2,3] local therapy offers a significant advantage for lesions involving the mouth and hypopharynx alone. Local treatment can avoid immunosuppressive effects associated with systemic steroids. When given via a metered dose inhaler, steroids are applied directly to the oral mucosa. Only 10%–25% of an inhaled dose of beclomethasone dipropionate actually reaches the bronchial tree; the remainder is deposited in the mouth and throat or swallowed.[5]

Inhaled steroids offer several advantages over topical medications for oral lesions. Oral ulcers may be in areas where is difficult to apply medication. It is often difficult and inconvenient to apply medication directly to an oral lesion, especially if a mirror is not available. Inhalers are also easier to carry and much more convenient to apply to the oral mucosa.

Inhaled steroids should be considered as an alternative to topical or systemic preparations for the treatment of oral ulcers in HIV-seropositive patients.

The authors wish to thank Mrs G. Rich for her secretarial work in the preparation of this manuscript.

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2. Bach MC, Valenti AJ, Howell DA, et al: Odynophagia from aphthous ulcers of the pharynx and esophagus in the acquired immunodeficiency syndrome (AIDS). *Ann Intern Med* 1988; 109:338–339.
3. Bach MC, Howell DA, Valenti AJ, et al: Aphthous ulceration of the gastrointestinal tract in patients with the acquired immunodeficiency syndrome (AIDS). *Ann Intern Med* 1990; 112:465–467.
4. Phelan JA, Eisig S, Freedman PD, et al: Major aphthous-like ulcers in patients with AIDS. *Oral Surg Oral Med Oral Pathol* 1991; 71:68–72.
5. United States Pharmacopeial Convention. Drug Information for the health care professional. Kingsport TN: Arcata Graphics, 1990; 42–44.

Hyperbilirubinemia in the term infant

TO THE EDITOR: I thought Dr. Johnson's article on hyperbilirubinemia in the term infant in the November 1991 issue of the *Journal* was one of the finest summaries of the problem that I have read.[1] The literature survey was particularly good while the three cases of kernicterus in term babies with bilirubin levels of 30 mg/dL or under and the several other children with rapid bilirubin rises were particularly unsettling. However, I was unable to determine what the risk is for these untoward events. Were all seven children in Table I delivered at Pennsylvania Hospital? Over what period of time were they collected? How many deliveries a year are seen at Pennsylvania Hospital? These data would allow us to estimate the risk for one of these toward events occurring. As an illustration, if these cases were collected over five years at one hospital averaging 3,000 term births per year, the risk of an untoward event as described in the paper would be 7/3,000 × 5 = 7/15,000 or approximately one in 2,100 live births. If on the other hand, they were collected over ten years from 100 similar hospitals, we are talking of a risk of one in 430,000. For the practitioner, the risk factor has to be balanced against such other factors as the cost of bilirubin testing, the inconvenience of repeat bilirubin testing, the risk of nosocomial infection for children staying longer in the hospital, and the decrease in the number of mothers successfully nursing because of anxiety and concern associated with bilirubin testing.

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In reply. I thank Dr Joseph for his kind letter and the opportunity to address the question of incidence of classical sequelae of neonatal jaundice among healthy-term and near-term infants.

There is a general consensus that such sequelae are very rare, but how rare, we do not know. The requisite numerators and denominators simply are not available. The seven infants described in Table I of my paper were chosen to be representative of the outcomes seen in such term and near-term infants without Rh hemolytic disease. As shown by these examples, they range from an apparent absence of any central nervous system (CNS) deficit to classic signs of kernicterus. Not all seven infants were cared for at Pennsylvania Hospital. However, except for infant no 1, who was also the only infant discharged from the hospital after day two, all were born since 1985.

We obviously need more information on this increasingly timely subject. To answer the pertinent questions over a reasonable period of time and at a reasonable cost will probably require the cooperative efforts of practicing pediatricians, coordinated through local chapters of the Academy of Pediatrics, because the sample size needed for defining numerators and denominators for the general population and its sub-groups will be very large. Such investigations will need to address the incidence of jaundice above levels of 15, 20, and 25 mg/dL in relation to the incidence of sequelae, defined, at a minimum, by Baer, neurologic examination, and Bayley scores at age one year. The occurrence of jaundice should also be related to ethnic group, type of labor and delivery, approximate frequency, type and volume of feeding, before and after hospital discharge, and if feasible, such parameters as albumin and pH.[1]

With regard to the sequelae of importance, however, it is not at all clear to me that we need be concerned only with CNS insults which fit the definition of classic kernicterus. Jaundice-associated motor deficits found at age one year,[2] but no longer detectable by age four to seven,[3] are still an indication of insult. The absence of functional compromise at an older age testifies to the ability of the young child to compensate for early modest CNS insults. Does such a child have the same reserve as his undamaged counterpart and the same ability to withstand subsequent destabilizing events? When we speak of transient damage and threshold phenomenon, we need to think not only of the whole organism, but also of the cells, tissues, and organs which make up the whole. In our study relating binding and bilirubin levels to outcome, we found that jaundice-related suspect performances on the four and seven year speech, hearing, language, and psychometric exams were more common among children, with a family history of speech and learning disability.[4,5,6] As Dormandy has pointed out in his classic article on free radicals and antioxidants;[7] "Butter, to which sufficient antioxidant has been added to preserve it for a hundred days, tastes as buttery on the 99th day as it did on the first; but it is not the same butter." In this sense, the child with modest genetic or acquired cellular compromise has a decreased defensive reserve even though his performances fall within the means for the population.[1,8–10]

Related to this line of reasoning is the report of Seidman et al on 17-year outcome data among term infants born in Israel in 1971. This study begins to let us address the question of minor as well as classic sequelae of neonatal jaundice.[11] In this report, the incidence of IQ scores below 85 was found to be significantly increased among term male infants without hemolytic disease whose serum bilirubin levels in the newborn period had exceeded 20 mg/dL. I have discussed this paper, as well as those of Day et al among Rh-sensitized versus non-sensitized sibling pairs,[12,13] in a commentary on the paper by Newman and Maisels entitled "Evaluation and treatment of jaundice in the term infant: A kinder, gentler approach."[14] Its concluding paragraph is as follows: "If one accepts the premise that a downward shift in IQ scores can be taken as an end point, then an IQ above 85 (or, for the matter, above 120) is desirable and that each child has at least the semblance of a loving home in which to develop and use his intelligence, then the report of Seidman begins to address the issue of numerators and denominators. It suggests (given the skewness of the confidence limits around the found odds ratio) that, in a male population with an 8% incidence of severe jaundice, something in the range of 1 in 200 infants would have a threefold increase in likelihood of having an IQ below 85 if the neonatal bilirubin level exceeded 20 mg/dL. If this is so, close monitoring of bilirubin levels in this subset of infants is worth the effort, cost, and discomfort."

I would further submit that jaundice management need not necessarily compromise breast feeding. The mother-infant pairs most at risk for breast-feeding problems are also those most at risk for prolonged, exaggerated jaundice. Both situations may be helped by reassurance, counseling, and temporary formula supplements. It is simply not true that supplemental feedings necessarily lead to unsuccessful breast feeding.[6,15] The latter may as much related to the stigma attached, in some circles, to anything "artificial."[16,17] It is also not true that respect for the toxic potential of bilirubin necessarily leads to parental over-concern and the vulnerable child syndrome. This entire area needs to be addressed within the context of optimal, wholistic pediatric care. The loss of time for individualized counseling and support, imposed by early hospital discharge, should be supplanted by outpatient teaching by the nurse, physician assistant, or doctor, in the office, home, or by phone. During this exchange, the practiced eye (or ear) can identify the likelihood of greater degrees of jaundice, as well as of other potential problems, and institute appropriate management.

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1. Wennberg RP: Cellular basis of bilirubin toxicity. *NY State J Med* 1991; 91:493–496.

2. Scheidt PC, Mellits ED, Hardy JB, et al: Toxicity to bilirubin in neonates: Infant development during the first year in relation to maximum neonatal serum bilirubin concentration. *J Pediatr* 1977; 91:292–297.

3. Rubin RA, Balow B, Fisch RO: Neonatal serum bilirubin levels related to cognitive development at ages 4 through 7 years. *J Pediatr* 1979; 94: 601–604.

4. Johnson L, Boggs TR: Bilirubin-dependent brain damage: Incidence and indication for treatment, in Odell GB, Schaffer R, Simopoulous AP (eds): *Phototherapy in the Newborn: An Overview.* Washington, DC National Academy of Sciences 1974, pp 122–149.

5. Johnson L, Winchester R, Akins T: Central hearing and communication disorders and hyperbilirubinemia. *Pediatr Res* 1990; 27:210A.

6. Johnson L: Hyperbilirubinemia in the term infant: When to worry, when to treat. *NY State J Med* 1991; 91:483–489.

7. Dormandy TL: Free-radical oxidation and antioxidants. *Lancet* 1978; 647–650.

8. Shaywitz SE, Escobar MD, Shaywitz BA, et al: Evidence that dyslexia may represent the lower tail of a normal distribution of reading ability. *N Engl J Med* 1991; 326:145–150.

9. Needleman HL, Leviton A, Bellinger D: Lead associated intellectual deficit. *N Engl J Med* 1982; 306–367.

10. Bratlid D: Bilirubin toxicity: Pathophysiology and assessment of risk factors. *NY State J Med* 1991; 489–492.

11. Seidman DS, Paz I, Stevenson DK, et al: The effect of phototherapy for neonatal hyperbilirubinemia on long-term cognitive performance. *Pediatr Res* 1991; 29:265A.

12. Gerver JM, Day R: Intelligence quotient of children who have recovered from erythroblastosis fetalis. *J Pediatr* 1950; 36:342–348.

13. Day R, Haines MS: Intelligence quotients of children recovered from erythroblastosis fetalis since the introduction of exchange transfusion. *Pediatrics* 1954; 13:333–338.

14. Johnson L: Yet another expert opinion on bilirubin toxicity! *Pediatrics* 1992; 89:(In Press).

15. Gray-Donald K, Kramer MS, Munday S, et al: Effect of formula supplementation in the hospital on the duration of breast-feeding: A controlled clinical trial. *Pediatrics* 1985; 75:514–518.

16. Buchsbaum S: The bottle battles: Why moms are feuding over feeding? *Child* 1992; Feb: 75–83.

17. Jason J: Breast-feeding in 1991. *N Engl J Med* 1991; 325:1036–1038.

In lieu of a plaque

TO THE EDITOR: About years ago, I entered the very modest lobby in a small hospital in Brooklyn located across the street from Prospect Park, one of the most beautiful urban parks in the United States of America. I was a shy, young doctor who had just finished an anesthesia residency program and was starting my first job as an attending anesthesiologist in this hospital, which proudly called itself "the hospital by the Park." When I looked around, I saw a tall man, much over 200 pounds, trying to squeeze himself through the revolving door.

"What are you looking for?" he asked, sensing my helplessness.

"I am a new anesthesiologist," I replied. A bright smile lightened up his handsome face which somehow didn't match his large mass.

"Oh yes, we are waiting for you," he said.

So much warmth was in his voice that I felt at home, even in that drab lobby, full of cigarette butts on the yet uncleaned floor. Dr Raymond Kaplow, the man I had just met, lived across the street from the hospital. However, in real life, he lived in the hospital. He was a general surgeon with a special interest in orthopedic surgery which he performed with gusto. I wondered how he ever operated with so large a frame. However, I soon observed that his big abdomen simply disappeared under the table, whereas his hands were transformed into delicate instruments. He was never too proud or too shy to ask for help from a more experienced colleague. Although he raised some eyebrows by asking for help, I respected him most for realizing his professional limits. The specialists he called on were sometimes angry with him because his patients were often poor and could not pay their fees. We anesthesiologists spent many sleepless nights with him in the operating room, nights rich in the emotions of challenge, frustration, exhaustion, defeat, or hardly won triumph. After, there were many "pro bono" nights, while life and death changed hands in a heartbeat and our coronaries constricted with every fall in patient's blood pressure. We celebrated our victories in the cafeteria, forcing ourselves to keep our eyes open for the morning schedule.

His patients loved him, for he was their best friend. In spite of his large size, he was a fragile human being, often complaining of his own ills while taking patients' medical histories. He complained to them about his excessive weight and an inability to control his appetite. He had decided long ago to make the hospital his home and patients his immediate family. Sometimes I took calls from his wife. It was not easy for her to parent three children with an absent husband.

We followed him to the Plaza Hotel, where he organized the hospital's annual dinner-dance and were amazed by his graceful dancing. He always sent plants to the sick members of the medical staff, accompanied by a comforting and cordial note. When his wife died, he was unable to cope, and his personal and professional lives became difficult.

Newly trained, young, orthopedic physicians joined the staff, and new rules forced the administration to remove him from the orthopedic survice. The day the medical board dismissed him from orthopedic surgery was an extremely sad one in all our lives. In time, our hospital changed slowly into an inner city one with many empty beds. Many doctors gradually left the hospital on the Park, and the streets we walked on became full of broken glass from crack cocaine pipes. In spite of these changes, Raymond Kaplow remained faithful to the hospital. Eventually, he had to sell his house for he could not safely cross the street on his way to the hospital let alone live in the house. He moved from Brooklyn out to Queens, meaning longer hours in the hospital with little rest at home.

He often demanded, "Give me the best anesthesiologist for my very sick patient."

"But they are all good, Raymond," I would answer indignantly.

"Yes, but some are better than others," he would reply.

Well, he was right for he was the best on my list of the good surgeons.

One winter night, while driving home from the hospital in his specially built spacious car, he was killed on one of the S turns of the Interboro Parkway. At his wake, hundreds of his patients looked like fatherless children. I looked at his handsome face and while breathing the odor of the funeral flowers, I thought that if he had to die, the operating room would have been a more proper place.

A few years after his death, our institution merged with a larger hospital because of financial reasons. Long-time staff were dismissed or shuffled around. Some died, others retired, and very few now remember the surgeon who was once the spirit and mover of the hospital. I asked an old timer to put up a little plaque in memory of this exemplary, old fashioned doctor who had also left a substantial legency for the new hospital building.

"Impossible," he said. "The new administration doesn't know of Kaplow and has no loyalities toward the old hospital. Our hospital is considered an outside camp and as in the death camps if one dies, he is only remembered by the survivors."

And so it is for you, Raymond, in lieu of a plaque.

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FROM THE LIBRARY

RECONSTRUCTION OF THE THUMB

It is interesting to note that of all mammals, only the primates have opposable thumbs. In all other groups, the thumb is either absent or rudimentary in character. Fingers may be very nimble, but grasp is accomplished by opposing against a pad in the palm of the hand. A very definite advantage of a primate thumb, particularly in man, is the ability to grasp between the thumb and fingers in a position well away from the palm of the hand. This single ability tremendously increases the possibility of detailed and refined activities. In removing detailed activities from the palm, the thumb is as important to the hand as the arm is in removing the hand from the body.

There are four methods of reconstructing a thumb, which depend upon conditions present in the remaining portion of the hand. The first and simplest is that of deepening the web betwen the thumb and the palm of the hand. This is particularly useful in mangled hands where there is loss of the fingers and the thumb, or a tremendous shortening of the digits. A progression of the above clefting is carried out in phalangization of the metacarpals. As a rule, only two metacarpals are phalangized, and to many hand surgeons this seems adequate. There are cases reported in which all five metacarpals have been phalangized, but this seemingly inhibits rather than promotes function.

The second, and probably most frequent method of restoring a lost thumb, is that of constructing a tube pedicle on the abdomen and transplanting it to the site of the proposed thumb. Of course the extensiveness of this procedure is dependent upon the site of amputation.

The third, and rather popular but more difficult method, is pollicization of the index finger to replace the missing thumb.

The fourth method is that of replacing the thumb with a digit from the opposite hand or with a toe.

The most frequent total loss of the thumb and fingers occurs in severe frostbites. It is less commonly found in congenital deformities, severe burns, and in severe crushing wounds. A hand of this type has no more function than that of a forefoot of a dog or bear. These animals are limited in picking up and rolling objects between the paws. In this type of hand there are excellent possibilities for producing a grasping and pinching member.

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(*NY State J Med* 1949; 49:49–50)

Reprinted from the September/October 1991 issue of Epidemiology Notes (*Vol 6, No 8*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Overweight in New York State

Overweight is a common condition often implicated in the etiology of other medical problems including cardiovascular disease, diabetes and some cancers. Although obesity may arise from individual medical conditions, most obesity is a consequence of personal, preventable behaviors.

During 1988 and 1989, the New York State DOH measured the prevalence of overweight in New York State through telephone interviews conducted during the annual Behavioral Risk Factor Survey. The Behavioral Risk Factor Surveillance System (BRFSS) collects data used to estimate the number of adults who are at risk for poor health outcomes associated with personal behaviors. Thirty-seven states participated in the 1988 and 1989 BRFSS funded and coordinated by the US Centers for Disease Control. The BRFSS used standard questionnaire methods to conduct monthly random-digit-dialing telephone interviews to adults 18 or more years of age during 1988 and 1989.

The BRFSS measured the risk of overweight in New York State residents by requesting a respondent's weight and height without shoes. This information was used to calculate a measure of overweight known as the body mass index (BMI). BRFSS respondents were considered overweight if the calculated BMI value was greater than the 85th percentile of US adults aged 20–29 years as estimated from the Second National Health and Nutrition Examination Survey.

Previous BRFSS reports of overweight in the United States have shown that New York State ranks at approximately the median point of overweight.[1,2] Recently collected BRFSS data indicate that New York State's position relative to other states has not changed with 25.1% of all New York respondents defined as overweight in the 1988 survey and 23.1% of respondents defined as overweight in the 1989 survey (Table 1). These results are compare with median US proportions of 25.1% and 24.5% overweight during 1988 and 1989, respectively.

Although males appear to have a consistently higher proportion of overweight than females (26.7% vs 23.6%) and (24.6% vs 21.9%) in 1988 and 1989, these figures may be misleading because female respondents were twice as likely as male respondents to refuse answering height and weight questions. Among women, 5.8% did not respond compared with 2.6% of men. Other important relationships include increasing prevalence of overweight with age, increased prevalence among non-white respondents, and declining prevalence with increasing level of education and employment.

TABLE I. BRFSS 1988 and 1989

Characteristic	1988		1989	
	Obese* (%)	C.I.** (±)	Obese (%)	C.I. (±)
Total	25.1	3.7	23.1	3.4
Sex:				
Male	26.7	5.8	24.6	5.4
Female	23.6	4.8	21.9	4.3
Age:				
18–24	15.9	9.9	10.0	7.7
25–34	15.1	6.5	18.3	6.4
35–44	24.9	8.2	21.0	7.0
45–54	31.7	10.9	34.6	11.3
55–64	38.3	11.0	33.4	9.9
65+	30.2	8.8	25.7	8.4
Race/Ethnicity				
White	24.2	4.1	23.4	3.8
Nonwhite	28.7	8.6	22.0	7.8
Education				
<High School	31.9	4.8	31.0	9.0
High school graduate	26.6	7.4	25.1	6.2
Some college	23.1	16.6	20.8	7.7
College Graduate	20.4	9.8	18.5	5.5
Marital Status				
Married	27.6	5.4	25.2	4.9
Sep/Div/Wid	26.6	6.9	26.2	6.6
Never Married	17.0	7.3	15.5	6.2
Income				
<$15,000	28.8	8.3	31.5	8.6
$15,000–$24,999	26.4	9.2	20.2	7.9
$25,000–$34,999	26.6	10.6	24.3	9.1
$35,000+	24.5	5.7	22.2	6.3

*% obese represents all responses to both height and weight.

**Exact 95% confidence intervals are presented for the categories "total" and "sex." All other 95% confidence intervals are estimated.

A national strategy for improving the health of the nation has been formulated and published by the US Public Health Service as a document titled *Healthy People 2000: National Health Promotion and Disease Prevention Objectives.*[3] Objectives established by this planning document target overweight as a major risk-reduction objective with a goal to reduce overweight to a prevalence of no more than 20% among people aged 20 years and older. *Healthy People 2000* contains a number of recommendations for achieving this objective, including a reduction of dietary fat and increase in dietary fiber from a variety of foods such as vegetables, fruits, and grains. For adults who are overweight, achieving this objective will require dietary calorie restriction that is accompanied by increased physical activity. Future public health efforts will focus on preventing development of overweight and encouraging and facilitating weight reduction.

REFERENCES

1. The 1986 State Behavioral Risk Factor Surveillance System Coordinators. Prevalence of overweight in selected states—behavioral risk factor surveillance, 1986. *Chronic Disease and Health Promotion Reprints from the MMWR, 1985–1989: Volume 3 Behavioral Risk Factor Surveillance System.*
2. The 1987 State Behavioral Risk Factor Surveillance System Coordinators. Prevalence of overweight in selected states—behavioral risk factor surveillance, 1987. *Chronic Disease and Health Promotion Reprints from the MMWR, 1985–1989: Volume 3 Behavioral Risk Factor Surveillance System.*
3. *Healthy People 2000: National health promotion and disease prevention objectives* DHHS Publication No. (PHS) 91-50212, 1991.

JEWISH VALUES IN HEALTH AND MEDICINE

Edited by Rabbi Levi Meier, PhD. 201 pp, Lanham, Md, University Press of America, 1991. $16.75 (softcover)

Experienced physicians come to understand that the day-to-day practice of medicine is a complex and challenging endeavor, filled with overt and subtle demands on their intellectual, emotional, and ethical skills. Physicians must develop a foundation of consistent values and ethical beliefs on which they can rely. What assistance can such physicians find in this ongoing process? One invaluable resource is close attention to the patients themselves, allowing them to teach us what they need, how they are best served, and what their experience has really been. A second major aid is to listen to the thoughts of others who have spent a great deal of their time and intellectual energy considering these matters. These should include not only those with a strictly modern-day perspective, but also those with a more historical, philosophical, and religious background, since it is clear that physicians have been confronting the same basic issues since the dawn of medicine.

The Jewish religion, in particular, has a long, rich tradition that carefully considers the role and responsibility of both physicians and patients. Rabbi Levi Meier, the chaplain at Cedars-Sinai Medical Center and a clinical psychologist, has produced a brief but thorough collection of the Salick lectures on ethical issues in medicine; it will well serve as a valuable and provocative work for those who find themselves ever-searching for their own personal foundation of ethics in medicine, whether they be Jewish or not.

Part One was written by patients, who chronicle, in very intimate ways, their own experiences. The view of illness that emerges is a highly personal and private odyssey filled with great emotion and sensitivity. The nearly overwhelming sense of vulnerability and dependence patients feel comes through again and again. It is clear that the impact of a serious illness can be profound and that the consequent healing process can be life-long. Physicians will do well to listen carefully to the expressed perceptions of the doctor-patient relationship. Rabbi Schulweis comments, "What have they in common, the doctor and the patient? Most important, the healing and liberating knowledge that in every doctor there is a patient and that in every patient there is a doctor."

Part Two contains six essays addressing the foundations of medical ethics from a Jewish perspective. Rabbi Meier succinctly elucidates the three major principles of Jewish medical ethics. This is followed by the reflections of several noted physicians on the proper role and behavior of a physician on the basis of these principles. A conceptualized approach to such areas as terminal and hospice care is discussed.

Part Three specifically addresses the actual clinical problems we might confront, such as AIDS, experimental therapies, terminal care and surrogate parenting. Thus, in only 194 pages, Rabbi Meier has managed to present a broad range of perspectives on medical ethics, with a focus on the interaction between Jewish values and tradition and modern medicine. The practicing physician will find this collection more readable and to the point than many works in this general area; certainly it will be of great aid to those seeking some guidance from their Jewish heritage as to how to cope with today's ethical dilemmas and challenges.

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CLINICAL DERMATOLOGY

By Rona M. MacKie, MD. 364 pp, illustrated. New York, Oxford University Press, 1991. $29.95 (softcover)

The third edition of *Clinical Dermatology*, formatted as a *vade mecum* for the "white coat pocket" of the medical student and lower-level resident, attempts to cover the major areas of dermatology in 13 chapters with additional sections on the approach to the dermatology patient, basic cutaneous biology, and topical therapy. Distinctly British in its spellings and terminology, this readable text will confront and amuse many American students on their first exposure to "clamant itch," "blanket baths," an infant "swathed in damp napkins," a "keen" gardner, the "unfit" with herpes zoster, "basal cell papilloma" for seborrheic keratosis, and "cheiropompholyx."

Unfortunately, the text can be faulted throughout for its omission of key disease entities and therapies, inconsistent use of illustrations, and confusing therapeutics. Several obvious basic omissions include the failure to mention ecthyma, folliculitis, and furuncles with infections; space allotted to cutaneous tuberculosis without note of atypical mycobacterial infections; discussion of tinea pedis limited to the interdigital presentation without allusion to the moccasin scale and vesiculobullous variants; absence of lamellar ichthyosis and epidermolytic hyperkeratosis with ichthyosis. The American dermatologist does not usually associate tinea pedis with communal showers, does not extract the scabetic mite with a needle, uses viral cultures to identify and distinguish herpes simplex and zoster, and should always do a fungal culture in addition to a potassium hydroxide examination for certain dermatophyte identification.

Both the quality and number of illustrations are inconsistent. I found the eyeblack on the photos a distraction and a detractor from disease appreciation. The blepharitis noted in the caption of Figure 5.8 is obscured by the eyeblack. Several illustrations, (eg, tuberculosis leprosy and plane warts) provide no hint of body site, while other prints are too distant and not adequately focused to appreciate specific cutaneous changes (eg, SLE and contact dermatitis). The common and prototypical rhus contact dermatitis is conspicuously absent from the British text and the figures of patch testing fail to show the vesiculation of a positive patch test. The illustrations of before and after 4-months of acne therapy reflect poorly the dermatologist's therapeutic skills. Finally, I question the selection of photos of the same patient to demonstrate both urticaria and urticaria secondary to penicillin.

Approaches to therapy differ across the ocean (eg, the "puncturing of individual mollusca" to "stimulate an inflammatory reaction") and bed rest for herpes zoster. Topical agents such as 10% ichthyol in H_2O are generally not current in present American therapy. British medications are named throughout the text without identification of American counterparts. The table of topical steroid classification is not only incomplete and outdated, but again limited to generic and British product identification.

The chapter on history taking and laboratory investigation makes good points but lacks organization and interjects confusing clinical statements with method (eg, "mild itch" associated with drug eruption and "pain. . .a symptom of vasculitis and pemphigus.")

What the student needs in a pocket edition is a clear, concise outline for history taking and a reproducible approach to the examination and description of cutaneous changes. All descriptions must be accompanied by an illustration. An atlas format with a crisp illustration accompanying every precise and pithy word description would familiarize the neophyte with the classic morphologies of dermatologic diseases. Armed with this basic information, the student can then be directed to a larger text or a readily available literature search for in-depth study. *Clinical Dermatology* does not meet the requirements for either "an illustrated textbook" or a primer for the budding dermatologist.

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AIDS PREVENTION AND TREATMENT: HOPE, HUMOR, AND HEALING.

Edited by M. Ross Seligson, PhD, and Karen E. Peterson, PhD. 276 pp. Hemisphere Publishing Corp, 1991. $29.95 (softcover)

As the editors state in the preface, this book was written to provide information to laypersons and professionals who are interested in working toward ameliorating the AIDS crisis. The book is meant to educate and provide specific guidelines and educational outlines. The authors willingly share their experience in educational and psychological support efforts aimed at preventing further spread of the virus and improving the quality of life of infected individuals and those around them.

Both editors have PHD and have extensive experience with HIV-infected individuals. They are psychologists by training and are actively engaged in educational programs dealing with the book's topic. The experience and educational backgrounds of the other contributors is not described, but the writing style makes it clear that all have intimate knowledge of and experience with HIV-positive patients.

The book contains ten sections and an appendix written by a group of contributors. The chapters are divided into three major sections: Prevention and Treatment, Specific Populations, and Social Policy. The sections vary in the number of chapters, the comprehensiveness of coverage, and the objectivity of style.

The chapters themselves are interesting, and sometimes riveting, being amalgams of scientific information, anecdotal observations, and social opinions. Issues of psychotherapy and supportive techniques are well covered and there are many useful hints for anyone who is in contact with persons who are HIV positive. There is a discussion of the appropriate use of humor as a therapeutic technique. This discussion contains some convincing arguments, which might sway the most cynical medical professional. There is also an element of flexibility built in to all the suggestions. This implies that the authors as a group are not dogmatic and appear to be both sincere and humanistic in their approaches.

The style of the text makes for easy reading, although the interspersal of frequent reference annotations directly in the text using the author and the year of publications, does make for some difficulty in following the print. Some discussions are superficial and the reader wishes for more details or examples. Saying that "...research suggests that the immune system and the psyche interrelate" and not providing any further discussion or clarification leaves the reader at the edge of a cliff. The book is progressive in its suggestions, vigorously supporting needle exchange programs and occasionally getting preachy. It uses phrases such as "Now is the time for all good administrators to come out of the ivory tower. AIDS is a real phenomenon, and is not going away overnight." In addition, there are some medical simplifications that border on inaccuracies, such as the description of a wet mount preparation of an oral specimen for *Candida albicans* as a "swab" rather than a scraping. The inaccuracies are minimal, however, and every medical professional, no matter how learned, can appreciate many of the thoughts and ideas in this book. Occasionally, discussions are very simplified, as would be expected in a text written for both laypersons and medical professionals. The effort to use understandable terminology should not affect the credibility of this book.

The discussions of social supports, central nervous system (CNS) complications, and cognitive rehabilitation suggestions are very helpful. Thoughts on assisting with grief of both the patient as well as the survivors are to the point and helpful. The appendix provides very useful outlines for educational programs related to prevention as well as psychological adaptation to infection. I have seen no book that so successfully provides useful information about HIV infection to both the layperson and the medical professional. This book is easy to read and provides the reader of any educational background with food for thought, as well as succinct practical suggestions for prevention and management. I would recommend this text to anyone interested in learning from an intelligent, progressively oriented group of competent behaviorists and scientists. The medical professional may find some of the text simplistic, but will be frequently startled by valuable observations and thoughtful suggestions. The format allows for easy reading and makes the difficult and sometimes overwhelming subject of AIDS treatment seem more manageable and fulfilling.

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PSYCHIATRY SPECIALTY BOARD REVIEW

By William M. Easson, MD, and Nicholas L. Rock, MD. 160 pp. New York, Brunner/Mazel Publishers, 1991. $19.95 (softcover)

This Psychiatry Specialty Board Review text was put together by two senior faculty from the Louisiana State University Medical Center. It is divided into 11 chapters, the first nine of which involve the various categories of psychiatry. The final two chapters have questions from case studies and a mock examination.

The questions presented cover a diverse area of psychiatry, and the material presented in the answers is informative but brief. To those taking the specialty boards in psychiatry, this type of book may offer some minimal assistance. However, the better way to review for the board examination is to study a more lengthy psychiatric text.

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HEALTH CARE OF THE AGED: NEEDS, POLICIES, AND SERVICES

Edited by Abraham Monk. 183 pp. New York, Haworth Press, 1990. $22.95 (hardcover)

This volume represents the collective work of its editor, Abraham Monk, and other health care professionals. Their chapter discuss current health care issues facing today's elderly, new governmental regulations and their potential effects, and health care models designed to care for this population. This book is part of the *Journal of Gerontological Social Work* series.

The editor and contributors are all recognized authorities in the area of social work and gerontology. Dr Monk is professor of Social Work at the Columbia University School of Social Work in New York. The other authors are working in academic centers or serve as directors of health care services for the elderly.

Because of the complexity of providing needed services to this segment of the population, there is no one model of care that would fit all communities. Funding sources are rapidly changing and as a result, the viability of any health care program is always in jeopardy. Certainly, innovative ideas are needed more than ever to continue providing important services to older adults in need. *Health Care of the Aged,* accomplishes this task in an average fashion. The contributing authors describe and compare various models for acute and long-term care that exist across the country. The topics that Dr. Monk selected do not focus on one select portion of the older adult population, but rather the examine issues for the well, fully functional adult as well as the frail elderly person.

This reviewer believes that the book is well balanced in terms of writing styles and format of presentation of materials. The book does not contain an index of key words and topics, but does have a fairly detailed table of contents for each chapter. There are adequate bibliographies and reference sections. Detracting from the overall editorial presentation is a below average production of the book and variability of typeface, especially for the tables in each chapter.

Certainly the social work disciplines are leading the research effort in gerontology. There is an interesting number of gerontology publications that appear similar in format and content. The repetition ultimately will detract from the significance of publications such as this book. This is not to denigrate Dr. Monk's book; one of the strengths of this publication is the editor's and contributing authors' attempt to clarify many of the current trends in funding of geriatric programs, especially for catastrophic health care insurance. The first two chapters of this book present very concise summaries on health care economics, DRGs, and political issues of aging.

Health Care of the Aged: Needs, Policies and Services is recommended for health care professionals who are involved in program planning in various health care settings. It will have limited appeal to those who deliver direct clinical services to the elderly.

Unfortunately, experts such as Dr Monk can only speculate on the future of the health care delivery system for the elderly. We are all involved in a health care crisis that threatens the viability of our current programs and ultimately the health of ourselves and our patients. Hopefully, our social work colleagues and those in other medical disciplines will soon work more closely together to continue and improve on our current level of care to this growing and sometimes vulnerable segment of the population.

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FACILITATING SELF CARE PRACTICES IN THE ELDERLY

Edited by Barbara J. Horn, PhD, RN. 185 pp. Binghamton, NY, The Haworth Press, Inc, 1990. $24.95 (hardcover)

Facilitating Self Care Practices in the Elderly is an interesting publication dealing with patient education. The issues discussed are limited to educational programs in the home setting. The editor, Barbara J. Horn, RN, PhD, presents the results of a three year study, incorporating the Delphi process, of the identification of clinical problems observed by 178 Medicare-certified home health agencies in Alaska, Idaho, Montana, Oregon, and Washington. The goals of this study were to identify the impact of early hospital discharge on various populations of older adults, and then to develop approaches for managing critical clinical problems identified by home health care agencies. The three topic areas presented in this book are the most critical of the issues found in this study.

The chapter dealing with medication compliance for the home care client certainly is one that all clinicians will agree is sometimes the most difficult. The authors of this section review basic physiological considerations of pharmacology, the problems of polypharmacy, and how these issues relate to compliance issues. Most of the chapter deals with a process of problem identification, implementation of plans to correct or modify the problems of medication errors, and methods to assess the effectiveness of plan. This process is present in all the chapters.

The other issues covered in this book are teaching the older adult in the home setting and family coping. The section on teaching the elderly at home contains many helpful teaching aids and diagrams that could be used by many clinicians. There is a helpful reference list of publications on this area and agencies that provide educational materials.

I believe that the chapter dealing with helping the family cope with the impaired older adult is the least effective of the entire book. The authors do not present any approaches that the clinician would find helpful in dealing with individual problems. Psychological impairment may also be the most difficult problem in keeping the person in the home setting and requires solutions that exceed the scope of this book.

There have been many books and articles on patient education. I do not believe that there are any new areas discovered in this study. I do believe that it is helpful for physicians to look at the various processes that our nursing colleagues used in problem identification and problem solving. The method presented in this publication could be used by other medical and social work disciplines in their research areas.

I believe that the book is well balanced in terms of writing style and presentation. There were inconsistencies in typeface and presentation of the data tables that detract from the overall attractiveness of the publication. Each chapter has adequate bibliographies and reference sections. There is no key word or topic index.

I have mixed feelings about the overall recommendation of this publication to the readership of the *Journal*. I do not think that there are any new of unique areas discovered in this study. At the same time, I think that it does effectively summarize many of the issues dealing with patient education in general.

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CURRENT PRACTICE OF INTERVENTIONAL RADIOLOGY

Edited by Saadoon Kadir, MD. 779 pp, illustrated. Philadelphia, BC Decker Inc, 1991. $149.00

This is an ambitious attempt to define the state of the art in vascular and nonvascular radiologic interventions. The text uses contributions by 180 authors to address vascular topics from angioplasty to Z-stents, as well as biliary and renal interventions, abscess drainage, biopsies, tubal recanalization, and a host of other therapeutic interventions.

An initial section on principles of interventional techniques includes appropriate material on organization of an interventional practice, with its requirements for dedicated space and equipment, support personnel and hospital in-patient services. Basic techniques are well described. The remainder of the text organizes vascular and nonvascular interventions by anatomic region, a structure which initially seemed cumbersome, but ultimately works well.

Doctor Kadir is remarkably successful in dealing with such a broad and diverse field. The numerous topics are dealt with in crisp and concise chapters written by a group of national and international authorities. The emphasis is on "how I do it," with the inclusion of specific procedural details not often provided in textbooks. This is certainly to the reader's benefit, although alternative procedural approaches are not always discussed. Traditional referencing is replaced by a list of suggested readings at the end of each chapter.

In spite of the number of contributors, there is little duplication. Excellent editorial work has made for a smooth and coherent text. There is good cross referencing to related topics by other contributors. Illustrations are plentiful; radiographic reproduction is excellent. The variety of specialized equipment is readily identified by manufacturer.

Dr Kadir has produced the best current text in this broad and dynamic field. It will be useful to the practicing interventionist, the radiologist-in-training, and the nonradiologic clinician who uses these services.

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PRACTICAL GUIDE TO THE CARE OF THE MEDICAL PATIENT

Second Edition. By Fred F. Ferri, MD. 804 pp St. Louis, Mo, Mosby-Year Book, 1991. $25.95 (softcover)

The author states that this is a reference book for the busy clinician in need of immediate medical information but is not intended as a substitute for a reference text. It is approximately 7-1/2 inches long by 5-inches wide and easily fits into a lab coat pocket, unlike the *Manual of Therapeutics,* edited by Orland and Saltman (1989) or *Internal Medicine Diagnosis and Therapy*, edited by Stain (1991). The book sells for $25.95, comparable to other such manuals.

The style of the manual is described as concise with few unnecessary words; it is essentially a list of diagnoses and approaches to evaluation and treatment. There is very little descriptive text here, but rather one line sentences and tables. The editor, Dr Fred Ferri, Chief of the Division of Internal Medicine at St. Joseph's Hospital in Rhode Island, is also, I assume, the author of all chapters not otherwise listed under other contributors' names (a sizeable portion of this manual).

A chapter on "Medical Record Abbreviations" contains nine pages of abbreviations, many of which I have never encountered in practice or used. They might make many records unintelligible, and their use should not be promoted.

The chapter on "Approach to the Medical Patient" is good for first year house officers, but should not be needed for the clinician in practice. Chapter six lists differential diagnoses for 35 signs or symptoms and is a good check-off list for a possible missed diagnosis.

I liked the approach to the management of venous thromboembolism, ie, early initiation of coumadin, but question the need for a separate chapter on rhabdomyolysis.

I could find no reference to substantiate the use of glucagon as the preferred antidote to beta blocker poisoning. Similarly, primary aldosteronism is suggested as the diagnosis for hypertensive patients with low potassium, with no mention of the more common use of diuretics. Diazoxide, not nifedipine or labetalol, is listed as the second choice drug for hypertensive emergencies after nitroprusside.

The Preventive Medicine section (Chapter 4) does not address the American Cancer Society guidelines for persons at high risk for colon cancer, but rather only lists screening guidelines for the general population. The treatment of acute gout in this manual, as well as in the *Manual of Medical Therapeutics* and *Internal Medicine Diagnosis and Therapy* (1988) still includes the use of repeated doses of oral colchicine. Although effective, the diarrhea that ensues may last for days, and this historical treatment should no longer be used.

The chapter on Medications, however, is handy, listing available preparations, adult dosages, indications, contraindications, and precautions. The book does contain up-to-date information on the treatment of human immunodeficiency virus (HIV) infections (although oxygen pressure guidelines are not given for initiation of steroids in pneumocystis pneumonia), prophylaxis of endocarditis in adults, and management of alcohol withdrawal.

This manual is concise, although occasionally contains minor inaccuracies; it will fit into the pocket of a house officer's lab coat. Many sections are up-to-date and contain valuable information, easily obtained. I do not agree with the premise that it is a reference for the busy clinician. It is rather a set of "cliff notes" for a standard medical text.

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MARKETING FOR HEALTH AND WELLNESS PROGRAMS

Edited by Donald R. Self, DBA, and James W. Busbin, PhD. 249 pp. New York, Haworth Press, 1990. $29.95 (hardcover)

The editors have succeeded in their efforts to compile an "...eclectic cross-section of current research and commentary..." in the areas of wellness and prevention. The book is organized into sections reflecting important marketing concepts such as product, pricing, promotion, and distribution with a final section on future research focus. Within these areas, the editors have selected articles from the literature to relate practical ideas about how to market different types of health and wellness programs and services.

The preface by William J. Winston, Senior Editor for Marketing of the Haworth Press, shows a keen understanding of the place of wellness and prevention within the health care market. The introduction attempts to clarify the definition of health promotion, wellness, and prevention as a non-traditional health care concept.

The first section, What is the Wellness/Prevention Product?, reflects the eclectic nature of the book by highlighting marketing in a dental practice and marketing as it relates to addiction treatment. The introductory article is a good overview of employee wellness and its occupational medicine origins as well as its relationship to the health care industry. It provides a concise explanation of the players: providers (hospitals, specialty clinics), intermediaries (HMOs, PPOs, insurance carriers), and receivers (government and special interest groups).

The second section, Market Segments for Wellness and Prevention Programs, cuts across a wide spectrum of ages with its focus on suggestions on how to reach pregnant women, adolescents, and older adults. This is accomplished through such methods as marketing through mutual support groups; and using social referral systems already in place such as courts/attorneys, health care, education, clergy, social programs, teachers, counselors, parents and in the case of older adults, their decision makers. The articles selected demonstrate the value of examining the product and the target market carefully to determine how best to reach them. The article by Plank and Gould concludes that by raising health consciousness, people may be more receptive to the marketing of wellness. It also suggests that future research should focus on the spouse, significant other, and decision maker effect on health care consumption behavior.

The third section, Channels of Distribution, presents strong opinions about the value of wellness in general and in particular as it relates to employee wellness. It presents the idea of the "Well-Being Company (WBC)" and the potential value of being a WBC to individual business, the economy, and society. The section proceeds to present the concept of hospitals as channel leaders in health services using vertical marketing systems. Case examples of both successful and unsuccessful wellness programs in hospital settings are presented. These case studies are packed with valuable information for any hospital considering employee or community wellness for employee benefits, goodwill, or public relations purposes.

The final section categorizes research in the area and provides an insightful prescription for future research based on the need of providers, intermediaries, and receivers.

The articles are well written and appropriately categorized. Naturally, there is no single identifiable style since the book is a compilation of articles and commentary. A persistent, field experienced reader will be rewarded with an expanded perception of marketing for not only wellness programs, but also for health care in general. The editors bring an authoritative and practical perspective to the book that bridges the gap between research and practice. Expanded commentary by the editors on the various sections could greatly enhance this work.

It is this reviewer's opinion that academic and clinical health professionals may expect better descriptions of the wellness product and more elaboration concerning the techniques for marketing these products. However, this is not the primary purpose of the book. It is recommended that hospital administrators, academic physicians, and other health care professionals seeking a comprehensive perspective of Wellness/Prevention Services seek another resource. This book would be a valuable supplemental resource to an experienced professional who seeks accurate, timely information on the evolution and status of the emerging Wellness/Prevention Services as a product category within the health care industry.

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BOOKS RECEIVED

CARDIOLOGY

5/1 Progress in Cardiology. Edited by Douglas P. Zipes and Derek J. Rowlands. 170 pp, illustrated. York, Pa, Williams & Wilkins, 1992. $45.00 (softcover)

Rapid Analysis of Electrocardiograms. A Self-Study Program. Second Edition. By Emanuel Stein, MD. 404 pp. York, Pa, Williams and Wilkins, 1992. $26.95 (softcover)

CLINICAL PATHOLOGY

Interpretation of Diagnostic Tests. A Synopsis of Laboratory Medicine. Fifth Edition. By Jacques Wallach, MD. Boston, Massachusetts, Little, Brown & Company, 1992. $29.50 (softcover)

GERIATRICS

Handbook of Prescribing Medications for Geriatric Patients. By Judith C. Ahronheim, MD. 465 pp. Boston, Mass, Little, Brown & Co, 1992. $26.50 (softcover)

Vision and Aging. Issues in Social Work Practice. Edited by Nancy D. Weber, MSW. 196 pp. Binghamton, NY, Haworth Press, 1991. $14.95 (softcover)

Residential Care Services for the Elderly. Business Guide for Home-Based Eldercare. Edited by Doris K. Williams, PhD. 106 pp. Binghamton, NY, Haworth Press, 1991. $19.95 (hardcover)

Story Writing in a Nursing Home. A Patchwork of Memories. By Martha Tyler John, MD. 137 pp, illustrated. Binghamton, NY, Haworth Press, 1991. $19.95 (hardcover)

HEALTH SERVICES

Set No Limits. A Rebuttal to Daniel Callahan's Proposal to Limit Health Care for the Elderly. Edited by Robert L. Barry, OP, and Gerard V. Bradley. 134 pp. University of Illinois Press, 1991. $22.95 (hardcover)

Improving Access to Health Services for Children and Pregnant Women. By Joshua M. Wiener and Jeannie Engel. 90 pp. Washington, DC, Brookings Institution, 1991. $8.95 (softcover)

Strategic Health Planning. Methods & Techniques Applied to Marketing and Management. By Allen D. Spiegel and Herbert H. Hyman. 440 pp. Norwood, New Jersey, Ablex Publishing Corp, 1991. $45.00 (softcover)

Kidney Failure and the Federal Government. Edited by Richard A. Rettig and Norman G. Levinsky. 426 pp. Washington, DC, National Academy Press, 1991. $49.95 (hardcover)

HEMATOLOGY

Blood. Atlas and Sourcebook of Hematology. Second Edition. By Carola T. Kapff, SH, and James H. Jandl, MD. 158 pp, illustrated. Little, Brown & Co, 1991. $95.00 (hardcover)

Fibrinolysis, Thrombosis, and Hemostasis. Concepts, Perspectives, and Clinical Applications. By Sol Sherry, MD. 318 pp, illustrated. Philadelphia, Lea & Febiger, 1992. $59.50 (hardcover)

Genetics of Hematological Disorders. Edited by Christos S. Bartsocas and Dimitris Loukopoulos. 306 pp, illustrated. Bristol, Pa, Hemisphere Publishing Corp, 1992. $95.00 (hardcover)

INFECTIOUS DISEASES

Manual of Antibiotics and Infectious Diseases. Seventh Edition. By John E. Conte, Jr, MD, and Steven L. Barriere, PharmD. 419 pp. Philadelphia, Lea & Febiger, 1992. $29.95 (softcover)

MEDICAL EDUCATION

Beyond Flexner. Medical Education in the Twentieth Century. Edited by Barbara Barzansky and Norman Gevitz. 246 pp. Westport, CT, Greenwood Publishing Group, Inc, 1992. $49.95 (hardcover)

MEDICAL MALPRACTICE

The Guide to Medical Professional Liability Insurance. By Kirk B. Johnson, JD; Martin J. Hatlie, JD, Illene Davidson Johnson, JD. 146 pp. American Medical Association, 1991. $24.00 (softcover)

Grand Rounds on Medical Malpractice. By Francis X. Campion, MD. 372 pp. Chicago, American Medical Association, 1990. $40.00 (softcover)

MISCELLANEOUS

Family Systems Application to Social Work. Training and Clinieal Practice. Edited by Karen Gail Lewis, EdD. 203 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

The Day America Told the Truth. What People Really Believe About Everything That Really Matters. By James Patterson and Peter Kim. 270 pp. New York, Prentice Hall Press, 1991. $19.95 (hardcover)

First Aid for the Boards. A Student-to-Student Guide. By Vikas Bhushan, MD, Edward Chu, Jeffrey Hansen, MD. 136 pp. Norwalk, Ct, Appleton & Lange, 1992. $9.95 (softcover)

Crusading Doctor. My Fight for Cooperative Medicine. By Michael A. Shadid, MD. 283 pp. Norman, Oklahoma, University of Oklahoma Press, 1992 (reprint). $18.95 (hardcover)

The American Dentist. A Pictorial History with a Presentation of Early Dental Photography in America. By Richard A. Glenner, DDS, Audrey B. Davis, PhD, Stanley B. Burns, MD. 194 pp, illustrated. Missoula, Montana, Pictorial Histories Publishing Co, 1990. $19.95 (hardcover)

Multigenerational Family Therapy. By David S. Freeman, DSW. 398 pp. Binghamton, NY, Haworth Press, 1992. $24.95 (softcover)

The Effect of Children on Parents. By Anne-Marie Ambert, PhD. 308 pp. Binghamton, NY, Haworth Press, 1992. $19.95 (softcover)

NEPHROLOGY

Renal and Electrolyte Disorders. Fourth Edition. Edited by Robert W. Schrier, MD. 846 pp, illustrated. Boston, Ma, Little, Brown & Co, 1992. $45.00 (hardcover)

OBSTETRICS AND GYNECOLOGY

Menstrual Health in Women's Lives. Edited by Alice J. Dan and Linda L. Lewis. 301 pp, illustrated. Urbana, Illinois, University of Illinois Press, 1992. $16.95 (softcover)

PEDIATRICS

Adolescents and Their Families. By Mark Worden, PhD. 209 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

Adverse Effects of Pertussis and Rubella Vaccines. Edited by Christopher P. Howson, Cynthia J. Howe, and Harvey V. Fineberg. 367 pp. Washington, DC, National Academy Press, 1991. $39.95 (hardcover)

PSYCHIATRY

Disorders of Learning in Childhood. By Archie A. Silver, MD, and Rosa A. Hagin, PhD. 683 pp, illustrated. New York, John Wiley & Sons, Inc, 1990. $69.96 (hardcover)

Women, Girls, and Psychotherapy. Reframing Resistance. Edited by Carol Gilligan, Annie G. Rogers, Deborah L. Tolman. 272 pp. Binghamton, NY, Harrington Park Press, 1991. $14.95 (softcover)

Drug-Induced Dysfunction in Psychiatry. Edited by Matcheri S. Keshavan and John S. Kennedy. 339 pp. Bristol, Pa, Hemisphere Publishing Corp, 1992. $59.50 (hardcover)

Integrating Individual and Family Therapy. By Larry B. Feldman, MD. 210 pp. New York, Brunner/Mazel Publishers, 1992. $25.95 (hardcover)

Family-of-Origin Therapy: An Intergenerational Approach. James L. Framo, PhD. 248 pp. New York, Brunner/Mazel Publishers, 1992. $29.95 (hardcover)

The Couple's Hypnotic Dance. Creating Ericksonian Strategies in Marital Therapy. By Carol J. Kershaw, EDD. 250 pp. New York, Brunner/Mazel, 1992. $29.50 (hardcover)

Treating Sex Offenders in Correctional Institutions and Outpatient Clinics. A Guide to Clinical Practice. By William E. Prendergast, PhD. 233 pp, illustrated. Binghamton, NY, Haworth Press, 1991. $14.95 (softcover)

SUBSTANCE ABUSE

Cocaine, AIDS, and Intravenous Drug Use. Edited by Barry Stimmel, MD. 124 pp. Binghamton, NY, Haworth Press, 1991. $8.95 (softcover)

What Works in Drug Abuse Epidemiology. Edited by Barry Stimmel, MD. 150 pp. Binghamton, NY, Haworth Press, 1991. $29.95 (hardcover)

Feminism and Addicition. Edited by Claudia Bepko, MSW. 224 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

Homelessness and Drinking. A Study of a Street Population. By Bernard Segal, MD. 150 pp. Binghamton, NY, Haworth Press, 1991. $24.95 (hardcover)

VASCULAR SURGERY

Laser Disobstruction and Laser-Assisted Balloon Angioplasty. A Color Atlas. By Teruo Matsumoto. 97 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1991. $75.00 (hardcover).

OBITUARY LISTINGS

Leatrice G. Borofsky, MD, Hicksville. Died December 19, 1991; age 63. Dr Borofsky was a 1954 graduate of the Medical College of Pennsylvania, Philadelphia. She was a Diplomate of the American Board of Pediatrics and Fellow of the American Academy of Pediatrics. She was a member of the American Neurological Association, the Nassau County Medical Society, and the Medical Society of the State of New York.

Sydney Bressler, MD, New York. Died February 11, 1992; age 85. Dr Bressler was a 1932 graduate of St Louis University School of Medicine, St Louis, Missouri. He was a Diplomate of the American Board of Surgery, a Diplomate of the American Board of Thoracic Surgery, a Fellow of the American College of Surgeons, a Fellow of the American College of Chest Physicians, and a Qualified Fellow of the International College of Surgeons. His memberships included the New York Academy of Medicine, the New York Society for Thoracic Surgery, the American Thoracic Society, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Francis Dickson Brown, MD, Hobart. Died February 6, 1992; age 84. Dr Brown was a 1933 graduate of the Albany Medical College. He was a member of the Delaware County Medical Society and the Medical Society of the State of New York.

William Thomas Clark, MD, Woodgate. Died March 15, 1991; age 91. Dr Clark was a 1925 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a member of the American Public Health Association, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Werner M. Cohn, MD, Wayne, New Jersey. Died December 3, 1991; age 79. Dr Cohn was a 1947 graduate of Medizinische Fakultaet der Humboldt Universitaet, Berlin, Germany. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Dutchess County Medical Society, and the Medical Society of the State of New York.

Alexander Conway, MD, Hempstead. Died February 5, 1991; age 86. Dr Conway was a 1929 graduate of the University of Toronto, Faculty of Medicine, Toronto, Ontario, Canada. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Paul Frederick DeGara, MD, Tenafly, New Jersey. Died August 3, 1991; age 90. Dr DeGara was a 1926 graduate of the Medizinische Fakultaet der Universitaet Heidelberg, Baden-Wurttemberg, Germany. He was a Fellow of the American College of Allergists. His memberships included the New York Academy of Medicine, the American Association of Immunologists, the Society for Experimental Biology and Medicine, the American Society of Internal Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

John Howard Egbert, MD, Amsterdam. Died September 26, 1991; age 80. Dr Egbert was a 1938 graduate of Medizinische Fakultaet, Universitaet Zurich, Zurich, Switzerland. He was a member of the Pan American Association of Ophthalmology, the American Academy of Otolaryngology, the Medical Society of the County of Montgomery, and the Medical Society of the State of New York.

Werner Herman Engel, MD, New York. Died November 1, 1991; age 91. Dr Engel was a 1925 graduate of Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Berlin, Germany. He was a Fellow of the American Psychiatric Association. His memberships included the American Psychiatric Association, the Association for the Advancement of Psychotherapy, the New York County Medical Society, and the Medical Society of the State of New York.

Irving Feldberg, MD, New Seabury, Massachusetts. Died October 30, 1991; age 88. Dr Feldberg was a 1931 graduate of the New York University Medical College. He was a Diplomate of the American Board of Ophthalmology and a Fellow of the American College of Surgeons. His memberships included the American Academy of Ophthalmology and Otolaryngology, the Steuben County Medical Society, and the Medical Society of the State of New York.

Abraham Fineman, MD, Miami Beach, Florida. Died February 11, 1991; age 96. Dr Fineman was a 1920 graduate of Columbia University College of Physicians and Surgeons. He was a Fellow of the American College of Physicians and a Fellow of the American College of Allergists. He was a member of the American Academy of Allergy, the New York County Medical Society, and the Medical Society of the State of New York.

Stephen Robert Goewey, MD, West Sand Lake. Died January 30, 1992; age 53. Dr Goewey was a 1965 graduate of Albany Medical College. He was a member of the Medical Society of the County of Albany and the Medical Society of the State of New York.

Rasela Goldstein, MD, New York. Died October 23, 1991; age 77. Dr Goldstein was a 1940 graduate of the Institutul de Medicina si Farmacie, Iasi, Romania. She was a Fellow of the American College of Obstetricians and Gynecologist. She was a member of the New York County Medical Society and the Medical Society of the State of New York.

Bernard D. Hannan, MD, Flagstaff, Arizona. Died October 30, 1991; age 93. Dr Hannan was a 1920 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. He was a member of the New York Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Joseph J. Jaksy, MD, New York. Died January 19, 1991; age 92. Dr Jaksy was a 1923 graduate of the Univerzita Komenskeho, Lekarska Fakulta, Bratislava, Czechoslovakia. His memberships included the New York State Urological Society, the American Fertility Society, the American Geriatrics Society, the New York County Medical Society, and the Medical Society of the State of New York.

Lawrence I. Kaplan, MD, Roslyn. Died February 14, 1992; age 75. Dr Kaplan was a 1948 graduate of the Faculte de Medecine, Universite de Geneve, Geneve, Switzerland. He was a Diplomate of the American Board of Physical Medicine and Rehabilitation. His memberships included the New York Society for Physical Medicine & Rehabilitation, the New York Academy of Medicine, the American Academy of Physical Medicine and Rehabilitation, the Nassau County Medical Society, and the Medical Society of the State of New York.

Dattatraya G. Lanjewar, MD, Pelham Manor. Died February 3, 1992; age 60. Dr Lanjewar was a 1957 graduate of the Indira Gandhi Medical College, Nagpur University, Nagpur Maharashtra, India. He was a Diplomate of the American Board of Anesthesiology. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Michael N. Lavacca, MD, New Rochelle. Died January 6, 1992; age 71. Dr Lavacca was a 1945 graduate of New York Medical College. His memberships included the New York State Surgical Society, the American Society of Abdominal Surgeons, the Bronx County Medical Society, and the Medical Society of the State of New York.

Josefina C. Lazo, MD, Watertown. Died February 27, 1992; age 51. Dr Lazo was a 1964 graduate of the Faculty of Medicine and Surgery, University of Santo Tomas, Manila, Philippines. She was a member of the American Psychiatric Association, the Medical Society of the County of Jefferson, and the Medical Society of the State of New York.

Herman A. LeBow, MD, Blue Point. Died December 6, 1991; age 74. Dr LeBow was a 1945 graduate of the University of Vermont College of Medicine. He was a member of the American Academy of Family Physicians, the Suffolk County Medical Society and the Medical Society of the State of New York.

F. Robert Levy, MD, Vienna, Virginia. Died January 18, 1991; age 84. Dr Levy was a 1937 graduate of Albertus Universitaet Medizinische Fakultaet, Konigsberg, Prussia, Germany. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Ambrose P. Merrill, MD, Delmar. Died December 22, 1991; age 83. Dr Merrill was a 1935 graduate of Stanford University School of Medicine, Stanford, California. He was a Fellow of the American College of Preventive Medicine. His memberships included the New York Academy of Medicine, the American Public Health Association, the American Geriatrics Society, the Bronx County Medical Society, and the Medical Society of the State of New York.

David John Natt, MD, Brooklyn. Died January 17, 1992; age 34. Dr Natt was a 1984 graduate of the Universidad del Noreste, Escuela de Medicina, Tampico, Tamaulipas, Mexico. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Daniel Richard Pack, MD, Armonk. Died December 31, 1991; age 39. Dr Pack was a 1981 graduate of the New York Medical College. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the Medical Society of the County of Westchester and the Medical Society of the State of New York.

Joseph Peluso, MD, Staten Island. Died February 19, 1992; age 65. Dr Peluso was a 1957 graduate of Facolta di Medicina e Chirurgia, Universita di Torino, Torino, Italy. He was a Fellow of the American College of Obstetricians and Gynecologist. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Eustace G. Phillies, MD, Williamsville. Died November 17, 1991; age 80. Dr Phillies was a 1938 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a member of the New York State Society of Internal Medicine, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

Albert C. Redmond, MD, Barneveld. Died February 9, 1992; age 82. Dr Redmond was a 1936 graduate of Cornell University Medical School. His memberships included the New York Academy of Medicine, the New York Cardiological Society, the New York State Surgical Society, the American Academy of Family Physicians, the Medical Society of the County of Oneida, and the Medical Society of the State of New York.

Antonio Rottino, MD, Teaneck, New Jersey. Died June 30, 1991; age 89. Dr Rottino was a 1929 graduate of New York University School of Medicine. He was a Diplomate of the American Board of Pathology and a Fellow of the American College of Pathologists. His memberships included the New York Pathological Society, the New York State Society of Pathologists, the International Academy of Pathology, the American Association of Pathologists and Bacteriologists, the Society for Experimental Biology and Medicine, the American Society of Clinical Pathologists, the New York County Medical Society, and the Medical Society of the State of New York.

Jakob Samson Schaechter, MD, Webster. Died December 16, 1991; age 79. Dr Schaechter was a 1938 graduate of the Universitaet Wien, Medizinische Fakultaet, Wien, Austria. He was a member of the Medical Society of the County of Monroe and the Medical Society of the State of New York.

John Louis Schimel, MD, New York. Died December 31, 1991; age 76. Dr Schimel was a 1943 graduate of Georgetown University School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatry Association, the American Academy of Psychoanalysis, the American Society of Adolescent Psychiatry the New York County Medical Society, and the Medical Society of the State of New York.

Sydney Shapin, MD, New Rochelle, Died January 20, 1992; age 97. Dr Shapin was a 1922 graduate of the University of Maryland School of Medicine. He was a Diplomate of the American Board of Otolaryngology. His memberships included the New York Rhino-Otolaryngological Society, the American Academy of Otolaryngology, the Medical Society of the County of Kings, and the Medical Society of the State of New York.

C. Garret Sickles, MD, Rye. Died February 16, 1991; age 62. Dr Sickles was a 1956 graduate of Columbia University College of Physicians and Surgeons. His memberships included the New York Academy of Medicine, the American Rheumatism Association, the Medical Society of the County of Westchester, and the Medical Society of the State of New York.

Walter R. Stern, MD, Glens Falls. Died January 19, 1992; age 67. Dr Stern was a 1949 graduate of the University of Rochester School of Medicine and Dentistry. He was a Diplomate of the American Board of Pathology and a Fellow of the American College of Pathologists. His memberships included the New York Pathological Society, the International Academy of Pathology, the American Society of Clinical Pathologists, the Medical Society of the County of Warren, and the Medical Society of the State of New York.

Carl A. Stettenbenz, MD, Snyder. Died February 7, 1992; age 81. Dr Stettenbenz was a 1935 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a Diplomate of the American Board of Pediatrics and a Fellow of the American Academy of Pediatrics. He was a member of the Medical Society of the County of Erie and the Medical Society of the State of New York.

Elmer George St. John, MD, Ormond Beach, Florida. Died November 16, 1991; age 86. Dr St. John was a 1933 graduate of Albany Medical College. He was a Diplomate of the American Board of Nuclear Medicine, a Diplomate of the American Board of Radiology, and a Fellow of the American College of Radiology. His memberships included the Radiological Society of New York State, the Pan American Medical Association. the Radiological Society of North America, Inc, the American Roentgen Ray Society, the Medical Society of the County of Schenectady, and the Medical Society of the State of New York.

H. Egon Susbac, MD, Palm Desert, California. Died January 24, 1992; age 92. Dr Susbac was a 1928 graduate of Schlesisch-Friedrich-Wilhelms Universitaet Medizinische Fakultaet, Breslau, Germany. He was a member of the Medical Society of the County of Kings and the Medical Society of the State of New York.

Louis Venet, MD, New York. Died February 21, 1991; age 78. Dr Venet was a 1937 graduate of the New York University School of Medicine. He was a Diplomate of the American Board of Surgery and a Fellow of the American College of Surgeons. His memberships included the New York State Surgical Society, the American Public Health Association, the New York County Medical Society, and the Medical Society of the State of New York.

George H. Wiedeman, MD, New York. Died December 27, 1991; age 80. Dr Wiedeman was a 1937 graduate of the Deutsche Univerzitat Medizinische Fakulta, Praha, Czechoslovakia. He was a Diplomate of the

American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the American Psychoanalytic Association, the New York County Medical Society, and the Medical Society of the State of New York.

W. Brewster Wolfe, MD, Newburgh. Died December 7, 1991; age 67. Dr Wolfe was a 1949 graduate of Harvard Medical School. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians. He was a member of the New York State Society of Internal Medicine, the American Society of Internal Medicine, the Medical Society of the County of Orange, and the Medical Society of the State of New York.

Irving Yellen, MD, Buffalo. Died November 16, 1991; age 92. Dr Yellen was a 1926 graduate of the State University of New York at Buffalo School of Medicine and Biomedical Science. He was a member of the New York Academy of Medicine, Medical Society of the County of Erie, and the Medical Society of the State of New York.

Robert John Zwirecki, MD, East Amherst. Died October 13, 1991; age 58. Dr Zwirecki was a 1960 graduate of the Medical College of Wisconsin. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a member of the American Academy of Neurology, the Medical Society of the County of Erie, and the Medical Society of the State of New York.

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BC/BE OBSTETRICIAN/GYNECOLOGIST needed for four physician group practice. Philadelphia Community Hospital with tertiary affiliations. Salary commensurate with training and experience. Malpractice insurance paid plus other fringe benefits. Send CV to Northwest OB/GYN Associates, 5800 Ridge Avenue, 2nd floor, Philadelphia, PA 19128 or call Geraldine DeLuca, (215) 487-4583.

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Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

Dosage and Administration: Experimental dosage reported in treatment of erectile impotence.[1,3,4] 1 tablet (5.4 mg) 3 times a day, to adult males taken orally. Occasional side effects reported with this dosage are nausea, dizziness or nervousness. In the event of side effects dosage to be reduced to ½ tablet 3 times a day, followed by gradual increases to 1 tablet 3 times a day. Reported therapy not more than 10 weeks.[3]

How Supplied: Oral tablets of Yocon® 1/12 gr. 5.4 mg in bottles of 100's NDC 53159-001-01 and 1000's NDC 53159-001-10.

References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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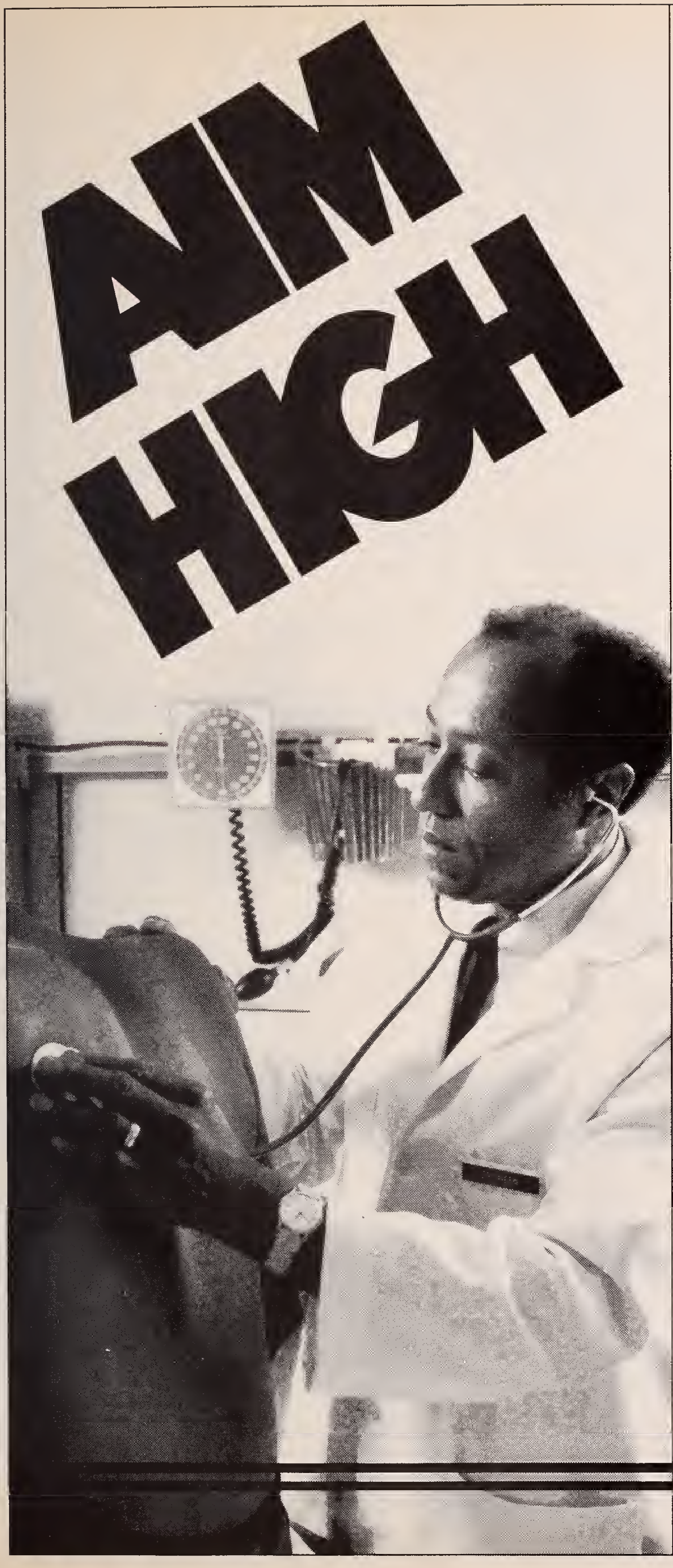

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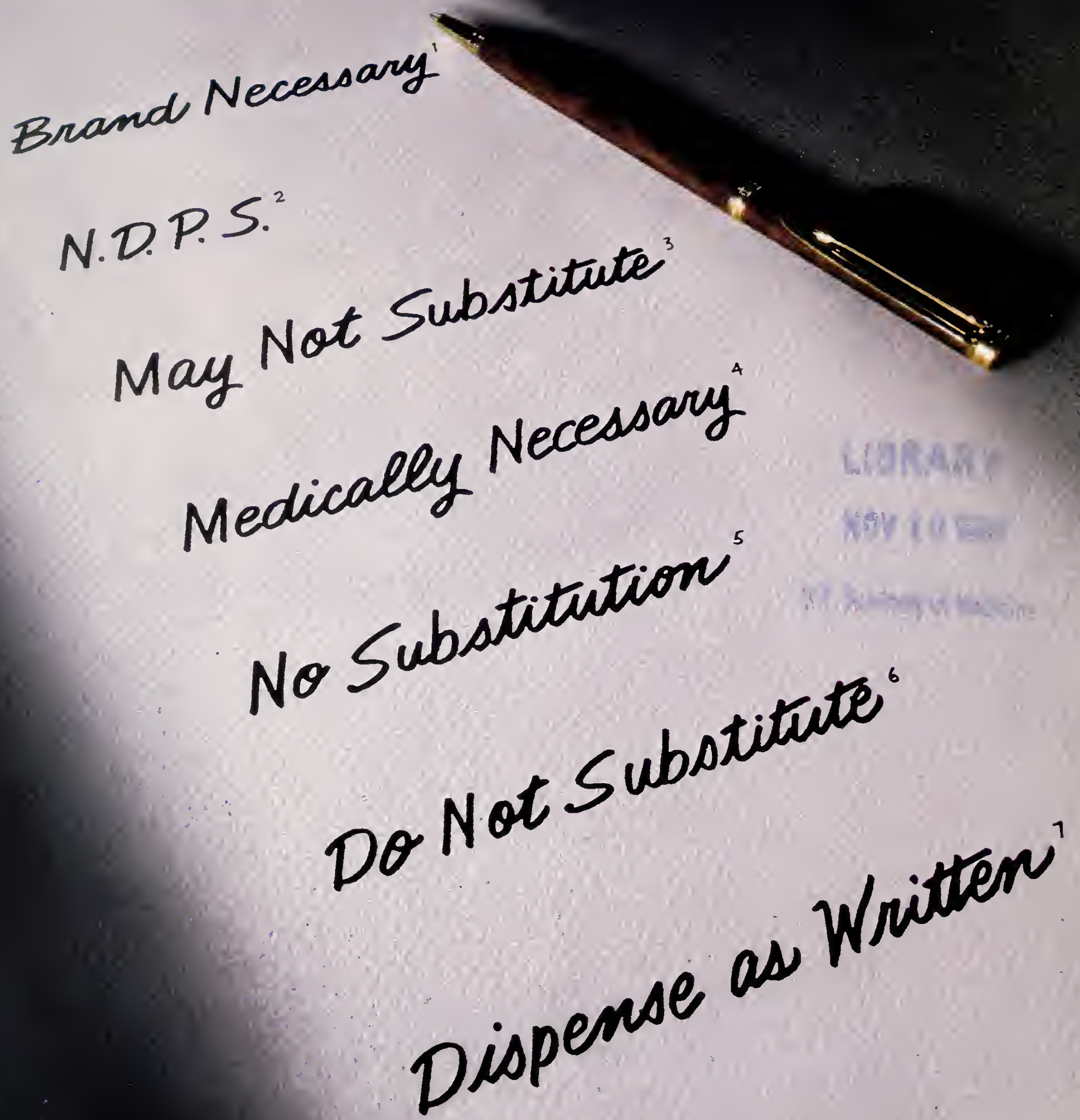

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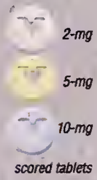

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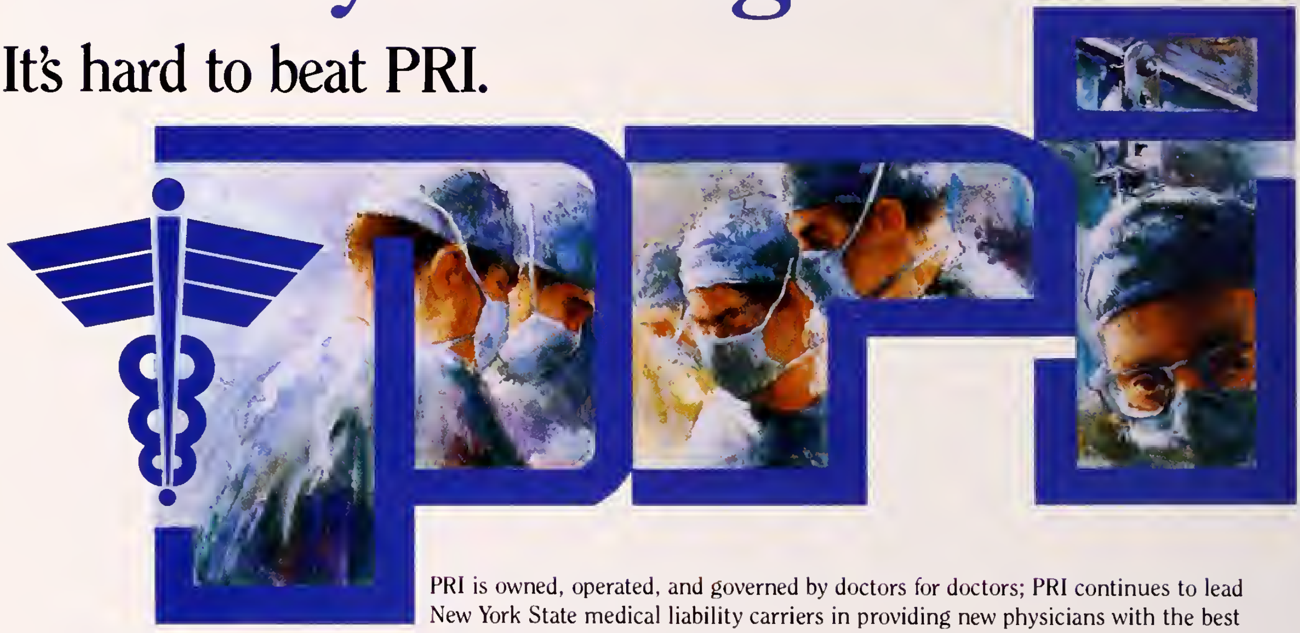

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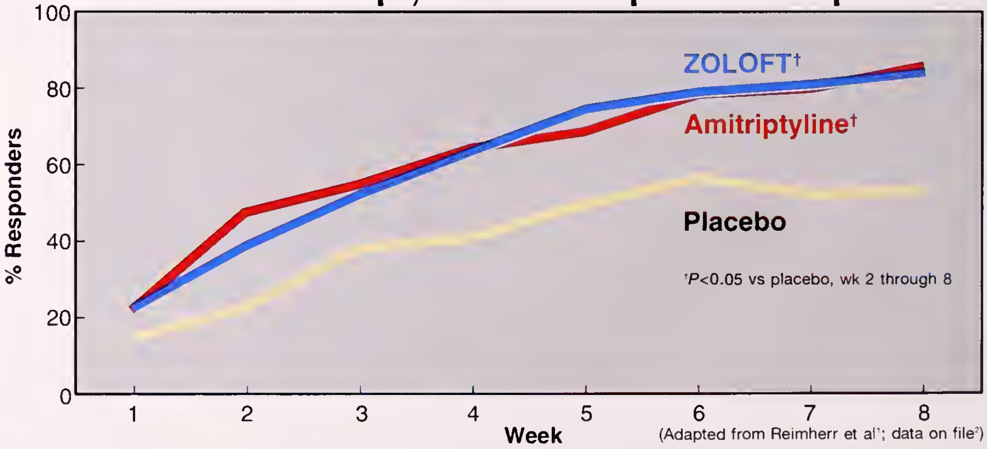
ZOLOFT vs Amitriptyline: Antidepressant Response*[1,2]



(Adapted from Reimherr et al[1]; data on file[2])

*Observed cases, evaluable patients (ie, patients who took drug on or after double-blind treatment day 11 and who had efficacy evaluations within that same time frame); response is defined as a CGI-Improvement score of 1 (very much improved) or 2 (much improved) at time point. Patient numbers for each regimen: ZOLOFT—wk 1=117, wk 2=120, wk 3=106, wk 4=98, wk 5=93, wk 6=94, wk 7=83, wk 8=80; amitriptyline—wk 1=116, wk 2=113, wk 3=111, wk 4=104, wk 5=102, wk 6=92, wk 7=89, wk 8=76; placebo—wk 1=124, wk 2=120, wk 3=116, wk 4=108, wk 5=97, wk 6=89, wk 7=94, wk 8=85.

Double-blind, multicenter, placebo-controlled, parallel study of antidepressant effects of ZOLOFT vs amitriptyline in 448 subjects who met DSM-III criteria for major depression. Symptoms were assessed after a single-blind, 7-day to 14-day placebo washout. Patients were randomly assigned to ZOLOFT, amitriptyline, or placebo for 8 wk. Daily dosage ranges: ZOLOFT—50-200 mg; amitriptyline—50-150 mg.

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ZOLOFT...Effective Continuation Therapy[2,3]

- Responders to ZOLOFT during an 8-week acute-treatment phase were randomized to continue on ZOLOFT or placebo
- A significantly lower relapse rate* was observed for patients treated with ZOLOFT compared with those on placebo, in an assessment of the first 8 weeks of the continuation period
- The effectiveness of ZOLOFT has not been systematically evaluated in long-term use for more than 16 weeks

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- Low risk of toxicity in overdose
- ZOLOFT has a lower incidence of tricyclic-like side effects and a low incidence of nervousness, anxiety, and agitation
- The most common side effects of ZOLOFT include nausea, diarrhea or loose stools, tremor, insomnia, somnolence, and dry mouth

ZOLOFT...Once-Daily Dosing May Enhance Patient Compliance

- ZOLOFT is available in 50-mg and 100-mg scored tablets for flexible, once-daily dosing
- ZOLOFT can be taken in the morning or evening, with or without food

*Relapsing patients were those who became ill (CGI-Severity score ≥4 at any visit) during the double-blind continuation phase and who remained ill during the rest of their time in the study.

Responders in an 8-wk, open-label, acute-treatment study entered a double-blind continuation phase and were randomized to ZOLOFT or placebo. ZOLOFT daily dosage range: 50-200 mg.

References: **1.** Reimherr FW, Chouinard G, Cohn CK, et al. Antidepressant efficacy of sertraline: a double-blind, placebo- and amitriptyline-controlled, multicenter comparison study in outpatients with major depression. *J Clin Psychiatry.* 1990;51(12, suppl B):18-27. **2.** Data on file. Pfizer Inc, New York, NY. **3.** Doogan DP, Caillard V. Sertraline in the prevention of depression. *Br J Psychiatry.* 1992;160:217-222.

Please see brief summary of prescribing information on adjacent page.

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Zoloft™

(sertraline HCl) *50 mg, 100 mg scored tablets*

BRIEF SUMMARY

ZOLOFT™ (sertraline HCl)

INDICATIONS AND USAGE: ZOLOFT (sertraline hydrochloride) is indicated for the treatment of depression.

CONTRAINDICATIONS: None known. **WARNINGS: In patients receiving another serotonin reuptake inhibitor drug in combination with a monoamine oxidase inhibitor (MAOI), there have been reports of serious, sometimes fatal, reactions including hyperthermia, rigidity, myoclonus, autonomic instability with possible rapid fluctuations of vital signs, and mental status changes that include extreme agitation progressing to delirium and coma. These reactions have also been reported in patients who have recently discontinued that drug and have been started on an MAOI. Some cases presented with features resembling neuroleptic malignant syndrome. Therefore, it is recommended that ZOLOFT (sertraline hydrochloride) not be used in combination with an MAOI, or within 14 days of discontinuing treatment with an MAOI. Similarly, at least 14 days should be allowed after stopping ZOLOFT before starting an MAOI.**

PRECAUTIONS ***General:*** **Activation of Mania/Hypomania** - During premarketing testing, hypomania or mania occurred in approximately 0.4% of ZOLOFT (sertraline hydrochloride) treated patients. Activation of mania/hypomania has also been reported in a small proportion of patients with Major Affective Disorder treated with other marketed antidepressants. **Weight Loss** - Significant weight loss may be an undesirable result of treatment with sertraline for some patients, but on average, patients in controlled trials had minimal, 1 to 2 pound weight loss, versus smaller changes on placebo. Only rarely have sertraline patients been discontinued for weight loss. **Seizure** - ZOLOFT has not been evaluated in patients with a seizure disorder. These patients were excluded from clinical studies during the product's premarket testing. Accordingly, like other antidepressants, ZOLOFT should be introduced with care in epileptic patients. **Suicide** - The possibility of a suicide attempt is inherent in depression and may persist until significant remission occurs. Close supervision of high risk patients should accompany initial drug therapy. Prescriptions for ZOLOFT should be written for the smallest quantity of tablets consistent with good patient management, in order to reduce the risk of overdose. **Weak Uricosuric Effect** - ZOLOFT is associated with a mean decrease in serum uric acid of approximately 7%. The clinical significance of this weak uricosuric effect is unknown, and there have been no reports of acute renal failure with ZOLOFT. **Use in Patients with Concomitant Illness** - Clinical experience with ZOLOFT in patients with certain concomitant systemic illness is limited. Caution is advisable in using ZOLOFT in patients with diseases or conditions that could affect metabolism or hemodynamic responses. ZOLOFT has not been evaluated or used to any appreciable extent in patients with a recent history of myocardial infarction or unstable heart disease. Patients with these diagnoses were excluded from clinical studies during the product's premarket testing. However, the electrocardiograms of 774 patients who received ZOLOFT in double-blind trials were evaluated and the data indicate that ZOLOFT is not associated with the development of significant ECG abnormalities. ZOLOFT is extensively metabolized by the liver. The pharmacokinetics of ZOLOFT have not been studied in patients with significant hepatic dysfunction nor have patients with significant hepatic dysfunction been evaluated during treatment with ZOLOFT. Accordingly, ZOLOFT should be used with caution in such patients. Since ZOLOFT is extensively metabolized, excretion of unchanged drug in urine is a minor route of elimination. However, until the pharmacokinetics of ZOLOFT have been studied in patients with renal impairment and until adequate numbers of patients with severe renal impairment have been evaluated during chronic treatment with ZOLOFT, it should be used with caution in such patients. **Interference with Cognitive and Motor Performance** - In controlled studies, ZOLOFT did not cause sedation and did not interfere with psychomotor performance. ***Information for Patients:*** Physicians are advised to discuss the following issues with patients for whom they prescribe ZOLOFT: Patients should be told that although ZOLOFT has not been shown to impair the ability of normal subjects to perform tasks requiring complex motor and mental skills in laboratory experiments, drugs that act upon the central nervous system may affect some individuals adversely. Patients should be told that although ZOLOFT has not been shown in experiments with normal subjects to increase the mental and motor skill impairments caused by alcohol, the concomitant use of ZOLOFT and alcohol in depressed patients is not advised. Patients should be told that while no adverse interaction of ZOLOFT with over-the-counter (OTC) drug products is known to occur, the potential for interaction exists. Thus, the use of any OTC product should be initiated cautiously according to the directions of use given for the OTC product. Patients should be advised to notify their physician if they become pregnant or intend to become pregnant during therapy. Patients should be advised to notify their physician if they are breast-feeding an infant. ***Laboratory Tests:*** None. ***Drug Interactions:*** **Potential Effects of Coadministration of Drugs Highly Bound to Plasma Proteins** - Because sertraline is tightly bound to plasma protein, the administration of ZOLOFT (sertraline hydrochloride) to a patient taking another drug which is tightly bound to protein (e.g., warfarin, digitoxin) may cause a shift in plasma concentrations potentially resulting in an adverse effect. Conversely, adverse effects may result from displacement of protein-bound ZOLOFT by other tightly bound drugs. In a study comparing prothrombin time AUC (0-120 hr) following dosing with warfarin (0.75 mg/kg) before and after 21 days of dosing with either ZOLOFT (50-200 mg/day) or placebo, there was a mean increase in prothrombin time of 8% relative to baseline for ZOLOFT compared to a 1% decrease for placebo ($p<0.02$). The normalization of prothrombin time for the ZOLOFT group was delayed compared to the placebo group. The clinical significance of this change is unknown. Accordingly, prothrombin time should be carefully monitored when ZOLOFT therapy is initiated or stopped. **CNS Active Drugs** - In a study comparing the disposition of intravenously administered diazepam before and after 21 days of dosing with either ZOLOFT (50 to 200 mg/day escalating dose) or placebo, there was a 32% decrease relative to baseline in diazepam clearance for the ZOLOFT group compared to a 19% decrease relative to baseline for the placebo group ($p<0.03$). There was a 23% increase in T_{max} for desmethyldiazepam in the ZOLOFT group compared to a 20% decrease in the placebo group ($p<0.03$). The clinical significance of these changes is unknown. In a placebo-controlled trial in normal volunteers, the administration of two doses of ZOLOFT did not significantly alter steady-state lithium levels or the renal clearance of lithium. Nonetheless, at this time, it is recommended that plasma lithium levels be monitored following initiation of ZOLOFT therapy with appropriate adjustments to the lithium dose. The risk of using ZOLOFT in combination with other CNS active drugs has not been systematically evaluated. Consequently, caution is advised if the concomitant administration of ZOLOFT and such drugs is required. **Hypoglycemic Drugs** - In a placebo-controlled trial in normal volunteers, administration of ZOLOFT for 22 days (including 200 mg/day for the final 13 days) caused a statistically significant 16% decrease from baseline in the clearance of tolbutamide following an intravenous 1000 mg dose. ZOLOFT administration did not noticeably change either the plasma protein binding or the apparent volume of distribution of tolbutamide, suggesting that the decreased clearance was due to a change in the metabolism of the drug. The clinical significance of this decrease in tolbutamide clearance is unknown. **Atenolol** - ZOLOFT (100 mg) when administered to 10 healthy male subjects had no effect on the beta-adrenergic blocking ability of atenolol. **Microsomal Enzyme Induction** - Preclinical studies have shown ZOLOFT to induce hepatic microsomal enzymes. In clinical studies ZOLOFT was shown to induce hepatic enzymes minimally as determined by a small (5%) but statistically significant decrease in antipyrine half-life following administration of 200 mg/day for 21 days. This small change in antipyrine half-life reflects a clinically insignificant change in hepatic metabolism. **Electroconvulsive Therapy** - There are no clinical studies establishing the risks or benefits of the combined use of electroconvulsive therapy (ECT) and ZOLOFT. **Alcohol** - Although ZOLOFT did not potentiate the cognitive and psychomotor effects of alcohol in experiments with normal subjects, the concomitant use of ZOLOFT and alcohol in depressed patients is not recommended. ***Carcinogenesis, Mutagenesis, Impairment of Fertility:*** Lifetime carcinogenicity studies were carried out in CD-1 mice and Long-Evans rats at doses up to 40 mg/kg in mice (10 times on a mg/kg basis and the same on a mg/m^2 basis as the maximum recommended human dose) and at doses up to 40 mg/kg in rats (10 times on a mg/kg basis and 2 times on a mg/m^2 basis, the maximum recommended human dose). There was a dose-related increase in the incidence of liver adenomas in male mice receiving sertraline at 10-40 mg/kg. No increase was seen in female mice or in rats of either sex receiving the same treatments, nor was there an increase in hepatocellular carcinomas. Liver adenomas have a variable rate of spontaneous occurrence in the CD-1 mouse and are of unknown significance to humans. There was an increase in follicular adenomas of the thyroid in female rats receiving sertraline at 40 mg/kg; this was not accompanied by thyroid hyperplasia. While there was an increase in uterine adenocarcinomas in rats receiving sertraline at 10-40 mg/kg compared to placebo controls, this effect was not clearly drug related. Sertraline had no genotoxic effects, with or without metabolic activation, based on the following assays: bacterial mutation assay; mouse lymphoma mutation assay; and tests for cytogenetic aberrations *in vivo* in mouse bone marrow and *in vitro* in human lymphocytes. A decrease in fertility was seen in one of two rat studies at a dose of 80 mg/kg (20 times the maximum human dose on a mg/kg basis and 4 times on a mg/m^2 basis). ***Pregnancy—Pregnancy Category B: Teratogenic Effects*** - Reproduction studies have been performed in rats and rabbits at doses up to approximately 20 times and 10 times the maximum daily human mg/kg dose (4 to 4.5 times the mg/m^2 dose), respectively. There was no evidence of teratogenicity at any dose level. At doses approximately 2.5-10 times the maximum daily human mg/kg dose, sertraline was associated with delayed ossification in fetuses, probably secondary to effects on the dams. There are no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. **Non-teratogenic Effects** - There was also decreased neonatal survival following maternal administration of sertraline at doses as low as approximately 5 times the maximum human mg/kg dose. The decrease in pup survival was shown to be most probably due to *in utero* exposure to sertraline. The clinical significance of these effects is unknown. **Labor and Delivery** - The effect of ZOLOFT on labor and delivery in humans is unknown. **Nursing Mothers** - It is not known whether, and if so in what amount, sertraline or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when ZOLOFT is administered to a nursing woman. **Pediatric Use** - Safety and effectiveness in children have not been established. **Geriatric Use** - Several hundred elderly patients have participated in clinical studies with ZOLOFT. The pattern of adverse reactions in the elderly was similar to that in younger patients.

ADVERSE REACTIONS ***Commonly Observed:*** The most commonly observed adverse events associated with the use of ZOLOFT (sertraline hydrochloride) and not seen at an equivalent incidence among placebo-treated patients were: gastrointestinal complaints, including nausea, diarrhea/loose stools and dyspepsia; tremor; dizziness; insomnia; somnolence; increased sweating; dry mouth; and male sexual dysfunction (primarily ejaculatory delay). ***Associated with Discontinuation of Treatment:*** Fifteen percent of 2710 subjects who received ZOLOFT in premarketing multiple dose clinical trials discontinued treatment due to an adverse event. The more common events (reported by at least 1% of subjects) associated with discontinuation included agitation, insomnia, male sexual dysfunction (primarily ejaculatory delay), somnolence, dizziness, headache, tremor, anorexia, diarrhea/loose stools, nausea, and fatigue. ***Incidence in Controlled Clinical Trials:*** The table that follows enumerates adverse events that occurred at a frequency of 1% or more among ZOLOFT patients who participated in controlled trials comparing titrated ZOLOFT with placebo. Most patients received doses of 50 to 200 mg per day.

Treatment-Emergent Adverse Experience Incidence in Placebo-Controlled Clinical Trials*

	(Percent of Patients Reporting)			(Percent of Patients Reporting)	
Adverse Experience	**ZOLOFT** (N=861)	**Placebo** (N=853)	**Adverse Experience**	**ZOLOFT** (N=861)	**Placebo** (N=853)
Autonomic Nervous System Disorders			Hot Flushes	2.2	0.5
Mouth Dry	16.3	9.3	Fever	1.6	0.6
Sweating Increased	8.4	2.9	Back Pain	1.5	0.9
Cardiovascular			**Metabolic and Nutritional Disorders**		
Palpitations	3.5	1.6	Thirst	1.4	0.9
Chest Pain	1.0	1.6	**Musculoskeletal System Disorders**		
Central and Peripheral Nervous System Disorders			Myalgia	1.7	1.5
			Psychiatric Disorders		
Headache	20.3	19.0	Insomnia	16.4	8.8
Dizziness	11.7	6.7	Sexual Dysfunction-Male (1)	15.5	2.2
Tremor	10.7	2.7	Somnolence	13.4	5.9
Paresthesia	2.0	1.8	Agitation	5.6	4.0
Hypoesthesia	1.7	0.6	Nervousness	3.4	1.9
Twitching	1.4	0.1	Anxiety	2.6	1.3
Hypertonia	1.3	0.4	Yawning	1.9	0.2
Disorders of Skin and Appendages			Sexual Dysfunction-Female (2)	1.7	0.2
Rash	2.1	1.5	Concentration Impaired	1.3	0.5
Gastrointestinal Disorders			**Reproductive**		
Nausea	26.1	11.8	Menstrual Disorder (2)	1.0	0.5
Diarrhea/Loose Stools	17.7	9.3	**Respiratory System Disorders**		
Constipation	8.4	6.3	Rhinitis	2.0	1.5
Dyspepsia	6.0	2.8	Pharyngitis	1.2	0.9
Vomiting	3.8	1.8	**Special Senses**		
Flatulence	3.3	2.5	Vision Abnormal	4.2	2.1
Anorexia	2.8	1.6	Tinnitus	1.4	1.1
Abdominal Pain	2.4	2.2	Taste Perversion	1.2	0.7
Appetite Increased	1.3	0.9	**Urinary System Disorders**		
General			Micturition Frequency	2.0	1.2
Fatigue	10.6	8.1	Micturition Disorder	1.4	0.5

*Events reported by at least 1% of patients treated with ZOLOFT are included.

(1) - % based on male patients only: 271 ZOLOFT (primarily ejaculatory delay) and 271 placebo patients.

(2) - % based on female patients only: 590 ZOLOFT and 582 placebo patients.

Other Events Observed During the Premarketing Evaluation of ZOLOFT (sertraline hydrochloride): During its premarketing assessment, multiple doses of ZOLOFT were administered to approximately 2700 subjects. Events are further categorized by body system and listed in order of decreasing frequency according to the following definitions: frequent adverse events are those occurring on one or more occasions in at least 1/100 patients (only those not already listed in the tabulated results from placebo-controlled trials appear in this listing); infrequent adverse events are those occurring in 1/100 to 1/1000 patients; rare events are those occurring in fewer than 1/1000 patients. Events of major clinical importance are also described in the PRECAUTIONS section. **Autonomic Nervous System Disorders**—*Infrequent:* flushing, mydriasis, increased saliva, cold clammy skin; *Rare:* pallor. **Cardiovascular**—*Infrequent:* postural dizziness, hypertension, hypotension, postural hypotension, edema, dependent edema, periorbital edema, peripheral edema, peripheral ischemia, syncope, tachycardia; *Rare:* precordial chest pain, substernal chest pain, aggravated hypertension, myocardial infarction, varicose veins. **Central and Peripheral Nervous System Disorders**—*Frequent:* confusion; *Infrequent:* ataxia, abnormal coordination, abnormal gait, hyperesthesia, hyperkinesia, hypokinesia, migraine, nystagmus, vertigo; *Rare:* local anesthesia, coma, convulsions, dyskinesia, dysphonia, hyporeflexia, hypotonia, ptosis. **Disorders of Skin and Appendages**—*Infrequent:* acne, alopecia, pruritus, erythematous rash, maculopapular rash, dry skin; *Rare:* bullous eruption, dermatitis, erythema multiforme, abnormal hair texture, hypertrichosis, photosensitivity reaction, follicular rash, skin discoloration, abnormal skin odor, urticaria. **Endocrine Disorders**—*Rare:* exophthalmos, gynecomastia. **Gastrointestinal Disorders**—*Infrequent:* dysphagia, eructation; *Rare:* diverticulitis, fecal incontinence, gastritis, gastroenteritis, glossitis, gum hyperplasia, hemorrhoids, hiccup, melena, hemorrhagic peptic ulcer, proctitis, stomatitis, ulcerative stomatitis, tenesmus, tongue edema, tongue ulceration. **General**—*Frequent:* asthenia; *Infrequent:* malaise, generalized edema, rigors, weight decrease, weight increase; *Rare:* enlarged abdomen, halitosis, otitis media, aphthous stomatitis. **Hematopoietic and Lymphatic**—*Infrequent:* lymphadenopathy, purpura; *Rare:* anemia, anterior chamber eye hemorrhage. **Metabolic and Nutritional Disorders**—*Rare:* dehydration, hypercholesterolemia, hypoglycemia. **Musculoskeletal System Disorders**—*Infrequent:* arthralgia, arthrosis, dystonia, muscle cramps, muscle weakness; *Rare:* hernia. **Psychiatric Disorders**—*Infrequent:* abnormal dreams, aggressive reaction, amnesia, apathy, delusion, depersonalization, depression, aggravated depression, emotional lability, euphoria, hallucination, neurosis, paranoid reaction, suicide ideation and attempt, teeth-grinding, abnormal thinking; *Rare:* hysteria, somnambulism, withdrawal syndrome. **Reproductive**—*Infrequent:* dysmenorrhea (2), intermenstrual bleeding (2); *Rare:* amenorrhea (2), balanoposthitis (1), breast enlargement (2), female breast pain (2), leukorrhea (2), menorrhagia (2), atrophic vaginitis (2).

(1) - % based on male subjects only: 1005; (2) - % based on female subjects only: 1705.

Respiratory System Disorders—*Infrequent.* bronchospasm, coughing, dyspnea, epistaxis; *Rare:* bradypnea, hyperventilation, sinusitis, stridor. **Special Senses**—*Infrequent:* abnormal accommodation, conjunctivitis, diplopia, earache, eye pain, xerophthalmia; *Rare:* abnormal lacrimation, photophobia, visual field defect. **Urinary System Disorders**—*Infrequent:* dysuria, face edema, nocturia, polyuria, urinary incontinence; *Rare:* oliguria, renal pain, urinary retention. **Laboratory Tests** In man, asymptomatic elevations in serum transaminases (SGOT [or AST] and SGPT [or ALT]) have been reported infrequently (approximately 0.8%) in association with ZOLOFT administration. These hepatic enzyme elevations usually occurred within the first 1 to 9 weeks of drug treatment and promptly diminished upon drug discontinuation. ZOLOFT therapy was associated with small mean increases in total cholesterol (approximately 3%) and triglycerides (approximately 5%), and a small mean decrease in serum uric acid (approximately 7%) of no apparent clinical importance.

DRUG ABUSE AND DEPENDENCE ***Controlled Substance Class*** - ZOLOFT (sertraline hydrochloride) is not a controlled substance. ***Physical and Psychological Dependence*** - ZOLOFT has not been systematically studied, in animals or humans, for its potential for abuse, tolerance, or physical dependence. However, the premarketing clinical experience with ZOLOFT did not reveal any tendency for a withdrawal syndrome or any drug-seeking behavior. As with any new CNS active drug, physicians should carefully evaluate patients for history of drug abuse and follow such patients closely, observing them for signs of ZOLOFT misuse or abuse (e.g., development of tolerance, incrementation of dose, drug-seeking behavior).

OVERDOSAGE ***Human Experience*** - There have been 3 cases of ZOLOFT (sertraline hydrochloride) overdosage (approximately 750-2,100 mg). No specific therapy was required for any of the 3 patients, all of whom recovered completely. ***Management of Overdoses*** - Establish and maintain an airway, insure adequate oxygenation and ventilation. Activated charcoal, which may be used with sorbitol, may be as or more effective than emesis or lavage, and should be considered in treating overdose. Cardiac and vital signs monitoring is recommended along with general symptomatic and supportive measures. There are no specific antidotes for ZOLOFT. Due to the large volume of distribution of ZOLOFT, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. In managing overdosage, consider the possibility of multiple drug involvement. The physician should consider contacting a poison control center on the treatment of any overdose.

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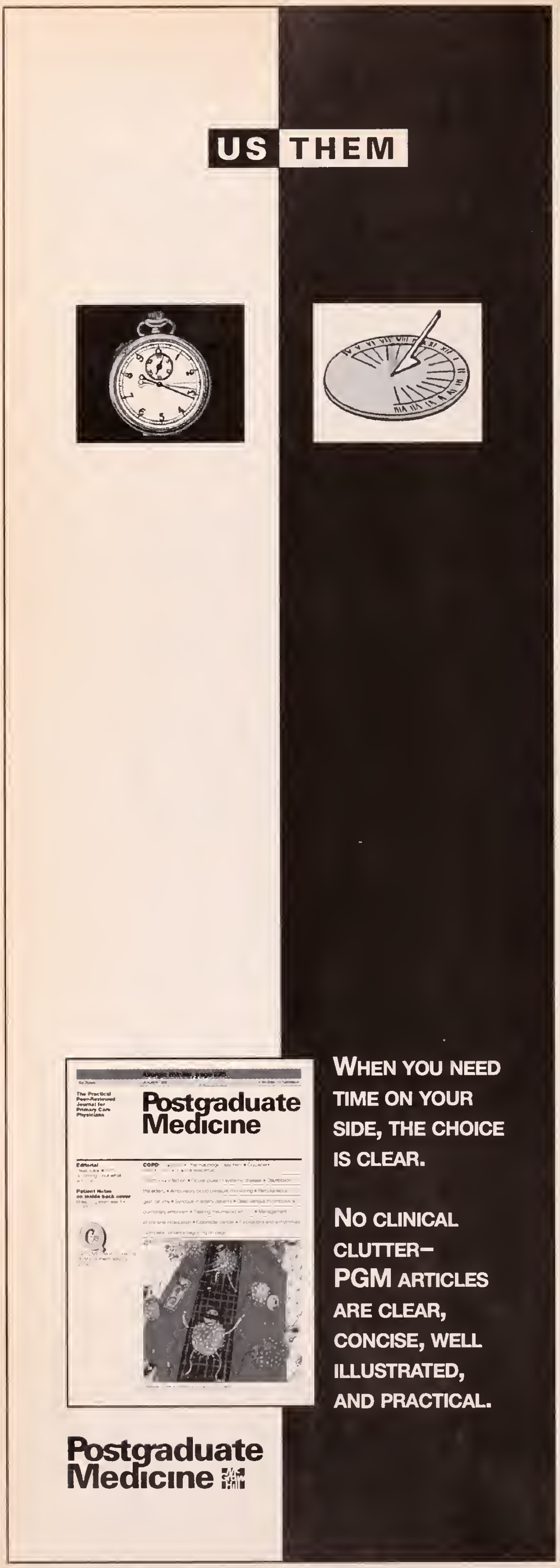
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JULY 1992

AROUND THE STATE

LAKE PLACID

Jul 22–24. **Third Annual Conference on Primary Care Rheumatology.** Grandview Holiday Inn. Contact: Barbara G. Post, Program Coordinator, Regional Medical Education, Sunnyview Hospital and Rehabilitation Center, 1270 Belmont Ave, Schenectady, NY 12308. Tel: (518) 382-4500.

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Jul 8–10. **Interventional Laparoscopy and Stapling Techniques in Urology.** Cabrini Medical Center, New York. Contact: NYU Post-Graduate Medical Center, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295.

AROUND THE NATION

CALIFORNIA

Jul 12–15. **Clinical and Molecular Cytogentics of Developmental Disorders.** Stanford University. Contact: Professional Services Department, March of Dimes Birth Defects Foundation, 1275 Mamaroneck Ave, White Plains, NY 10605. Tel: (914) 428-7100.

Jul 17–19. **Vascular Imaging and Intervention for the 90s.** Ritz Carlton Resort Hotel, San Francisco. *Jul 20–24.* **Clinical**

(*Continued on p 18A*)

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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New York State Journal of Medicine

JUNE 1992 / Volume 92, Number 6

Contents

COMMENTARIES

233 **Inaugural address of the President**
MORTON M. KURTZ, MD

234 **Common interests and shared objectives**
KEVIN M. CAHILL, MD

235 **Providing access to obstetric care in New York State**
RICHARD H. SCHWARZ, MD

RESEARCH PAPERS

237 **Professional liability reform and access to Medicaid obstetric care in New York State**
CAROL C. KORENBROT, PhD; CIARAN PHIBBS, PhD; BARBARA J. LAMB-ABEL, MS

245 **Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening**
DAVID PINNELAS, MD; ROBERT De Le TORRE, MD; JUDITH PUGH, RN; CALVIN STRAND, MD; STEVEN F. HOROWITZ, MD

REVIEW ARTICLE

250 **The antihypertensive effects of exercise training**
CHRISTOPHER C. DUNBAR, MPH, PhD

ESSAYS IN PEDIATRICS

256 **Skin changes associated with hematologic and oncologic diseases in children**
DOROTHY J. GANICK, MD

SPECIAL ARTICLE

262 **Waiting for the doctor**
JACQUELINE GALLOWAY

(continued on p 10A)

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New York State Journal of Medicine

HISTORY

264 **The story of Lassa fever— Part II: Learning more about the disease**
JOHN D. FRAME, MD

CASE REPORTS

268 **Low-dose vincristine-associated bilateral vocal cord paralysis**
P. ALFREDSUNDER, MD; MELVIN C. HOCHMAN, MD; BARRY H. KAPLAN, MD

269 **Tuberculous peritonitis as an initial manifestation of HIV infection**
AYMAN O. SOUBANI, MD, AARON E. GLATT, MD

270 **An unusual manifestation of testicular lymphoma**
JEFFREY N. WEISS, MD; MICHAEL ZIEGELBAUM, MD; LARRY HIRSCHFIELD, MD

272 **Intralymphatic immunotherapy of glioblastoma**
ARNOLD E. EGGERS, MD; JOHN I. MILLER, MD; SALVATORE SCLAFANI, MD

LETTERS TO THE EDITOR

275 ***Nocardia brasiliensis* soft tissue infection in an immunocompromised host**
BARBARA J. BERGER, MD; CHRISTIAN G. ANDERSON, MD; SOL LAVIE, PhD

276 **Low technology and the prevention of nosocomial AIDS**
NEIL J. NUSBAUM, JD, MD

276 **Illegal drug use**
ANTONIO BOBA, MD; JAMES F. CHMIEL, MD; ALAN A. BLOOM; MD

ESSAY REVIEW

279 **The roots of the modern medical malpractice crisis**
RICHARD A. YOUNG, LLB

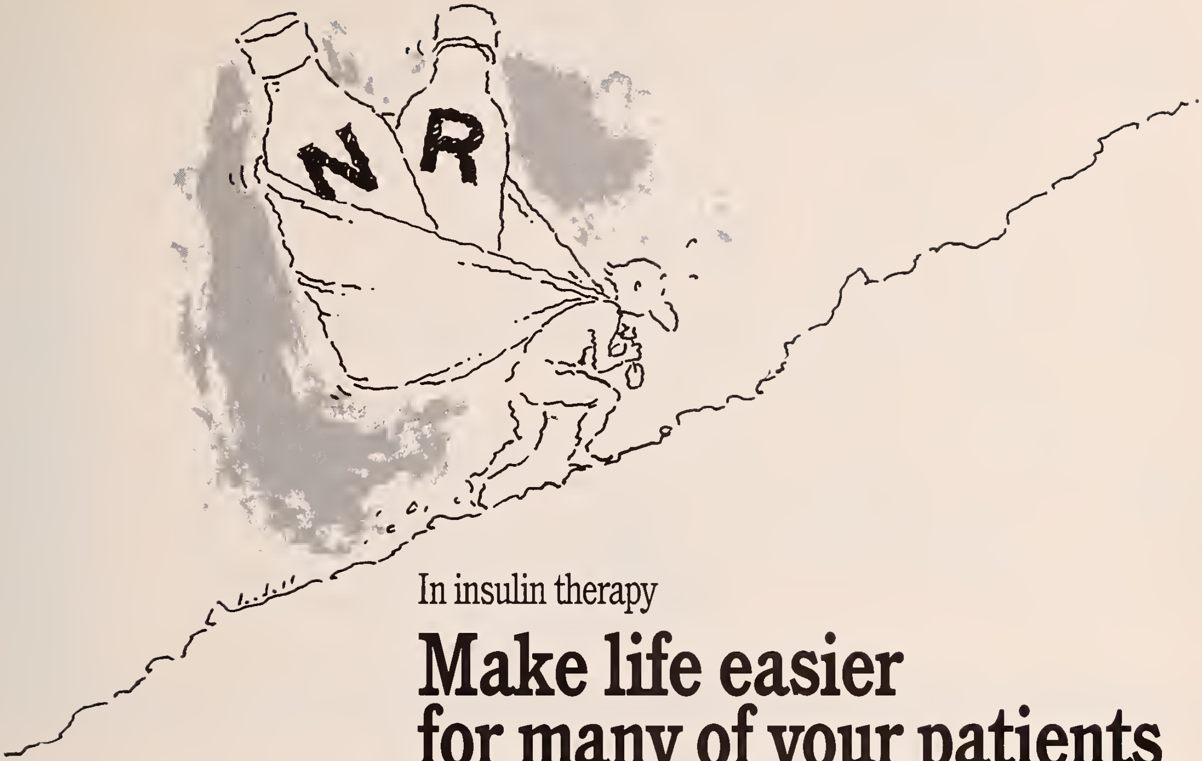
280 LEADS FROM EPIDEMIOLOGY NEWS

283 BOOK REVIEWS

287 OBITUARY LISTINGS

290 AUTHOR GUIDELINES

6A MEDICAL MEETINGS AND LECTURES



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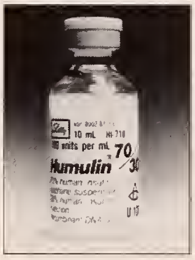

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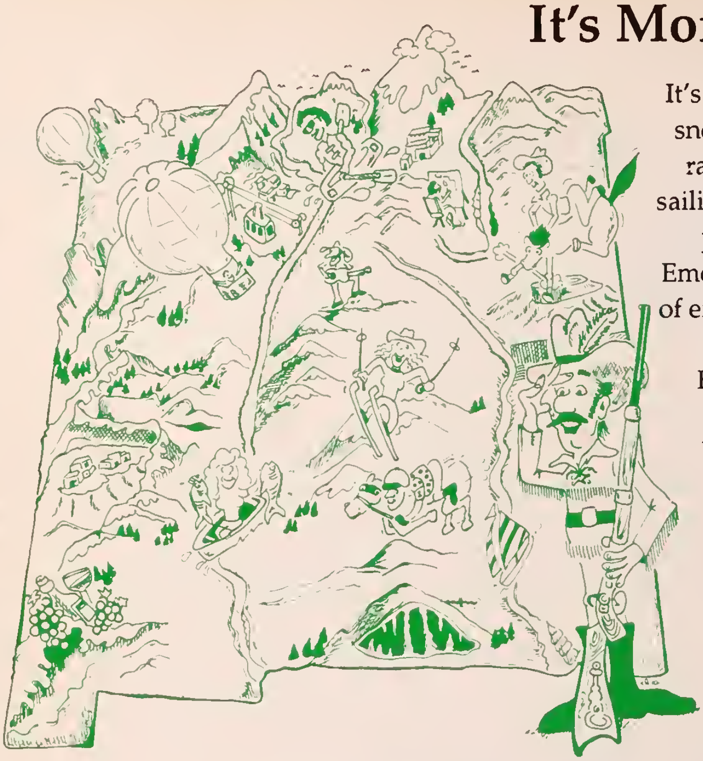

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New York State
Journal of Medicine

COMMENTARIES

Inaugural address of the President

One who has not been in my situation cannot understand the apprehensions felt as this office is assumed. You are as aware as I am that there are no magic recipes to solve the problems our Society faces. The distinguished presidents who have served you previously have tried with great ability and effort. Still our problems became greater each year. You will wonder then what my plan will be. I will answer: to expand and enhance our organization, its services and its philosophy. Since a president can only be a focal point for the ability and knowledge of the members, that will be my role. The initiatives I will follow are necessarily multiple and new, since the dangers are multiple and new.

- We must enhance our support for those health officers who well serve the public and the profession. We will need to give them our aid in obtaining the funds and authority circumstances require.
- The issue of AIDS continues vital, and we must maintain our effort to remove this disease from the arena of politics to the scientific. Really, AIDS is a communicable and sexually transmissible disease.
- The new danger of drug-resistant tuberculosis must be addressed immediately and in depth. The funds required and the measures needed must be made available. The authorities must have the capacity to assist institutions in containing the infection, to increase quick diagnostic facilities, to do effective contact tracing and provide needed treatment, to actively oversee treatment compliance, to assure that therapy required is offered and given, and to mandate and enforce restraint with due process, when required.

This issue is as much political as scientific, and we must be activists for the benefit of the public. The funds and personnel necessary to contain this new epidemic must be obtained. If necessary, we will need to take from Peter and give to a more urgent Paul.

Medicine has always advocated true peer review, but now we see an imposed review by governmental, quasi-governmental and entrepreneurial review organizations which are not true peer reviews. Multiple incidents of judgmental unfairness and injustice have become evident. There is also a thrust making the reviewers judges of the process of medical reasoning and procedure followed in a specific ex post facto situation. Such retrospective review demands a true, reasoned peer evaluation by boards of physicians who have "been there" and such retrospection permits for imposition of arbitrary and unjust views not consistent with the accepted state of the art. The process now imposed is in fact a practice of medicine and must adhere to rules of behavior established by the profession. I will therefore urge that we direct an effort to make peer review the practice of medicine under law. This will create an environment of considered restraint and judgment, which will be just. In the interim, when instances of wrongful judgment become known we should make every effort to correct the wrong. Our actions should be delivered with grace of style and felicity of expression, obviously. But our response would best have the vigor of Patton not Polyanna.

We must continue our efforts to correct flaws in the new Research Based Relative Value Scales (RBRVS). The errors are many and demand attention. As we become more adjusted to the system we will raise the issues not yet addressed. Essentially, now that we work under a mandated structure of fixed fees, what are the plans to control the other economic cost factors for rent, salaries, supplies, and services? A responsive and beneficient government surely would have considered these price-uncontrolled factors. It is reasonable to request information-specific answers. What is the intention of government?

You are aware that our Society has been involved in planning to increase access to health care. The work of our Committee on Insuring the Uninsured has been monumental. I believe we must expand our horizons. We should advocate universal access to necessary basic care for all New York citizens, with a right of choice of plans. We must assist in establishing the methods of payment: tax changes, transfer of budget funds, reduction of administrative requirements, capturing presently untaxed income and so on. This is essential, otherwise

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Address presented before the Annual House of Delegates meeting of the Medical Society of the State of New York, March 21, 1992.

we will be assaulted by cries that it is not affordable. Yet it is, if you use imagination and hard horse sense, and it is necessary. Our nation is in a historic flood tide. Our citizens simply must not be terrified by worry over the lack of access and the financial destruction because of health disasters. A compassionate society cannot accept this. A compassionate profession must demand that the public has access to the care it requires.

As change becomes the ordinary, new delivery schemes will become evident. You will also hear of "rationalization of care" which implies a system of health organization by hospitals, institutions, and government. It strikes me with all the new schemes the only parties not directly involved are the principals; the patient and the physician. Therefore, I ask the House of Delegates to seriously consider and authorize a statewide, multifaceted operating health delivery organization; an organization to be a working partnership between the patient and the physician: a Health Cooperative which would serve as a yardstick of measure for what can be done to maintain and improve the health of the public. In effect, a TVA (Tennessee Valley Authority) of Health serving as the TVA did so successfully. If the profession does not directly participate in delivery operation, others will assume complete control, and the principals will become underlings and irrelevant. This must not happen!

The ideas I have enunciated clearly will require a great resource of effort, thought, and knowledge. Therefore, I ask that we mobilize our capacities by urging a voluntary "Tithe of Six;" six hours a year for Society work, contributing abilities and wisdom, and giving us a tremendous resource bank.

No gracious welcome should be worn out, and I will now stop. Thank you for your attention. Now, it is for you.

MORTON M. KURTZ, MD
President
Medical Society of the
State of New York
420 Lakeville Road
Lake Success, NY 11042

Common interests and shared objectives

We who spend our lives in medicine sometimes need role models to remind us of the nobility of a profession in peril of being submerged by governmental guidelines, constant concerns about escalating health costs and malpractice rates. The series of articles on Lassa fever by John Frame (the second of which is in this issue of the Journal) offers examples that should encourage us to see anew the daily wonders of clinical care and to appreciate the heritage and ancient traditions that are too easily forgotten in the daily struggle to survive.[1]

The story of Lassa fever unfolds like a modern morality play, a mystery tale of unknown illnesses and mortal dangers, on locations ranging from primitive jungles to modern laboratories, and with a heroine dying because she is faithful to her duties. There is an elemental beauty and timeless quality to this tale of death and discovery; it emphasizes the devotion of a missionary doctor and nurse, the curiosity of a caring and thoughtful clinician, and the ultimate conquest by courageous scientists. It offers physicians, and the public we serve, a timely reminder of the challenges that health workers face every day in this AIDS era and that our forebearers have willingly accepted since the time of Hippocrates.

There are other facets of the Lassa fever story that also deserve our attention. Think, for example, of the young American woman, Dr Jeanette Troup, who is noted in today's article to be both "missionary as well as physician." The history of tropical medicine—indeed the whole history of exploration—is full of incredible tales of missionary men and women who journeyed into the unknown to share their vision with strangers they saw as brothers and sisters.

In so doing, they recorded new languages and described new mountains and rivers and plants. They also defined new diseases and discovered new drugs. Medicine has always been an integral part of mission work, for one could hardly seek to save souls without caring for the body. There were occasional abuses to this linkage, and clinical care was sometimes administered too close to a proselytizing sermon. However, in an era when criticism of virtually every aspect of colonial influence is an almost essential part of modern nationalism in the tropics, the good that the missionary physician did is remembered with gratitude and love.

The United States of America might well look to the lessons of the Lassa fever story as they seek ways to communicate with the developing nations. In the "new world order" the poorer countries of the tropics are not only independent but usually suspicious, if not overtly hostile to our designs. A diplomacy of persuasion rather than the traditional diplomacy of pressure just might work; certainly it can be no worse than the present approach. If one can define common interests and shared objectives, if we, as a nation, could emphasize the compassion, competency and even the ultimate sacrifice of a Dr Troup, we could use medicine as an untapped resource in international diplomacy, a desperately needed bridge to peace.

Another aspect of the Lassa fever story is the New York connection. Dr Troup's medical training was in America while her work was in Africa. Her clinical care was, as it is for most missionaries, in that gateway to the world, New York City. For the past quarter century, John Frame cared for most of the Protestant missionaries whereas I seemed to have the Catholics. To give one idea of the scope of the undertaking, we have over 8,400 priests, brothers, and nuns in our program at The Tropical Disease Center of Lenox Hill Hospital.

In this missionary medical program, we not only try to protect our patients with training, inoculations, and medications before they travel overseas, but also offer a clinical and diagnostic service when they return. Maybe most importantly we remain in contact through letters, shortwave radio, and telephone, providing a reassuring contact and advice that frequently allows the missionary to stay in the field and continue his chosen work.

These missionary patients provide some of the most difficult diagnostic challenges in a New York tropical medicine practice. Studies of their long-term health experience can also provide important information that is helpful in protecting such diverse groups as American tourists, peace corps volunteers, and the military. For example, one of our early studies demonstrated the uselessness of a single γ-globulin prophylaxis before prolonged travel, for virtually every missionary who worked overseas for ten years had serologic evidence of hepatitis.[2]

Finally, it should be noted that the missionaries also serve as epidemiologically important sentinel cases, for there is almost no other group of Americans who live for such prolonged periods in the most remote areas of the world. Their contact with and knowledge of isolated tribes, as well as their own carefully studied clinical experience, provide an early warning system that is impossible to duplicate. As the flame of medicine flickers in the changing winds of society it is helpful to have a role model such as Dr Troup, a missionary physician of unwavering faith in and dedication to the careful and compassionate practice of medicine.

KEVIN M. CAHILL, MD
President
The New York Society of
Tropical Medicine
850 Fifth Avenue
New York, N.Y. 10021

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1. Frame JD: The story of Lassa fever—Part II: Learning more about the disease. *NY State J Med* 1992; 92:264–267.
2. Woodson RD, Cahill KM: Viral hepatitis abroad: Incidence in Catholic missionaries. *JAMA* 1972; 219:1191–1193.

Providing access to obstetric care in New York State

Most would agree that there is a crisis in America's health care system and a need for reform. Access to health care specifically has received unprecedented attention as a national concern in this election year. Here in New York State, the legislature, the executive branch, the Medical Society of the State of New York, and the Hospital Association of New York State have prepared proposals to address the complex healthcare needs in our state. The decreasing availability of obstetric care is a particularly distressing indicator of deficiencies in our system.

Even if we were to achieve a miracle and all of the problems of funding, education, and outreach were to be swept away, we cannot be certain that New York's families would have the sheer number of maternity services professionals needed to adequately meet the needs of pregnant women and their young children.

In spite of our fine hospitals and medical education program, New York State ranks 34th among the states in infant mortality. Access to and use of prenatal care are key components in the prevention of prematurity and low birthweight and the resulting infant mortality. While under utilization of health care is often ascribed to societal factors, the availability of obstetric care is an obvious essential in assuring access to prenatal care for all pregnant women.

In New York State, we are experiencing a crisis in maintaining the number of physicians who provide obstetric care. In response to concerns about the accessibility of prenatal care, The American College of Obstetricians and Gynecologists (ACOG), District II with support from the Greater New York Chapter of the March of Dimes conducted a survey of ACOG Fellows and members of the New York State Chapter of the Academy of Family Physicians to determine what policy changes would result in an increase in availability of obstetric care. The results of this survey, reported in this issue of the *Journal*, show that one in six obstetrician-gynecologists and 70% of family physicians have given up the practice of obstetrics.[1] Information from a separate survey has recently documented that less that 50% of the obstetrician-gynecologists who completed residency programs in New York State in 1991 remained in the state to practice.

The gaps in available obstetric care are evident both in our major cities and the rural areas of the state. At least 20 counties have inadequate access to obstetric care. In New York City last year, the Maternal and Infant Care clinics were unable to schedule appointments for over 1,400 women because of obstetric staff shortages. One of seven pregnant women in New York City receives late or no prenatal care. New York City's infant mortality rate is nearly 34% higher than the national average, and the waiting period for essential prenatal care has grown to as much as three months.[2] Syracuse has the seventh highest infant mortality rate for cities in the country.[3]

At the same time, the economic resources of childbearing women have been diminishing. It is now estimated that approximately one-third of women requiring prenatal care are eligible for assistance from the Medicaid Prenatal Care Assistance Program (PCAP).

The article by Korenbrot et al in this issue of the *Journal* reports on the results of the ACOG/March of

Dimes survey with an emphasis on Medicaid findings. In brief, the survey confirms that liability issues are considered the greatest barrier to providing essential primary and preventive care to pregnant women. Fueled largely by an enormous increase in litigation involving neurologically impaired infants, costs and risks associated with delivering quality maternity services in our state have spiraled out of control. The recent $90 million award for a neurologically impaired infant in New York City clearly demonstrates the need for reform.

Last year, Governor Mario Cuomo proposed legislation that would replace the need for litigation with an infant compensation plan for newborns who suffer birth-related neurological injuries. Senator Michael Tully and Assemblywomen Elizabeth Connelley and Pat McGee introduced a similar bill in 1990.

The results of the survey by Korenbrot et al present serious challenges to our profession:

1. There is need for policy change and we must be active advocates for that change.
2. If change is implemented, we must ensure that there are adequate professional resources to provide the care for all women regardless of their resources.

Physicians are most often more comfortable in their one-on-one relationship with a patient, working in the office, the labor unit, or the delivery room. However, that will not be enough. We must become politically active in shaping the policies effecting both the public health of our state and our own professional well being.

Public awareness of the need for liability reform is growing. The public is also aware that failure to achieve that reform impairs access to maternity services and results in escalating costs. Support for obstetric liability reforms comes from diverse groups in New York, including the Kiwanis, the League of Women Voters, the Council of Churches, and the March of Dimes. We must join forces with these advocates and seek the help of others. Although the role of the physician as a concerned care giver is vital and must be strengthened, there is a need for physician commitment to create proactive positive change and ensure provider services.

Remember, a child is born every two minutes in New York State. In the time you have read this, another three children began their journey to the future. Each of us shares in the responsibility for maximizing every child's opportunity for success. That opportunity must begin with good health care for both mother and child.

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1. Korenbrot CC, Phibbs C, Lamb-Abel BJ: Professional liability reform and access to Medicaid obstetric care in New York State. *NY State J Med* 1992; 92:237–245.
2. New York City Department of Health, 1992.
3. New York State Department of Health, 1992.

Professional liability reform and access to Medicaid obstetric care in New York State

CAROL C. KORENBROT, PHD; CIARAN PHIBBS, PHD; BARBARA J. LAMB-ABEL, MS

ABSTRACT. Professional liability costs and fear of lawsuits have made participation in Medicaid difficult for office-based physicians who provide prenatal care, both obstetricians and family physicians. We assessed the possible impact of changes in three liability policy reforms on expanding access of Medicaid-eligible pregnant women to these private physicians. We surveyed members of the New York State District of the American College of Obstetricians and Gynecologists and the New York Academy of Family Physicians to explore whether they would start, expand, or resume obstetric service to Medicaid patients in response to a ceiling on litigation awards, no-fault insurance and a subsidy for liability expenses. We then compared the reported increases in participation on the basis of liability reforms to those in response to changes in Medicaid policies. We found in general that the three liability reforms would have similar impacts on Medicaid participation, although a subsidy was indicated by fewer physicians than the ceiling or no-fault approaches. The support for the liability reforms was as effective as proposals of greater reimbursement rates. The proportion of obstetricians or family physicians increasing their participation depended more on whether they would be starting Medicaid participation, expanding existing Medicaid participation or resuming former Medicaid participation than on the particular liability policy. For obstetricians who now provide obstetric care that includes Medicaid patients, nearly half (40%–47% depending on the policy) would increase the number of Medicaid maternity patients in their practice in response to liability reform, but nearly a fifth (16–18%) of those obstetricians currently in practice who exclude Medicaid patients would do so (significantly less for each policy at the $p < 0.0001$ level). Similarly half (50%–54%) of the obstetricians who have discontinued obstetrics that had included Medicaid patients would resume care and include Medicaid service, but fewer (7%–13%) of those who had excluded Medicaid patients would do so (significantly less for each policy at the $p < 0.0001$ level). For family practice physicians, the same generalization held for those who have discontinued obstetrics: those who had included Medicaid patients were significantly more likely to resume obstetrics and increase their Medicaid obstetric patients in response to any of the three liability policies (32%–45% versus 5% for all three policies; $p < 0.0005$). However, too few family physicians currently providing obstetric care excluded Medicaid patients to compare them reliably with those who included them.

(NY State J Med 1992; 92:237–245)

The physicians who provide obstetric care in private practice, both obstetrician-gynecologists and family physicians, report that over the recent past they have avoided or limited pregnant Medicaid patients in response to rising professional liability premiums and fear of lawsuits.[1] Rising liability premiums affect the extent of Medicaid participation largely because they result in increased costs of professional practice, which have been shown to translate in turn into increased fees.[2] For example, in a national survey in 1985 more than 80% of obstetrician-gynecologists reported increasing their fees because of their increase in liability costs.[3] In New York State, 73% of family physicians providing maternity care and surveyed in 1986 reported raising fees because of malpractice premiums.[4] As fees rise, the gap between fees paid by private insurers and those paid by Medicaid widens because, in an effort to contain costs, Medicaid does not readily adjust its rates.[5,6] It is the magnitude of this gap between Medicaid and private paid rates that has been shown to affect the proportion of physicians that participate in Medicaid.[5,6] Besides economics, fear of lawsuits also plays a role in the extent of Medicaid participation because some obstetric physicians perceive that reduction in care to Medicaid and low-income women is an effective means to limit their exposure to high-risk patients.[1]

When those physicians currently in practice who avoid or limit Medicaid-eligible pregnant women are added to the physicians who discontinued all obstetric care because of liability, the number of office-based physicians available to low-income women declines even further.[3,7–10] In New York State, 17% of obstetricians indicated that they had dropped obstetrics because of liability.[11] Nationally, 19% of family physicians who had ever provided obstetric care indicated that they stopped because of liability.[8,13] To improve access of low-income women to prenatal care, reduction of liability barriers that limit participation in Medicaid of the two primary types of physicians that provide the care, obstetricians and family physicians, is critical.[13–14] Studies have looked at the impact of Medicaid rates and other Medicaid policies on physician participation in the program.[5,6,14–17] Other studies have examined the effects of professional liability on aspects of physician practice.[2–4,7–9,11–13,18,19] However, generally studies have not directly explored effects of liability reform on physician participation in Medicaid.[1] Questions remain as to what extent those who now avoid or limit Medicaid patients in their current obstetric practice would be willing to start or expand their participation with liability reform, and how would their willingness compare with those who currently include Medicaid patients. Of those who have discontinued

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obstetric care but would be willing to resume, how willing would they be to include Medicaid patients with reform of liability, and how much does this depend on whether they included Medicaid patients in the past?

The 1986 New York Medical Liability Reform Act addressed many of the liability concerns of physicians,[20,21] yet premium rates for obstetricians continued to rise into 1990.[22] Three types of proposals are now under consideration by the New York State legislature that might reduce the impact of liability on Medicaid participation by obstetric physicians in private practice: no-fault insurance, a ceiling on litigation awards, and a subsidy for liability premiums. To assess which type of liability policies would be most effective in increasing Medicaid participation of obstetric physicians in New York State, and with what groups of physicians, we surveyed obstetrician-gynecologists and family and general practice physicians through their state professional societies. We evaluated the extent to which those who currently practice obstetrics would either start to include or to expand the number of Medicaid patients in their practice in response to each of these changes in liability policies. We further analyzed those who already include Medicaid patients to see whether those who currently limited the number of Medicaid patients they served were as likely to expand Medicaid participation as those who did not already limit them. We then asked those who discontinued obstetric care but were willing to resume, if they would would be influenced to start or resume care of Medicaid patients by each of these policies. Finally, since direct reform of Medicaid policies are associated with increased physician participation in Medicaid,[5,6,18–20] we also compared the effectiveness of the liability policies to specified Medicaid policies. In this way, the relative expected impact of these different liability reforms on intended Medicaid participation of different groups of physicians in providing obstetric care could be evaluated.

METHODS

Survey Sample. The survey mailing list of members of both the New York State district of the American College of Obstetricians and Gynecologists (ACOG, District II) and the New York State Academy of Family Physicians (NYSAFP) included all active members and graduates of both United States and foreign medical schools. Military and/or federal employees and physicians providing no direct patient care were excluded. A total of 1,903 New York ACOG and 1,573 NYSAFP members who were either junior fellows (board eligible) or full fellows (board certified) were included. Physicians were sent a survey with a code number for the first time in March 1990. Code numbers of returned surveys were logged onto a computer. Physicians not responding to the first mailing received a second mailed survey four weeks after the first.[23]

Data Acquisition. Our survey was based on ACOG surveys of professional liability and the 1987 ACOG survey of obstetric/gynecologic care for underserved women.[3,9–12] The survey consisted of questions that queried physicians about 1) their demographics; 2) the features of their obstetric practice; 3) the obstetric services they provide to Medicaid patients; 4) if applicable, the reasons for discontinuing or resuming obstetric practice; 5) their future plans for their Medicaid obstetric practice. All questions were reviewed by physicians selected by the ACOG, District II, and NYSAFP, as well as the New York State Medicaid Program analysts (Departments of Social Services and Health) for the appropriateness of the questions, and revised according to their recommendations. Family and general practice physicians and obstetrician-gynecologists all received the same version of the survey.

In the survey, the policy changes were divided into three sections: liability policies, billing policies and patient policies. Four liability policy changes were specified: 1) a ceiling on litigation awards; 2) more medical back-up for high-risk obstetric patients; 3) no-fault insurance; and 4) a subsidy for liability expenses. Three are now before the New York State legislature (1, 3, and 4), and one was suggested by the New York State ACOG Committee for Underserved Women (2). Six Medicaid billing policy changes were suggested that had previously been found to influence Medicaid participation by physicians.[5,6,18] One patient policy change was included: the ability to limit Medicaid patients. This policy change was also suggested by the New York State ACOG Committee for Underserved Women. Although respondents were given an opportunity to specify "other" policy changes in each of the three categories, too few such responses were made to be included in the analyses.

All responses were computer-entered by employees of a contract service bureau, with verification of entry accuracy. Data files were created and analyzed using SAS statistical software.

Data Analysis. To analyze the effectiveness of proposed policy changes on increased private office-based physician participation in Medicaid, we first identified among respondents those whose primary obstetric outpatient service site was an office-based practice (solo or group), and then divided the respondents to the policy changes into the following: 1) those who currently practice obstetrics, and 2) those who have discontinued their obstetric practice but indicated that they were willing to resume it. Physicians who discontinued their obstetric practice and would not resume it were not considered further. The former group was then divided into those who include and those who do not include Medicaid patients in their obstetric practice. Those who presently include Medicaid patients were further divided according to whether they limit the number of their Medicaid patients or not. The latter group was divided into those who, while in practice, had included Medicaid patients and those who had not.

In response to each of the suggested policy changes, respondents were directed to indicate whether they "would increase," "would maybe increase," or "would not change," the numbers of Medicaid obstetric patients for whom they would care. In our analysis, we considered a policy change to be effective only if the answer was "would increase." Those responding "would maybe increase" were combined with "would not change" to be conservative in stating the potential impact of a given policy change.

Comparisons between groups of physicians were carried out using χ^2 tests to determine the statistical significance of differences between subgroups.[24] Differences between groups of physicians are indicated as statistically significant whenever the p value of χ^2 statistic was less than 0.05. Comparisons within a group are presented without χ^2 testing because respondents were allowed to indicate all the policies that would influence them to change and therefore their responses are not independent samples, in violation of the assumptions of χ^2 analyses.[24]

Three separate multiple logistic regression models were developed to determine the characteristics of obstetricians and their obstetric practices who would increase the numbers of Medicaid patients in response to each of the three liability policies currently before the legislature.[25] A fourth model was developed to do the same for the greater Medicaid reimbursement billing policy so that the characteristics of those supporting changes in liability policies to increase their Medicaid participation could be compared with those supporting direct increases in Medicaid reimbursement. The characteristics tested to be associated with whether or not a physician would increase Medicaid participation were age (more than 55 years of age or 55 or younger), gender (male or female), race (white or nonwhite), population density of the area served (urban, suburban, or rural), clinical risk (high risk, moderate risk, or low risk), time spent in prenatal care (more than 10 hours per week or 10 hours or less), currently practicing obstetrics (compared with discontinued obstetrics and would resume), extent of Medicaid obstetric patients in the obstetric practice (no Medicaid obstetric patients, less than 10%, or equal to or more than 10%). Physicians themselves assessed whether the community in which they practiced was urban, suburban or rural, the amount of time they spent in prenatal care and the proportion of obstetric patients who were Medicaid patients. The risk level of their obstetric practice was assessed by asking whether they always, usually, sometimes, or never referred patients whom they found to be of high clinical risk. In a low-risk practice, high-risk patients were always referred, in a high-risk practice, they were never referred, and in a moderate-risk practice, they were sometimes or usually referred. In the final models presented, the variable, volume of deliveries (>50 attended per year compared with ≤ 50 per year), was eliminated because it was linearly correlated with hours spent in prenatal care and was not as strongly associated with increasing Medicaid obstetric patients.

Using the models, the odds ratios generated are measures of association between the physician characteristics and increasing participation in Medicaid after adjustment for all the other characteristics.[25] Each odds ratio indicates the probability a physician with the characteristic indicated would increase Medicaid patients in response to the policy divided by that probability for a physician without the characteristic.[24,25] An odds ratio of 1.00 indicates no difference between physicians who have the characteristic from physicians who do not. If a ratio of 1.00 is included within the 95% confidence interval around the odds ratio, it can be said that physicians with and without the characteristic are not statistically different in their response to the policy with 95% confidence.

Similar multiple logistic regression analyses for family physicians were not appropriate because of the small numbers of physicians in two key groups: those who currently did not include Medicaid (14 physicians) and those who had discontinued obstetrics that did not include Medicaid and favored liability reforms (seven physicians for each policy).

RESULTS

The response rate for New York ACOG was 67% (1,281 physicians) and for the NYSAFP, it was 61% (961 physicians). The age and gender distributions of the respondents did not differ significantly from that of the membership in either organization (Table I). Our analyses were restricted to physicians in office-based practice because, in general, they can personally decide whether or not to provide service to Medicaid patients. Physicians who are employed, whether by a private or a public health organization, generally do not make a personal choice in regard to Medicaid participation. Among the obstetrician-gynecologists who responded and indicated that they practiced obstetrics, 84% (1,076 physicians) practiced obstetrics in a private office, either solo or in a group. This was true for 69% (663 physicians) of family and general practice physicians who responded and indicated they practiced obstetrics. Members of New York ACOG and NYSAFP were analyzed as two separate groups because they practice obstetrics under different constraints imposed by their training. In general, family physicians tend to deal with lower-risk patients, they provide a wider range of primary care to those patients, and they rarely provide surgical services; obstetricians, on the other hand, provide a wider range of obstetric services to higher-risk patients and most perform surgery.[14]

TABLE I. Demographics of Survey Respondents and Membership Surveyed

	NY ACOG		NY AFP	
	Members (%)	Respondents (%)	Members (%)	Respondents (%)
Age				
<35	12.3	9.9	20.6	22.5
35–39	17.4	15.9	24.4	24.7
40–44	14.8	14.1	16.0	16.3
45–49	12.6	12.1	7.2	5.7
50–54	13.0	13.0	5.5	3.3
55–59	12.7	13.7	7.2	7.0
60–64	10.7	13.0	9.5	9.9
>65	6.7	8.4	9.5	10.6
Gender				
Male	79.5	78.7	81.5	80.1
Female	20.5	21.3	18.5	19.9
Total number	1,903	1,281	1,573	961

*No group of members differs statistically from respondents at the $p < 0.05$ level.

OBSTETRICIANS

Obstetricians in Current Obstetric Practice. Obstetricians in current practice were divided into those who presently include Medicaid patients ($n = 373$, 46%) and those who do not ($n = 430$, 54%). Table II details the proportion of the obstetricians in each group who would increase the number of Medicaid patients they serve in response to each of the specified policy changes. Almost half (40%–47%, depending on the policy) of the obstetricians who include Medicaid patients would expand their Medicaid practice in response to any of three out of the four suggested liability policy changes. These three are the ones before the state legislature at this time. Most indicated support for no-fault insurance (177 obstetricians), followed by a ceiling on litigation (169) and a subsidy (151). The fourth liability reduction policy, more medical back-up for high-risk patients, was only supported by 28% (104). Among changes in billing policies, three relating to improved reimbursement, especially greater reimbursement, were selected by about half (44%–58%) of obstetricians as arguments that would encourage them to expand their Medicaid volume.

Among the obstetricians who now include Medicaid patients, some limit the number served ($n = 184$, 52%), and others do not ($n = 172$, 48%) (Table III). It is important to know whether those who currently exclude from their practice would be willing to increase their Medicaid participation. These who limited their Medicaid obstetric practice were as likely as those who did not to increase the patients they would serve, if any of the liability reforms were instituted ($p > 0.05$).

Of those practicing obstetricians who do not currently include Medicaid patients, nearly a fifth (16%–18%) would start to include these patients in response to changes in any of the three legislative liability policies, only about half as many (9%) in response to more medical back-up (Table II). More of these physicians would include Medicaid patients in response to improved reimbursement policies (21%–32%) than in response to any of the changes in liability policies. Every one of the policy changes considered would be significantly more effective in improving Medicaid participation by physicians who now include these patients in their practice than by those who do not ($p < 0.001$).

TABLE II. Obstetricians Who Currently Practice Obstetrics and Would Increase Medicaid Patients

OB/GYN Proposed Policy Changes	Currently Practice Obstetrics: Do Include Medicaid Number	Percentage*	Do Not Include Medicaid Number	Percentage
Liability Policies				
Ceiling on litigation awards	169	45	72	17
More medical back-up for high risk	104	28	40	9
No-fault insurance	177	47	78	18
Subsidy for liability expenses	151	40	70	16
Billing Policies				
Fewer delayed claims	179	48	99	23
Faster reimbursement	165	44	91	21
Greater reimbursement	216	58	138	32
Help in Medicaid billing	139	37	68	16
Improved Medicaid communications	154	41	84	20
Medicaid training in audit criteria	85	23	49	11
Patient Policies				
Limit to Medicaid patient volume	91	24	65	15
Total Number	373	100	430	100

*$p < 0.0001$.

Obstetricians Not in Current Obstetric Practice. Obstetricians, who have discontinued their obstetric practice but who would consider resuming it, were divided into those who had included Medicaid patients in their obstetric practice ($n = 24$, 20%) and those who had not ($n = 94$, 80%). Half (50%–54%) of those who had included Medicaid patients would again include them if any of three legislative liability policies were changed. Only 33% would include Medicaid patients again given added medical back-up (Table IV). In addition, about a half (42%–50%) of these obstetricians would include Medicaid patients if any one of the four improved reimbursement changes were enacted. In contrast, only about a tenth (7%–13%) of those who had not included Medicaid patients would include them in their renewed practice in response to the three liability policies before the legislature. About the same proportion (10%–13%) would be influenced to include Medicaid patients in response to all but one of the billing policies (Medicaid training in audit criteria, 5%).

Characteristics of Obstetricians and their Practices Who Would Increase Medicaid Obstetric Patients. The results above indicate that the three liability policies now in front of the legislature were favored over the fourth policy in the survey. Among the billing policies, greater reimbursement was generally favored over other policies. We explored whether the characteristics of the obstetricians (or of their practices) who would increase Medicaid participation in response to each of the three liability policies and greater Medicaid reimbursement were independent of whether they were current or discontinued in their obstetric practice, and whether they included Medicaid patients or not. Table V lists the characteristics investigated and includes the characteristics found to be significant above: whether the physicians currently practiced obstetrics or not and included Medicaid or not. Four of the other characteristics were not associated with increased likelihood of Medicaid participation for any of the policies: age, population density of area served, hours spent in prenatal care per week and, for those who include Medicaid patients, the proportion of their practice accounted for by these patients.

Gender and race were significant characteristics for nearly all four of the policies. Males were more likely to increase Medicaid participation in response to the ceiling on litigation awards (female adjusted odds ratio = 0.64 with confidence interval 0.43–0.94) or no-fault approaches (female adjusted odds ratio = 0.61 with confidence interval 0.41–0.90), but not a subsidy for liability expenses (female adjusted odds ratio = 0.79, but with a confidence interval that includes one, 0.53–1.17). Whites preferred limited litigation awards (nonwhite adjusted odds ratio = 0.51 with confidence interval

TABLE III. Obstetricians Who Limit Medicaid Obstetric Patients But Would Increase Medicaid Obstetric Patients

OB/GYN Proposed Policy Changes	Currently Practice Obstetrics: Do Limit Medicaid Number	Percentage	Do Not Limit Medicaid Number	Percentage
Liability Policies				
Ceiling on litigation awards	78	42	83	48
More medical back-up for high risk	51	28	49	28
No-fault insurance	81	44	88	51
Subsidy for liability expenses	72	39	73	42
Billing Policies				
Fewer delayed claims	81	44	89	52
Faster reimbursement	78	42	79	46
Greater reimbursement	113	61	92	53
Help in Medicaid billing	66	36	67	39
Improved Medicaid communications	69	38	79	46
Medicaid training in audit criteria	38	21	44	26
Total number	184	100	172	100

*No differences between those who do and do not limit Medicaid patients at the $p < 0.05$ level.

TABLE IV. Obstetricians Who Discontinued Obstetrics But Would Resume and Would Increase Medicaid Participation

OB/GYN	Discontinued Obstetrics But Would Resume			
	Did Include Medicaid		Did Not Include Medicaid	
Proposed Policy Changes	Number	Percentage	Number	Percentage
Liability Policies				
Ceiling on litigation awards	12	50*	12	13
More medical back-up for high risk	8	33*	5	5
No-fault insurance	12	50*	9	10
Subsidy for liability expenses	13	54*	7	7
Billing Policies				
Fewer delayed claims	10	42**	10	11
Faster reimbursement	10	42**	10	11
Greater reimbursement	12	50*	12	13
Help in Medicaid billing	11	46*	9	10
Improved Medicaid communications	9	38**	9	10
Medicaid training in audit criteria	3	13	5	5
Patient Policies				
Limit to Medicaid patient volume	10	42*	8	9
Total number	24	100	94	100

*$p < 0.0001$.
**$p < 0.001$.

0.35–0.75) and non-whites favored a no-fault system (non-white adjusted odds ratio = 1.48, with confidence interval, 1.25–2.65) or a subsidy (non-white adjusted odds ratio = 1.82, with confidence interval 1.02–2.15). We found that obstetricians who were male (female adjusted odds ratio 0.54, confidence interval 0.37–0.78) or non-white (non-white adjusted odds ratio, 1.80, confidence interval, 1.24–2.61) were more likely to indicate they would increase Medicaid patients in response to greater reimbursement.

One practice characteristic, a high-risk practice in which the obstetricians never referred patients they perceived as high risk, was associated with not increasing Medicaid patients in spite of changes in awards ceilings or no-fault insurance (high-risk adjusted odds ratios less than one, and confidence intervals that do not include one). Having a moderate-risk obstetric practice in which high-risk patients were "sometimes" referred, or even "usually" referred, was no different from a low-risk practice that always referred high-risk patients. The population density of the community served by the practice, the level of clinical risk in practice, hours spent in prenatal care per week and, for those who include Medicaid patients, the extent of this service, were not associated significantly with

TABLE V. Association of Practice Characteristics of Obstetricians with Increased Medicaid Participation

OB/GYN Physician and Practice Characteristics	Ceiling on Litigation Awards		No-Fault Insurance	
	Odds Ratio*	95% Confidence Interval	Odds Ratio*	95% Confidence Interval
Age >55 years	0.97	(0.68,1.40)	1.00	(0.70,1.42)
Gender, female	0.64**	(0.43,0.94)	0.61**	(0.41,0.90)
Race, nonwhite	0.51**	(0.35,0.75)	1.48**	(1.02,2.15)
Population, suburban	0.89	(0.64,1.23)	0.91	(0.66,1.23)
Population, rural	1.08	(0.60,1.68)	0.94	(0.57,1.56)
Clinical risk, high	0.60**	(0.37,0.98)	0.59**	(0.37,0.96)
Clinical risk, moderate	0.87	(0.61,1.25)	0.89	(0.62,1.28)
Hours of prenatal care >10	1.30	(0.90,1.88)	1.24	(0.86,1.78)
Dropped obstetrics & would resume	0.45**	(0.24,0.84)	0.30**	(0.15,0.58)
Current, includes no Medicaid	0.25**	(0.17,0.37)	0.25**	(0.17,0.37)
Current, includes <10% Medicaid	0.88	(0.59,1.31)	0.83	(0.56,1.24)
	Subsidy for Liability Expenses		Greater Reimbursement	
	Odds Ratio*	95% Confidence Interval	Odds Ratio*	95% Confidence Interval
Age >55 years	0.89	(0.62,1.30)	0.83	(0.59,1.16)
Gender, female	0.79	(0.53,1.17)	0.54**	(0.37,0.78)
Race, nonwhite	1.82**	(1.25,2.65)	1.80**	(1.24,2.61)
Population, suburban	1.06	(0.76,1.48)	1.21	(0.89,1.64)
Population, rural	0.83	(0.49,1.41)	0.87	(0.52,1.44)
Clinical risk, high	0.65	(0.40,1.06)	0.83	(0.53,1.30)
Clinical risk, moderate	0.87	(0.60,1.26)	1.12	(0.78,1.59)
Hours of prenatal care >10	1.07	(0.74,1.55)	1.04	(0.74,1.46)
Dropped obstetrics & would resume	0.34**	(0.17,0.66)	0.32**	(0.17,0.61)
Current, includes no Medicaid	0.28**	(0.19,0.42)	0.40	(0.28,0.57)
Current, includes <10% Medicaid	0.86	(0.57,1.30)	0.99	(0.67,1.53)

*Odds ratios are adjusted for all other characteristics by multiple logistic regression.
**Statistically different from 1.00 at the 95% confidence interval.

TABLE VI. Family Physicians Who Currently Practice Obstetrics and Would Increase Medicaid Obstetric Patients

FP/GP	Currently Practice Obstetrics			
	Do Include Medicaid		Do Not Include Medicaid	
Proposed Policy Changes	Number	Percentage	Number	Percentage
Liability Policies				
Ceiling on litigation awards	27	26	5	36
More medical back-up for high risk	16	16	2	14
No-fault insurance	24	24	6	43
Subsidy for liability expenses	24	24*	7	50
Billing Policies				
Fewer delayed claims	36	35	6	43
Faster reimbursement	31	30	5	36
Greater reimbursement	42	41	6	43
Help in Medicaid billing	22	22	5	36
Improved Medicaid communications	23	23	5	36
Medicaid training in audit criteria	9	9	3	21
Patient policies				
Limit to Medicaid patient volume	10	10	3	21
Total number	102	100	14	100

*$p < 0.05$.

increasing Medicaid services in response to greater reimbursement (confidence intervals of adjusted odds ratios include one).

FAMILY PHYSICIANS

Family Physicians in Current Obstetric Practice. Family physicians in current obstetric practice were divided into those who presently include Medicaid patients ($n = 102$, 88%) and those who do not ($n = 14$, 12%). Table VI details the proportion of family physicians who would increase the number of Medicaid patients in their practice in response to each of the proposed policy changes. About a quarter (24%–26%) of the physicians who include Medicaid patients would expand their service if any one of the three liability policies before the legislature were changed. Just as with obstetricians, lower proportions of family physicians are interested in more medical back-up for high-risk patients to increase their Medicaid patient volume (16%). However, changes in billing policy relating to reimbursement, especially increased reimbursement, would cause more family physicians to expand their Medicaid service (30%–41%) than would the liability policies.

Among the family practitioners who now include Medicaid patients, some limit the number served ($n = 22$, 22%), whereas others do not ($n = 73$, 73%) (Table VII). Those who limited their Medicaid obstetric practice were as likely as those who did not to increase the patients they would serve if any of the liability reforms were instituted ($p > 0.05$), but the small size of the group who limited their Medicaid patients reduces the reliability of this finding. Still, significantly more of those who limited their participation preferred greater reimbursement (64%) than those who did not (34%, $p < .05$), as well as Medicaid training in audit criteria (41% vs 11%, $p < .005$).

For family physicians who do not include Medicaid obstetric patients, a third to one-half (36%–50%) would start to include them if the legislative liability policies changed, but only 14% would change in response to additional medical back-up (Table VI). Changes in billing policy would also cause an increase in Medicaid service (36%–43%). Because this group of physicians is small (a total of only 14 physicians), the apparently large range in preference among policies reflects the choice of very few individuals. The same limitation applies when comparing the responses from physicians who include Medicaid patients with those who do not. Nevertheless, one liability policy, subsidy, is preferred by significantly more of the physicians who do not include Medicaid ($p < 0.05$).

Family Practice Physicians Not in Current Obstetric Practice. Family practice physicians who discontinued obstetric

TABLE VII. Family Physicians Who Currently Practice Obstetrics and Would Increase Medicaid Obstetric Patients

FP/GP	Currently Practice Obstetrics			
	Do Limit Medicaid		Do Not Limited Medicaid	
Proposed Policy Changes	Number	Percentage	Number	Percentage
Liability Policies				
Ceiling on litigation awards	8	36	20	27
More Medical back-up for high risk	3	14	11	15
No-fault insurance	6	27	20	27
Subsidy for liability expenses	3	14	18	25
Billing Policies				
Fewer delayed claims	8	36	24	33
Faster reimbursement	8	36	20	27
Greater reimbursement	14	64*	25	34
Help in Medicaid billing	4	18	16	22
Improved Medicaid communications	5	23	16	22
Medicaid training in audit criteria	9	41**	8	11
Total number	22	100	73	100

*$p < 0.05$.
**$p < 0.005$.

TABLE VIII. Family Physicians Who Discontinued Obstetrics But Would Resume and Would Increase Medicaid Obstetric Patients

FP/GP Proposed Policy Changes	Discontinued Obstetrics: Did Include Medicaid Number	Did Include Medicaid Percentage*	Did Not Include Medicaid Number	Did Not Include Medicaid Percentage
Liability policies				
Ceiling on litigation awards	27	41	7	5
More Medical back-up for high risk	20	30	7	5
No-fault insurance	30	45	7	5
Subsidy for liability expenses	21	32	7	5
Billing policies				
Fewer delayed claims	23	35	12	9
Faster reimbursement	23	35	8	6
Greater reimbursement	24	36	14	11
Help in Medicaid billing	21	32	10	8
Improved medicaid communications	20	30	9	7
Medicaid training in audit criteria	12	18	8	6
Patient policies				
Limit to Medicaid patient volume	9	14	3	2
Total number	66		130	

*$p < 0.0005$.

practice but indicated they were willing to resume were divided into those who had included Medicaid patients ($n = 66$, 34%) and those who had not ($n = 130$, 66%). A third to nearly one-half (32%–45%) of physicians who had included Medicaid patients would again include them if any one of the three legislative liability policies were changed (Table VIII). In this group, 30% of physicians even supported increased medical back-up for high-risk patients, less favored by other groups. All billing policy changes that would improve reimbursement would expand the Medicaid service provided by this group of physicians (35%–36%). Only one-twentieth (5%) of the physicians who had not included Medicaid patients would start including them in response to any of the liability policies, and only 6%–11% would respond favorably to any changes in billing policies.

DISCUSSION

Improving access of low-income women to prenatal care by private physicians through Medicaid is a current aim of national and state policies in an effort to reduce infant mortality. Of the contributing factors to infant mortality it is the preventable complications of pregnancy and delivery that have seen little if any improvement in many years.[26] Yet, access of pregnant Medicaid-eligible women to private physicians is inhibited by professional liability in its present form. The findings of this study provide evidence that liability reform could increase the numbers of pregnant Medicaid eligible women obstetricians and family physicians would serve. Substantial proportions of these physicians indicated they were willing to increase their Medicaid participation in response to a ceiling on litigation awards, no-fault insurance, or a subsidy for liability premiums. Although all three of these proposed reforms had similar levels of support, the subsidy approach was indicated by fewer obstetric physicians than the ceiling or no-fault approaches. The proportion of obstetricians or family physicians increasing their participation in response to liability reforms depended more on whether they would be starting Medicaid participation, expanding existing Medicaid participation or resuming former Medicaid participation than on the particular liability policy. The levels of support for the liability reforms rivaled the support indicated for greater Medicaid reimbursement rates to increase participation.

The 1992 session of the New York State legislature is considering three changes in physician liability policies: 1) limits on non-economic damages in litigation awards (Bill S3388/A5274); 2) authorization of a pilot study of a subsidy of liability expenses for some obstetric physicians in exchange for increasing Medicaid obstetric patients (proposed in the 1991–92 New York State budget as a Department of Health Obstetrical and Pediatric Practitioner Demonstration Program); and 3) no-fault system for impaired infants (New York State Impaired Compensation Program and the New York State Neurologically Impaired Infants Compensation Act).

Limits on non-economic damages (pain and suffering) in litigation awards have been introduced annually for the past several years. These bills have traditionally passed the Senate but do not move in the Assembly. The bills do not directly seek increased Medicaid participation of physicians in exchange for the limits to awards, but instead it is expected that with lower settlements, lower premiums would follow and with lower premiums and settlements, more physicians would be interested in including Medicaid patients in their practices. The current bill (Bill S3388/A5274) seeks to place a $250,000 cap on jury awards for pain and suffering. In California, limits to pain and suffering awards were instituted after the first "malpractice crisis" in liability premium costs in 1976, and California did not see as steep a rise in obstetric liability premiums during the second "malpractice crisis" in the 1980s as did the states of New York and Florida.[3,9,11]

A pilot program of a liability premium subsidy was passed last year by the New York State legislature but without administrative funding needed to implement the program. The legislature will have the opportunity to reauthorize the funding needed by the Department of Health to operate the program this year. The program is designed to improve the accessibility and quality of obstetric services to medically needy women and pediatric services to children to the age of one (S6370/A8890). Five million dollars were appropriated in 1991 for a demonstration project in which the Department of Health would solicit applications from eligible practitioners proposing to increase services in the highest-need populations defined by the Department. However, work on the project has halted until there is a reappropriation. The program will have three components: coverage for new practitioners, coverage for existing practitioners in private practice, and coverage for Diagnostic and Treatment Centers and local health departments. The Department will contract with the selected practitioners to pay for a share of their professional liability premiums. In return, the practitioner will agree to expand covered services and provide care in accordance with quality assurance mechanisms established by the state Department of Health.

There are two no-fault proposals that seek to set up systems outside the tort system of law for compensation of impaired infants, with alternative methods for quality assurance of care. Both systems are based on no-fault compensation for impaired infants that have evolved from systems enacted by the states of Virginia and Florida.[27,28] They are the New York State Impaired Compensation Program (A8793, the "Governor's Bill") and the New York State Neurologically Impaired Infants Compensation Act (S471/A979, the "Tully Bill"). In each bill, an independent commission determines infants (and children up to 10 years of age because of the statute of limitations) who are eligible for compensation. The Governor's Bill covers a class of cases that "could be caused by negligence" (eg, cerebral palsy or blindness, an estimated 1,000 cases per year). The Tully Bill, sponsored in the Assembly by Connelly and McGee, covers infants whose level of impairment is above a defined score on a rating scale (about 600 cases per year). Both cover provider specialties that include obstetricians, pediatricians, anesthesiologists, family physicians, nurses, midwives and hospitals, or institutions. In both systems, the independent commission is the only means to resolve conflict for these cases, although the Governor's Bill would allow the exclusion of cases of willful or wanton negligence. Both bills include quality assurance and financing mechanisms. Physician conduct would be reviewed by the Office of Professional Medical Conduct medical board, with the Governor's Bill including a mechanism for recredentialing physicians. The financing for the Governor's Bill would be through savings achieved from reductions in provider and hospital liability costs. For the Tully Bill, it would be through the "second tier" of liability insurance for physicians in the state. The two bills are not mutually exclusive, and there is an opportunity for a single version that represents a compromise of the features of both to be passed.

Our survey of private physicians who provide obstetric care, both obstetricians and family physicians, showed that in general the three liability reforms could have similar impacts on Medicaid participation, although somewhat fewer physicians indicated that a subsidy for liability premiums would lead them to increase participation. The profile of obstetricians who would increase their Medicaid participation in response to a ceiling on litigation awards would be white males who are currently in obstetric practice that is not high-risk and already includes some Medicaid patients; the profile of those who would increase their participation in response to a subsidy for liability expenses would be non-white physicians of both genders who are currently in obstetric practice and already include some Medicaid patients; those favoring no-fault insurance would be non-white males who are currently in obstetric practice that is not high-risk and already includes some Medicaid patients. By way of comparison, the profile of obstetricians who would increase Medicaid participation in response to increased Medicaid reimbursement would be non-white male physicians who are currently in obstetric practice and already include some Medicaid patients. Characteristics that would not distinguish who would support any of the proposals are: age, having a practice that is urban, suburban or rural, having a practice of moderate or low risk, spending more than 10 hours a week in prenatal care or having a practice that is made up of more than 10% Medicaid patients.

Yudkowsky and collaborators[17] point out that some analysts and policymakers believe that physicians make location and other practice decisions that predetermine Medicaid participation. The fear is that liability reform is too crude an instrument to increase access of low-income pregnant women to private physicians because it benefits both those who include and do not include Medicaid patients. Whereas the lower rates at which obstetricians who do not currently provide obstetric care to any Medicaid-eligible women indicated they would start participation with liability reforms is consistent with this, it is important to note that our study indicates that the majority of obstetricians and family physicians currently in obstetric practice already provide obstetric care to Medicaid-eligible women. Furthermore, it is this group in which the largest numbers of physicians indicated they would increase their Medicaid practice in response to the liability reforms. Our findings provide evidence that liability reforms could have a substantial impact on increasing the numbers of Medicaid patients with access to private physicians.

Acknowledgments. This survey was funded by a grant to the American College of Obstetricians and Gynecologists (ACOG), District II by the New York State March of Dimes, with contributions from the New York State Departments of Health and Social Services (Medicaid), and the New York State Academy of Family Physicians. The results of the survey in their entirety are available in a report from the ACOG, District II.

REFERENCES

1. Institute of Medicine: *Medical Professional Liability and the Delivery of Obstetrical Care,* vol 1. Washington, DC, National Academy Press, 1989.
2. Reynolds RA, Rizzo JA, Gonzalez ML: The cost of medical professional liability. *JAMA* 1987; 257:2776–2781.
3. American College of Obstetricians and Gynecologists (ACOG). Professional liability insurance and its effects: Report of a survey of ACOG's membership. Washington, DC, Needham Harper Worldwide, Inc, 1985, pp 2–10, Tables 13–15, 29–30.
4. Robinson L: The impact of malpractice insurance crisis on family physicians in New York State. *NY State J Med* 1986; 86:392–393.
5. Mitchell JB, Schurman R: Access to private obstetrics/gynecology services under Medicaid. *Med Care* 1984; 22:1026–1037.
6. Perloff JB, Kletke PR, Neckerman KM: Physicians' decisions to limit Medicaid participation: Determinants and policy implications. *J Health Polit, Policy, Law* 1987; 12:221—235.
7. American Academy of Family Physicians (AAFP). A Professional Liability Study. Washington DC, Division of Research and Information Services, 1986, pp 1–4.
8. American Academy of Family Physicians (AAFP). A Professional Liability Study. Washington DC, Division of Research and Information Services, 1987, pp 1–6.
9. American College of Obstetricians and Gynecologists (ACOG). Professional liability insurance and its effects: Report of a survey of ACOG's membership. Washington, DC, Porter, Novelli and Associates, 1983, pp 2–6, Tables 20–26.
10. American College of Obstetricians and Gynecologists (ACOG). OB/GYN Services for Indigent Women: Issues raised by an ACOG Survey. Washington, DC, Needham Harper Worldwide, Inc, 1985, pp ii–ix.
11. American College of Obstetricians and Gynecologists (ACOG). Professional liability insurance and its effects: Report of a 1987 survey of ACOG's membership. Washington, DC, Opinion Research Corporation, 1988, pp ii–ix, Tables 8,14,15.
12. American College of Obstetricians and Gynecologists (ACOG). Professional liability insurance and its effects: Report of a 1990 survey of ACOG's membership. Washington, DC, Opinion Research Corporation, 1990, pp ii–ix.
13. Kruse J, Philips D, Wesley RM: Factors influencing changes in obstetric care provided by family physicians: A national study. *J Fam Pract* 1989; 28:597–602.

14. Mitchell JB: Medicaid participation by medical and surgical specialists. *Med Care* 1991; 21:929–938.
15. Davidson SM: Physician participation in Medicaid: Background and issues. *J Health Polit, Policy Law* 1982; 6:703–717.
16. Fossett JW, Peterson JA: Physician supply and Medicaid participation: The causes of market failure. *Med Care* 1989; 27:386–396.
17. Yudkowsky BK, Cartland JDC, Flint SS: Pediatrician participation in Medicaid 1978 to 1989. *Pediatrics* 85: 567–577.
18. Bredfeldt R, Colliver JA, Wesley RM: Present status of obstetrics in family practice and the effects of malpractice issues. *J Fam Prac* 1989; 28:294–297.
19. Rosenblatt RA, Wright CL: Rising malpractice premiums and obstetric practice patterns. *West J Med* 1987; 146:246–248.
20. Foy DF: Evaluating the impact of the 1986 New York State liability legislation. *NY State J Med* 1987; 87:85–87.
21. O'Keefe DF: Efforts of the medical society of the state of New York to effect medical liability reform. *NY State J Med* 1986; 86:339–341.
22. Medical Liability Mutual Insurance Company. Renewal Information for Policyholders. NY: June 20, 1990, pp 1–2.
23. Korenbrot CC, Lamb-Abel BJ: Obstetric Care in New York State: Report of a 1990 Survey in New York State by the American College of Obstetricians and Gynecologists, District II and the New York State Academy of Family Physicians. Albany, NY, American College of Obstetricians and Gynecologists (ACOG), District II, 1991, pp 9–14.
24. Fleiss JL: *Statistical Methods for Rates and Proportions.* ed 2. NY, John Wiley and Sons, 1981, p 21.
25. Hosmer DW, Lemeshow S: *Applied Logistic Regression.* NY: John Wiley and Sons, 1989.
26. US Department of Health and Human Services: *The 1990 Health Objectives for the Nation: A Midcourse Review.* Washington, DC, 1986.
27. Freeman AD: No-fault cerebral palsy insurance: An alternative to the obstetrical malpractice lottery. *J Health Polit, Policy Law* 1989; 14:707–718.
28. Gallup CL: Can no-fault compensation of impaired infants alleviate the malpractice crisis in obstetrics? *J Health Polit, Policy Law* 1989; 14:691–705.

Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening

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ABSTRACT. Asians historically have low total cholesterol levels and low incidence of coronary heart disease. In performing a coronary heart disease and total cholesterol screening in New York City's Chinatown, we obtained data supportive of previous studies, which focused on Japanese populations and described the influence of environment on total cholesterol. In the present study, Chinese living in Chinatown have higher levels of total cholesterol than would be expected from studies of Chinese living in Shanghai. For every age group examined, Asian-born Chinese living in Chinatown had higher total cholesterol levels than both urban and rural Chinese in Shanghai. In subjects ≦ 30 years of age, urban males in Shanghai had cholesterol levels of 160 ± 34 mg/dL, compared with 206 ± 38 mg/dL for Asian-born males in Chinatown. For females in the same age category, cholesterol levels were 162 ± 35 mg/dL vs 224 ± 45 mg/dL, respectively.

Applying National Cholesterol Education Panel (NCEP) guidelines, the distribution of desirable, borderline-high, and high blood cholesterol levels for the Chinese in Chinatown (41%, 35%, and 23%, respectively) were similar to a group of Caucasians screened (42%, 32%, and 26%) and the group recently used to determine the prevalence of high blood cholesterol in American adults (43%, 30%, and 27%). Studies are needed to stratify the risk of coronary heart disease in other groups that traditionally have a low incidence of coronary heart disease, but which now have adopted Western lifestyles.

(NY State J Med 1992; 92: 245–249)

The relationship between elevated total serum cholesterol, and the risk of developing coronary heart disease (CHD) has been well established. In addition, the treatment of elevated total serum cholesterol, may reduce the incidence of coronary events.[1,2] Current consensus holds that for each 1% fall in total serum cholesterol there is a 2% decrease in non-fatal myocardial infarction, CHD, or both.[3]

The vast majority of studies establishing a cholesterol-CHD link involve middle-aged Caucasian males in the United States and Europe. Risk factor stratification in minority populations within the United States, including measurement of total serum cholesterol, has not been extensively evaluated in the literature. The lack of information on CHD in minorities was a recurrent theme in the report of the Secretary's Task Force on Black and Minority Health in August 1985.[4]

One of the least studied groups is Asians and Pacific Islanders. In general, Asians have a lower incidence of CHD than Caucasians; however, CHD is the leading cause of death for all Asian Americans.[5] The incidence of and mortality from CHD appears to increase as Asians migrate from their countries of origin to the United States. Most of the reports on CHD in Asians living in the United States have focused on the Japanese.[6–9] In all studies, the incidence of CHD and the levels of total serum cholesterol were higher among Japanese living in Hawaii and California compared with those in Japan. Robertson et al[6,7] found the mean total serum cholesterol of Japanese men aged 45–68 years with CHD in Japan was 209.5 mg/dL vs 181.0 in subjects without CHD ($p < 0.01$). Japanese men with CHD living in Hawaii had a mean total serum cholesterol of 232.9 mg/dL vs 218.0 mg/dL in those free of CHD ($p < 0.01$). In the same study, the investigators noted that total serum cholesterol greater than 250 mg/dL was seen in 4.5% of men in Japan, compared with 18.1% of Japanese men in Hawaii.

Frerichs et al[10] looked at mortality from all causes and to cardiovascular disease among seven race-ethnic populations in Los Angeles County, California in 1980. He found that cardiovascular deaths, by race-ethnic group per 100,000, were as follows: Caucasians (430), Blacks (472), Hispanics (391), Japanese (255), Filipinos (84), Chinese (157), and Koreans (144).

To date, there have been no studies of native born Chinese currently living in a large urban environment in the United States. The present study evaluated total serum cholesterol among Asians measured during a self-referred

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screening for CHD risk factors and total cholesterol conducted in New York City's Chinatown district.

PATIENTS AND METHODS

As part of an ongoing program, this medical center conducted a self-referred CHD risk factor and total serum cholesterol screening in Chinatown in cooperation with the Chinatown Health Center. The screening was provided free of charge by the medical center. Participants completed a questionnaire that determined pertinent medical history; cardiac risk factors; whether or not the subject was already on a low fat; low cholesterol diet; birthplace; and, if foreign born, how many years of residence in the United States. Signed permission was obtained to use data anonymously for research purposes (Investigational Review Board approval). Questionnaires were available in both English and Chinese.

Total serum cholesterol was measured via the fingerstick method with the Reflotron Analyzer (Boerhinger Mannheim Diagnostics).[11] A pathologist and technicians from the medical center's Department of Pathology performed the fingerstick, operated the Reflotron, and performed quality control checks on the equipment. Registered nurses and physicians from the division of cardiology, personnel from the medical center's health information service, and Chinese speaking personnel from the Chinatown Health Center were on hand to provide immediate interpretation of results and counseling.

Subjects were referred for treatment according to the recommendations of the Expert Panel of the National Cholesterol Education Program.[12] Results of the study were compared with a similar screening at the medical center, in which 399 of the self-referred subjects were Caucasians.

RESULTS

Questionnaires were obtained on 219 of 239 subjects who were screened. Of these, 194 were Asian, 19 were Caucasian, and four were black. The present study analyzes the data from the 194 Asians screened. For the purposes of comparison, results were compared with previous screenings performed at the medical center.

There were 169 Asians born in a country other than the United States, averaging 15 ± 12 years in the United States. This group had a mean age of 52 ± 16 years and a mean total cholesterol level of 209 ± 42 mg/dL. Only 25 (13%) of the Asians gave their birthplace as the United States. They had a mean age of 29 ± 16 years and a mean total cholesterol of 178 ± 46 mg/dL.

The majority of Asians (62%) who stated that they were not born in the United States gave mainland China as their place of birth. The remainder came from Hong Kong (16%), Malaysia (11%), Vietnam (4%), Taiwan (3%), and Burma (3%). Fifteen percent did not list their country of origin.

We compared time spent in the United States vs total serum cholesterol for foreign born Asian subjects (Table I). We found no significant relationship between the duration of residence in the United States and total serum cholesterol. However, for both males and females, there appeared to be a trend toward increasing total serum cholesterol after ten years in the United States ($p = 0.12$).

Mean total serum cholesterol increased with age in this Asian population, similar to previously described Caucasian populations. The mean values for the Chinatown population screened, distributed by age, were compared with a group of 399 Caucasian subjects screened at Beth Israel Medical Center in June 1989. (Table II). (This was also a self-referred group). The mean age of the Caucasians was 54 ± 18 years compared with 52 ± 16 years for those Asians not born in the United States.

TABLE I. Time Spent in the United States vs Total Serum Cholesterol Levels for Foreign Born Asian Subjects Screened

Years in USA	*n*	Age (yr)	Cholesterol (mg/dL)
1–5			
All	44	45 ± 14	205 ± 41
Males	31	44 ± 14	202 ± 41
Females	12	46 ± 13	218 ± 39
6–10			
All	28	53 ± 19	203 ± 47
Males	16	58 ± 16	211 ± 50
Females	11	50 ± 19	196 ± 42
11–15			
All	15	46 ± 23	214 ± 31
Males	7	50 ± 18	205 ± 28
Females	8	44 ± 26	222 ± 32
16–20			
All	20	55 ± 10	215 ± 38
Males	12	54 ± 10	208 ± 33
Females	7	53 ± 9	234 ± 38
>20			
All	47	61 ± 11	215 ± 40
Males	27	62 ± 10	210 ± 37
Females	20	60 ± 12	222 ± 43

According to the medical history, 72% of the Caucasians had previously had their cholesterol level determined vs only 24% of the Asians. Among the Caucasian subjects, there was a 23% history of hypertension, 9% history of CHD, 17% family history of CHD, 2% history of diabetes, 5% history of cerebrovascular or peripheral vascular disease, and 14% currently smoked cigarettes. Asians reported a 23% history of hypertension, 5% history of CHD, 8% family history of CHD, 4% history of diabetes, 4% history of cerebrovascular or peripheral vascular disease, and 13% currently smoked cigarettes.

The 11 Asian subjects who gave a history of CHD had a mean total cholesterol of 249 ± 46 mg/dL vs 202 ± 42 mg/dL for the 178 Asians who did not. The mean age of those with no history of CHD was 48 ± 18 years vs 65 ± 11 years with a positive history. Finally, Asian subjects who had a history of CHD and who were not born in the United States spent a mean of 20 ± 14 years in the United States vs 15 ± 12 years for those without a history of CHD. None of the above findings reached statistical significance.

TABLE II. Total Serum Cholesterol (mg/dL) Levels in Asian vs 399 Caucasian Study Subjects by Age Distribution

Age (yr)	Asian	Caucasian
20–39		
All	186 ± 40	184 ± 40
Males	185 ± 45	184 ± 35
Females	189 ± 34	185 ± 45
40–59		
All	206 ± 40	210 ± 42
Males	207 ± 41	211 ± 38
Females	205 ± 39	210 ± 46
60–74		
All	224 ± 42	227 ± 41
Males	210 ± 39	204 ± 36
Females	251 ± 33	235 ± 40
≥75		
All	220 ± 39	226 ± 40
Males	203 ± 37	192 ± 29
Females	244 ± 28	238 ± 36

TABLE III. Distribution of Desirable, Borderline-High, and High Total Serum Cholesterol Using the Guideline of the Expert Panel of the National Cholesterol Education Panel

	Desirable (<200 mg/dL)	Borderline-high (200–239 mg/dL)	High (>239 mg/dL)
NHANESII[*]	43% (*n*)	30% (*n*)	27% (*n*)
BI[†] Caucasians	41% (163)	32% (126)	27% (110)
BI Caucasians (20–74 yr)	42% (145)	32% (111)	26% (89)
Chinatown			
All Asians	44% (86)	35% (67)	21% (41)
Asians (20–74 yr)	44% (67)	34% (53)	22% (34)
Foreign-born Asians	40% (68)	37% (62)	23% (39)
Foreign-born Asians (20–74 yr)	41% (58)	36% (50)	23% (32)

Modified from Sempos et al.[13]

[*] NHANESII = National Health and Nutrition Survey Group II.

[†] BI = subjects screened as outpatients at Beth Israel Medical Center.

DISCUSSION

Recently published reports indicate that approximately 36% of all Americans aged 20–74 years are candidates for medical advice and intervention for high blood cholesterol levels.[13] Sempos et al[13] took the National Health and Nutrition Survey Group (NHANESII) and applied the Expert Panel's guidelines for desirable, borderline-high, and high levels of cholesterol. The investigators found that 43% of adult Americans aged 20–74 years had desirable, 30% had borderline-high, and 27% had high levels of blood cholesterol. In June 1989, 399 Caucasian subjects were screened at this medical center using the same methods as in the Chinatown program. Of the 345 persons aged 20–74 years, 42%, 32%, and 26% had desirable, borderline-high, and high levels of blood cholesterol, respectively. This provided suggestive evidence that these self-referred screening populations were representative of the population at large when compared with the NHANESII group studied by Sempos et al. Applying the same guidelines to the foreign born Chinatown group, aged 20–74 years, we found 41% desirable, 36% borderline-high, and 23% high levels of blood cholesterol (Table III).

The most significant finding of the present study is that a group of 169 foreign born Asians averaging 15 ± 12 years of United States residency had the same distribution of desirable, borderline-high, and high levels of blood cholesterol as the NHANESII study group and as the group of 399 Caucasian Americans. The breakdown by age for mean total serum cholesterol in the Asians screened in Chinatown and the Caucasian subjects screened at this medical center were very similar as well.

A significant relationship in mean total cholesterol compared with time spent in residence in the United States did not occur in the present study, perhaps reflecting the small sample size, the effect of aging, or the rapidity of the effect of a change in diet on total serum cholesterol. Connor et al[14] showed that dietary changes can alter total cholesterol levels in as little as 3–4 weeks in patients eating the usual American diet who are then placed on a cholesterol free diet. These same patients had a mean increase of 36 mg/dL in total cholesterol when a 1,000 mg/day diet was resumed after a four-week cholesterol free diet.[14]

Han-zhong et al[15] examined serum lipid and lipoprotein levels in healthy subjects in Shanghai. The investigators

TABLE IV. Total Serum Cholesterol (mg/dL) of Chinese by Age Groups in Urban and Rural Shanghai vs Chinatown

Age (yr)	*n*	Urban[*]	*n*	Rural[*]	*n*	Chinatown[*]
Males						
10–19	63	125 ± 24	—	—	—	—
20–29	123	134 ± 31	10	147 ± 26	2	232 ± 11
30–39	339	152 ± 32	45	142 ± 22	8	177 ± 34
40–49	467	161 ± 31	222	149 ± 21	8	200 ± 29
50–59	335	163 ± 36	162	151 ± 25	14	210 ± 39
60–69	175	161 ± 34	50	132 ± 24	18	212 ± 28
≥70	43	178 ± 37	7	139 ± 18	14	210 ± 44
Females						
10–19	80	131 ± 24	—	—	2	150 ± 20
20–29	129	139 ± 27	8	148 ± 37	1	165
30–39	319	152 ± 32	54	133 ± 24	6	196 ± 38
40–49	590	157 ± 31	279	146 ± 28	7	196 ± 57
50–59	214	177 ± 37	155	155 ± 21	13	206 ± 23
60–69	101	185 ± 39	70	153 ± 23	13	244 ± 27
≥70	22	177 ± 31	10	148 ± 24	8	266 ± 37
>30 years of age						
Males	1,538	160 ± 34	486	147 ± 24	61	206 ± 38
Females	1,246	162 ± 35	568	148 ± 27	46	224 ± 45

Modified from Han-Zhong, et al.[15]

[*] Urban indicates Chinese in urban Shanghai; rural indicates Chinese in rural Shanghai; Chinatown indicates native born Chinese in New York City's Chinatown.

compared subjects by age group and by urban vs rural residency. Urban males more than 30 years old had a mean total cholesterol of 160 ± 34 mg/dL vs rural males with a cholesterol of 147 ± 24 mg/dL. Urban females had a mean level of 162 ± 35 mg/dL vs 148 ± 27 mg/dL for rural females. Table IV lists the values obtained by Han-zhong et al together with the values obtained from subjects in the present study who were born in China, Taiwan, or Hong Kong. Although a direct comparison is impossible (the investigators used fasting venous blood samples), the trend is apparent. For every age group, the Chinese living in Chinatown have higher total serum cholesterol levels. In addition, the investigators of the Shanghai study observed that in the urban population there was no difference in cholesterol levels in males and females before age 45, but after age 45, females had much higher levels than males. In the urban group of Chinese represented in the study reported here, we found this to be true as well (Table IV). In the subjects 45 years of age or older, who were born in China, Hong Kong, or Taiwan ($n = 84$), the mean total cholesterol was 220 ± 41 mg/dL; for males it was 210 ± 38 mg/dL, and for females it was 235 ± 41 mg/dL ($p < 0.01$).

Risk factor questionnaire responses were similar for hypertension, diabetes, cerebrovascular and peripheral vascular disease, and smoking between Asians and Caucasians. Seventeen percent of the Caucasians gave a family history of CHD, whereas only 8% of the Asians answered yes to this question, paralleling published differences in ischemic heart disease incidences between the two groups. This might be expected, since the majority of the Asians screened were not born in the United States, and we therefore assume that their parents and siblings were not born here either. Similarly, 5% of the Asians admitted to a history of CHD vs 9% of the Caucasian group. Twenty-four percent of the Asians vs 72% of the Caucasians stated they previously had their cholesterol level determined. This is due to the presumed better access to health care enjoyed by Caucasians in New York City.

The main limitation of this study is the small sample size of Asian subjects evaluated, and the question of whether this is a representative sample of the Chinese living in New York City. The comparisons between Asians and Caucasians in these two screenings also have to take into account a sample size differential of 2:1 (399 Caucasians vs 194 Asians). Additionally, the study was intended as a screening for total serum cholesterol and other CHD risk factors and not as a specific comparison of levels of serum cholesterol in Chinese living in China vs Chinese living in the United States. The data available for comparison with the indigenous Chinese were obtained via a different technique, namely a fasting sample. However, fasting levels vs non-fasting levels of total cholesterol are both adequate for screening purposes and tend to be similar.[16,17]

The effects of these limitations is that the statistical analysis fails to reach significance, although trends are apparent. Using a larger group of patients and the same method of measuring total serum cholesterol in both the Chinatown group and in mainland China would improve the study. However, this does not mean that a larger sample size would have made the results reach statistical significance. Furthermore, the Asians screened in Chinatown represent only a small fraction of the Asian population in the United States. The majority of these subjects (62%) gave mainland China as their country of birth. Therefore, the possibility of selection bias is present and caution must be used in the interpretation of the results. Additional studies would not only have to be larger, but would also need to incorporate a broader representation of the Asian population in the United States.

In performing a CHD risk factor and cholesterol screening in an ethnic community in New York City, we have provided interesting supporting data suggesting the influence of environment on mean total serum cholesterol. Chinese living in New York City's Chinatown district have higher levels of total cholesterol than would be expected from studies of Chinese living in China. For every age group examined, the Chinese living in New York City had higher total serum cholesterol levels than both urban and rural Chinese in Shanghai. Furthermore, the distribution of desirable, bordeline-high, and high blood cholesterol levels for the Asians in New York City was strikingly similar to a group of New York City Caucasians and to the group used to determine prevalence of high blood cholesterol levels in adults in the United States. Larger patient populations are needed to confirm the present study. Additionally, more studies are needed to stratify the risk of CHD in different ethnic groups now living in the United States, including those groups that historically have a low incidence of CHD, such as Asians.

Acknowledgments. This work was supported in part by the Dina and Raphael Recanati Cardiology Research Fund, The Charles and Mildred Schnurmacher Research and Education Fund of Medicine, and the Patrons of Research Fund, provided by Herbert M. Singer, Esq.

References

1. Lipid Research Clinic Program: The Lipid Research Clinics coronary primary prevention trial results: I. Reduction in the incidence of coronary heart disease. *JAMA* 1984; 251:351–364.
2. Frick MH, Elo O, Haapa K, et al: Helsinki Heart Study: Primary-prevention trial with gemfibrizol in middle-age men with dyslipidemia. *N Engl J Med* 1987; 317:1237–1245
3. Lipid Research Clinic Program: The Lipid Research Clinics coronary primary prevention trial results: II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. *JAMA* 1984; 251:365–374.
4. Margaret M. Heckler, Secretary: Report of The Secretary's Task Force on Black and Minority Health, United States Department of Health and Human Services. United States Government Printing Office, August 1985.
5. Yu EH, Chang CF, Lu WT, et al: Asian white mortality differentials: Are there excess deaths? Unpublished manuscript, Task Force on Black and Minority Health, Department of Health and Human Services. 1984.
6. Robertson TL, Kato H, Rhoads G, et al: Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California. Incidence of myocardial infarction and death from coronary heart disease. *Am J Cardiol* 1977; 39:239–243.
7. Robertson TL, Gordon T: Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California. Coronary heart disease risk factors in Japan and Hawaii. *Am J Cardiol* 1977; 39:244–249.
8. Marmot MG, Syme SL, Kagan A, et al: Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California: Prevalence of coronary and hypertensive heart disease and associated risk factors. *Am J Epidemiol* 1975; 102:514–525.
9. Worth RM, Kato H, Rhoads GG, et al: Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California: Mortality. *Am J Epidemiol* 1975; 102:481–490.
10. Frerichs RR, Chapman JM, Maes EF, et al: Mortality due to all causes and to cardiovascular diseases among 7 race-ethnic populations in Los Angeles County, 1980. *Int J Epidemiol* 1984; 13:291–298.
11. Sedor FA, Holleman CM, Heyden S, et al: Reflotron cholesterol measurement evaluated as a screening technique. *Clin Chem* 1988; 34:2542–2545.
12. The Expert Panel: Report of the National Cholesterol Education Program expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. *Arch Intern Med* 1988; 148:36–39.
13. Sempos C, Fulwood R, Haines C, et al: The prevalence of high blood cholesterol levels among adults in the United States. *JAMA* 1989; 262:45–52.

14. Connor WE, Connor SL: The dietary treatment of hyperlipidemia: Rationale, technique, and efficacy. *Med Clin North Am* 1982; 66:7:485–518.

15. Han-zhong Z, et al: Study of serum lipids and lipoproteins of healthy subjects in Shanghai. *Chin Med J* 1986; 99:657–659.

16. Garber AM, Sox HC, Littenberg B: Screening asymptomatic adults for cardiac risk factors: The serum cholesterol level. *Ann Intern Med* 1989; 110:622–639.

17. Cohn JS, McNamara JR, Schaefer EJ: Lipoprotein cholesterol concentrations in the plasma of human subjects as measured in the fed and fasted state. *Clin Chem* 1988; 34:2456–9.

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MSSNY Wins: Court Throws Out Amendment Shortening Registration

In a major victory for physicians, the New York State Supreme Court in Albany County ruled in favor of the Medical Society of the State of New York and Nathaniel H. Solomon, MD, plaintiffs in a lawsuit challenging the State law that amended the registration period for physicians' licenses. In a decision on April 10, Judge Thomas W. Keegan held that section 280 of Chapter 190 of the laws of 1990 is unconstitutional and ruled that the State could not justify impairment of physicians' rights by its need to raise revenues. The judge ordered the State to refund that portion of the registration applicable to 1991 that physicians had paid pursuant to section 280.

The Medical Society challenged section 280 because it required physicians to pay twice in order to be registered and to practice medicine in 1991. The registration period previously in effect was for the three-year period 1989–1991, with a total fee of $220, or $73 per year. Section 278 of chapter 190 shortened the registration period to two years and raised the fee to $330, or $165 per year. Section 280 of chapter 190 provided that the registration period which convened January 1, 1989, and was to extend until December 31, 1991, would end instead a year earlier and the new two-year registration period would commence on January 1, 1991. This meant that physicians who had paid for 1991 as part of their three-year registration fee would have to re-register and pay again as part of the new two-year registration period.

The ruling can mean that physicians who paid $330 for the 1991–1992 biennial registration and who also paid for the previous 1989–1991 triennial registration will be entitled to receive a refund of $165. The State has not determined at this time whether to appeal Judge Keegan's ruling. If the State appeals, the requirement to pay a refund will be delayed pending the determination of the appeal.

The antihypertensive effects of exercise training

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To what degree exercise training will alter resting blood pressure in hypertensive individuals remains controversial. Although exercise has been proposed as a therapeutic modality for most cases of moderate to severe hypertension, consensus clearly continues to favor pharmacologic intervention.[1,2] This latter approach appears to have been successful in reducing mortality. For example, the pharmacologic treatment of hypertension is believed to be a factor in the recent decrease in mortality from cardiovascular disease.[3] Reductions in the quality of life and adverse reactions to medication remain a problem however.[3-4] It is also well known that many patients discontinue taking medication on their own initiative or are sporadic in their compliance.[5] Because currently available medications do little to change the underlying factors that initially led to an elevation of blood pressure, the pressure will return to unacceptable levels in these situations. There is also concern that if pharmacologic treatment promotes regression of adaptive left ventricular hypertrophy and medication is discontinued, the consequences may be unpredictable.[6]

The benefits of pharmacologic treatment in some subgroups of hypertensive patients are also less clear. Although the efficacy of such treatment has been demonstrated in severe hypertensives[1,2] and for middle-aged men,[7] the appropriateness of medication for mild-to-moderate hypertension, younger patients,[8] asymptomatic older patients,[9] and, perhaps, women[10] is not as well established. Initial reports from the large-scale, Multiple Risk Factor Intervention Trial indicated that some forms of pharmacologic therapy may have actually increased mortality for a subgroup of hypertensives,[11,12] although the conclusions were later questioned.[13] More recent reports have also found increased mortality with both small and large decreases in diastolic blood pressure; the investigators speculated and found that these findings are possibly related to toxicity from pharmacologic interventions.[14] In that investigation, the small modest decreases (7–17 mm Hg) in blood pressure were associated with the least mortality.[14] It is likely that such modest decreases could often be achieved with non-pharmacologic means. In addition, the quality of life appears to improve after the discontinuation of blood pressure medication.[15]

The goal of this review is to examine the evidence concerning the antihypertensive effect of exercise.

SUGGESTIVE EVIDENCE

Miall and Oldham[16] were among the first to comment on the relationship between physical activity and resting blood pressure. In 1958, they reported that workers in South Wales whose jobs required heavy labor had lower resting blood pressures than workers in more sedentary jobs (Table I). That same year a paper published in the *British Medical Journal* reported that men in physically active jobs had less hypertension (defined as diastolic blood pressure > 110) and that hypertension appeared 10–15 years later in these individuals compared with workers in less physically demanding occupations. The latter study recommended future studies of leisure physical activity.

A report of leisure physical activity among college students published in 1969 concluded that physical activity in the form of sports participation was also related to a decreased incidence of hypertension later in life.[18] These findings have recently been confirmed in two reports involving Harvard alumni.[19,20] Adding further credence to the "exercise hypothesis" is the finding at the Institute for Aerobics Research in Dallas of an inverse relationship between physical fitness and incidence of hypertension[21] and the recently published work by Reaven et al[22] finding blood pressure (both systolic and diastolic) to be related inversely to the level of physical activity.

At least one epidemiologic investigation, however, failed to observe a relationship between physical activity and resting blood pressure. In this investigation, farmers in Iowa reported greater levels of physical activity than Iowa townspeople but no difference in either resting systolic or diastolic blood pressure was seen.[23] It is interesting to note that among the studies cited, this investigation had the smallest number of subjects. For example, blood pressure data were available for only 95 male farmers. In addition, although not different from the more sedentary townspeople, the mean blood pressure of these farmers was 123/78 mm Hg, a quite acceptable value. After examining the records of 62,000 deaths, this investigation also found cardiovascular and all-cause mortality rates for farmers to be lower than those of townspeople, in spite of a higher cholesterol intake.[22] If decreased mortality is the objective, the evidence of this and other investigations [24-28] suggests that physical activity is an important factor.

EXERCISE TRAINING STUDIES

Numerous exercise intervention trials with hypertensive individuals have appeared in the scientific literature over the past 25 years. Although it is recognized that any cutoff point for "normal" blood pressure is arbitrary,[29-31] for the purposes of this review a systolic pressure of > 140 mm Hg and/or a diastolic pressure of > 90 mm Hg was considered hypertensive. A Medlines search of the literature was conducted, and all pertinent papers meeting the above conditions were reviewed. A summary of these investigations can be found in Table II. The one exception to this rule is the

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TABLE I. Results of Major Epidemiologic Studies of the Relationship Between Physical Activity and Blood Pressure

Year (Ref no.)	Population	Major Finding
1958 (16)	Residents South Wales	Workers in occupations involving heavy labor had lower blood pressures than those in less strenuous occupations
1958 (17)	Residents United Kingdom	Workers in occupations involving heavy labor had a decreased incidence of hypertension
1969 (18)	Pennsylvania University Alumni	Engaging in >5 hr per week of sports or exercise during college was associated with a decreased incidence of hypertension later in life
1982 (22)	Residents Iowa	Farmers who reported more daily physical activity than townspeople had no difference in blood pressure
1983 (20)	Harvard alumni	Playing vigorous sports in college was associated with a reduced incidence of hypertension later in life
1984 (21)	Cooper Clinic members	Physical fitness was associated with a decreased incidence of hypertension
1986 (19)	Harvard Alumni	Increased weekly physical activity was associated with a decreased incidence of hypertension
1991 (68)	Women in Southern California	Increased physical activity was associated with a decreased incidence of hypertension

1983 paper by Hagberg et al.[32] Since the subjects in this investigation were adolescents, a blood pressure greater than the 95th percentile for age and sex was considered hypertensive in this report.

The 1960s. The earliest attempt we found to evaluate the effects of an exercise program on the blood pressure of hypertensive individuals was a 1967 paper by Johnson and Grover.[33] After ten weeks of training, the blood pressure of four subjects actually rose slightly from a pretraining value of 188/103 mm Hg to 195/105 mm Hg after exercise training. Certainly these results were not encouraging for proponents of exercise. This study remains one of the few that did not show an anti-hypertensive effect of regular exercise. It would seem likely that the results could be related to the choice of an "exhausting" intensity of exercise. Later authors have suggested that mild-to-moderate exercise intensities may be more effective for reducing resting blood pressure.[34]

The 1970s. Of the six training studies found that were published in the 1970s,[35–39] only one[40] failed to find a significant decrease in resting blood pressure after training. The exercise program in this investigation had the shortest duration of training (one month), and its subjects did exhibit a trend toward decreased pressures. Significant changes have been shown, however, using a similar duration of exercise, intensity, and number of subjects.[41,42] The initial mean value of systolic pressure in this investigation was higher than that of the other investigations, so perhaps a longer intervention period was necessary. Subjects in the other five studies[35–39] reported significant decreases in both systolic and diastolic blood pressures. A variety of different exercise protocols (Table II) were used, although none were of a severely demanding nature.

TABLE II. Results of Major Investigations Evaluating the Effects of Exercise Training on Resting Blood Pressure

Year	(Ref no.)	Baseline Blood Pressure (mm Hg)	Post-training Blood Pressure (mm Hg)	Intensity/Duration/Mode
1967	(32)	188/103	195/105	10 wks, 3×/wk, 35 min run at "exhausting" pace
1970	(38)	159/105	146/93	6 mo, 2×/wk, 30–35 min, walk/jog, 60%–70% maximum heart rate
1970	(37)	140/76	136/73	6 wks, 3×/wk, 30 min, run/walk/swim/calisthenics, heart rate <140 mm Hg
1970	(34)	150/86	134/75	7 mo, 3×/wk, 60 min, squash/paddleball basketball/"isotonic-isometric maneuvers"
1973	(35)	136/90	122/82	6 mo, 1×/wk, 60–90 min, jog/calisthenics volleyball and 10–15 min daily calisthenics
1974	(36)	148/97	135/83	12 wks, 3×/wk, 30–35 min, walk/jog, 70%–85% maximum heart rate
1977	(39)	182/99	176/98	4 wks, 5×/wk, 30 min, cycle, 70% $Vo_{2\,MAX}$
1980	(45)	162/104	158/104	3 mos, 3×/wk, 60 min, walk/jog/cycle/calisthenics, 60%–70% $Vo_{2\,MAX}$
1981	(46)	182/113	161/97	3 mos, 3×/wk, 30 min, walk/jog/cycle/calisthenics, 70% maximum heart rate
1981	(46)	179/113	159/95	12 mos, 3×/wk, 30 min, walk/jog/cycle/calisthenics, 70% maximum heart rate
1981	(46)	159/95	154/97	12 mos, 3×/wk, 30 min, walk/jog/cycle/calisthenics, 75%–85% maximum heart rate
1982	(44)	145/99	136/88	4 mos, 3×/wk, 50 min, walk/jog/cycle/x-country ski, 40%–66% maximum heart rate
1985	(33)	157/104	136/90	20 wks, 3×/wk, 60 min, cycle lactate threshold, 50% $Vo_{2\,MAX}$
1985	(43)	146/94	134/87	16 wks, 3×/wk, 60 min, walk/jog 70%–80% maximum heart rate
1987	(42)	156/103	144/98	10 wks, 3×/wk, 60 min, cycle lactate threshold,
1987	(40)	143/96	132/87	4 wks, 3×/wk, 40 min, cycle, 60%–70% workload maximum
1988	(49)	145/95	135/87	10 wks, 4×/wk, 40 min, walk/cycle/jog, 85% maximum heart rate
1989	(48)	155/100	140/94	10 wks, 3×/wk, 60 min, cycle lactate threshold, 40%–60% $Vo_{2\,MAX}$
1989	(47)	158/90	151/87	9 mos, 3×/wk, 51 min, walk, 53% $Vo_{2\,MAX}$
1989	(47)	160/100	154/91	9 mos, 3×/wk, 51 min, walk/jog/cycle, 73% $Vo_{2\,MAX}$
1990	(69)	137/95 139/96	130/85 130/90	10 wks, 4×/wk, 30 min, walk/jog/cycle, 65%–80% maximum heart rate

The 1980s. The majority of the recent intervention trials have also supported the concept of an attenuation of hypertension with regular exercise. A total of 11 English reports published during the 1980s were found.[32,34,41,43–50] If a paper reported results from more than one exercise protocol, these were treated as separate studies, yielding a total of 14 different intervention trials (Table II). Of these

14 attempts, only two failed to find significant decreases in blood pressure.[46,47] One of these failed attempts[47] seems fairly easy to account for, given the circumstances. This intervention consisted of 12 months of reasonably high intensity (75%–85% of maximum heart rate exercise after 12 months of low-intensity [70% of maximum heart rate]) exercise. The initial intervention reported in this paper was three months of low-intensity exercise, resulting in a significant decrease in both resting systolic and diastolic blood pressures. After three months of cessastion of training, resting blood pressure had nearly returned to the initial levels. A second, low-intensity training phase of 12-months duration led to another significant decrease in blood pressure, but 12 months additional training at high intensity elicited no further decreases in pressure. Overall from pretraining to the end of the third exercise protocol, the mean systolic blood pressure had decreased by 28 mm Hg and the mean diastolic pressure by 16 mm Hg. It seems likely that a second cessation of training before the last exercise protocol would have resulted in an increase in blood pressure, leading again to a significant decrease after training. The finding of the other paper in this group that failed to achieve a significant decrease after exercise training[46] is more difficult to account for. The control group in this investigation actually had a larger decrease (although not statistically significant) in blood pressure than the exercise group. Only ten subjects started in the exercise group, and four of these dropped out, so poor compliance and a low number of subjects may have influenced the results. It is still difficult to account for these results considering that other investigations with similar exercise protocols and numbers of subjects have produced significant results.

A simple averaging of the results from all 22 studies listed in Table II, without correcting for the differences in numbers of subjects, yields a mean decrease of 10.36 mm Hg in systolic blood pressure and 7.59 mm Hg in diastolic pressure. When results from the studies published since 1984, which appear to have corrected many of the methodologic flaws of earlier work, are averaged, the decreases were 11 mm Hg in systolic and 7.7 mm Hg in diastolic blood pressure. Decreases of these magnitudes lie within the range recently reported to be associated with the lowest mortality.[14]

STRONGEST EVIDENCE

Previous reviews of the literature have criticized exercise trials with hypertensive patients as being poorly designed. Seals and Hagberg[2] in a 1984 review identified several major flaws common to many previous studies including classification of hypertension based on one or two blood pressure measurements, lack of control groups, and not controlling for decreases in body weight and changes in diet and sodium intake. Several recent studies have addressed these concerns in design and will be described in more detail, as they appear to represent the strongest scientific evidence for an antihypertensive effect of exercise.

Duncan et al[44] measured subjects resting blood pressure three different times on three separate days before acceptance into the study. Subjects were then randomly assigned to control or exercise groups. No significant changes in bodyweight or dietary habits occurred during the period of study. The exercise group underwent a 16-week walk/jog program at an intensity of 70%–80% of maximum heart rate. Blood pressure decreased significantly in the exercise group (146/94 mm Hg vs 134/87 mm Hg) and was unchanged in the control group.

Tanabe et al[49] measured blood pressures on four separate occasions to establish baseline levels before assigning subjects at random to exercise or control treatments. Subjects were advised to maintain normal dietary patterns. Compliance with this request was verified by monitoring 24-hour sodium excretion. Subjects in the exercise group then trained three times per week for ten weeks on a bicycle ergometer at a workload that had been determined to coincide with the onset of blood lactate accumulation (OBLA). This intensity represented about 40%–60% of maximum ($\mathrm{Vo}_{2\ \mathrm{MAX}}$). The exercise group had a significantly decreased mean blood pressure, from a pre-training value of 155/100 mm Hg to 140/94 mm Hg after training. The mean blood pressure of the non-exercising control group did not change significantly over the ten-week period. Subjects in the exercise group also had significantly decreased bodyweight, however. A mean weight loss of 3 kg was reported for the exercisers compared with a nonsignificant 0.4-kg decrease in the control group.

Hagberg et al[48] measured blood pressures 12 times over a one month period to ensure that the pre-training values were not spurious. Subjects were then randomly assigned to either control, low intensity exercise, or moderate intensity exercise groups. The mean duration of exercise for the two exercise groups was 51 minutes. Training for the low-intensity group consisted of a home walking program. This group exercised three times per week in an unsupervised setting. The mean intensity of exercise was 53% $\mathrm{Vo}_{2\mathrm{MAX}}$. The moderate intensity group engaged in supervised activity three times a week. Exercises for this group included walking, jogging, and stationary cycling. The mean training intensity for this group was 73% $\mathrm{Vo}_{2\mathrm{MAX}}$. Training duration was nine months for both groups. Dietary records and 24-hour urine collections were used to ensure that dietary modifications did not confound the results. The greatest decreases in blood pressure were seen in the low-intensity group. Systolic pressure decreased 21 mm Hg, and diastolic pressure decreased 12 mm Hg in this group. Both of these decreases were statistically significant. In the moderate intensity group, systolic blood pressure showed a nonsignificant decrease of 10 mm Hg, whereas diastolic pressure exhibited a significant 11 mm Hg decrease. The mean blood pressures of the control group did not change significantly. It is interesting to note that in this study the only significant decrease in bodyweight occurred in the moderate intensity exercise group. Whereas a 3.5-kg decrease was seen in this group, the low-intensity exercise group showed the greatest decreases in blood pressure. A nonsignificant decrease in bodyweight of 0.5 kg was seen in both the control and the low-intensity exercise groups. The findings of this investigation are especially promising. The unsupervised, low-intensity walking program, which was most successful in reducing resting blood pressure, involves minimal inconvenience and essentially no expense to the participant. Compliance is likely to be high with this type of format.

The investigation published in 1987 by Urata et al[43] also used random assignment to control or exercise groups after following subjects for four weeks to establish baseline pressures. Blood pressure medications were discontinued six weeks before the initiation of the study. Diet, smoking habits, and alcohol intake were monitored by questionnaire and 24-hour urinary sodium excretion and were found to be unchanged over the study period. The body weights of subjects did not change significantly during the ten weeks of this investigation. Nonsignificant decreases in bodyweight of 0.5 kg for the exercise group and 1.0 kg for the control group were reported. Resting blood pressures were measured at least 48 hours after exercise to exclude the possibility of the acute effects of exercise on this variable. Stationary cycling performed three times per week for ten weeks for 60 minutes per session at an intensity equivalent to the onset of blood lactate accumulation was used as the exercise intervention. Resting blood pressure decreased from 156/103 mm Hg during the last week of the pretraining observation period to 144/98 mm Hg after training in the exercise group. No change was seen in the control group. This study, in addition to controlling for the typical confounding factors such as weight loss and changes in diet, also was notable for the achievement of 100% compliance.

A slightly different approach to the question of how much exercise is needed to induce changes in blood pressure was taken by Jennings et al.[40] Rather than focusing on intensity, the investigators examined the effects of varying the frequency of exercise. Again, an observation period (six weeks) was used before intervention in order to establish baseline pressures. Subjects were then randomly assigned to three treatments of four weeks each using a Latin square design. A single blind approach was used. The staff member who measured blood pressures was unaware of which treatment the subject was undergoing at that time. The three treatments options were no exercise, exercise three days a week, and exercise seven days a week. All exercise sessions consisted of stationary cycling for 40 min at a workload equal to 60%–70% of $\mathrm{Vo}_{2\mathrm{MAX}}$. Bodyweight and sodium excretion remained unchanged during the study period. Blood pressure decreased significantly after both exercise treatments but was unchanged after the sedentary treatment. The decrease in resting blood pressure was similar for three and seven-day-per week exercise regimens. Insulin sensitivity, as measured by the glucose clamp technique, was improved only after the three-day-per-week exercise treatment. Again, the moderate approach appears to work as well or better than the more intensive interventions. This paper also described the results of four other patients who exercised for a period of one year. The mean blood pressure of this group was 135/87 mm Hg while on β-antagonist medication before initiating exercise. When taken off of the medication, the mean pressure rose to 154/99 mm Hg. After one month of exercise, blood pressure decreased to 133/90 mm Hg. After one year of exercise, the mean blood pressure of this group was 139/89 mm Hg, a value quite similar to that seen on medication. The investigators concluded that the return of hypertension after discontinuing medication can be prevented by regular exercise. They also reported that in their several years of experience using exercise training with hypertensive patients, such training had been found to be quite safe.

Another well-designed and enlightening study was recently published by Ades et al.[50] After two weeks without medications, subjects underwent nine blood pressure measurements on three separate days. They were then randomly assigned to one of three groups: placebo, nonselective β-antagonist (80 mg propranolol twice daily), or β_1-selective β-antagonist (100 mg metoprolol twice daily). A double-blind design was used. All groups exercised four times per week for ten weeks, incorporating walking, jogging, and cycle ergometry. Intensity was 85% of maximum heart rate, monitored during exercise by telemetry. Bodyweight was not significantly different after training. The mean blood pressures of both the placebo and the metoprolol group decreased significantly after exercise training, whereas the blood pressure of the propranolol group remained unchanged. The investigators concluded that medication was not needed as long as the subjects exercised. It is noteworthy that after training, medication would have been deemed still necessary in the propranolol group, but if exercise alone had been the treatment used, medication would not have been required.

Martin et al[51] have recently provided further evidence of the blood pressure lowering effect of exercise, which appears to be independent of changes in body weight, body fat, and sodium or potassium excretion. Subjects in this study were "blinded," in that the control group participated in stretching and light calisthenic exercises with a frequency and duration that was the same as the frequency and duration of the exercise intervention group. Subjects in both groups received equal attention from the training staff and were told that the study was designed to evaluate "high" vs "low" intensity exercise, so all subjects were led to believe that they were in an intervention group. Subjects in the exercise group had significantly decreased diastolic blood pressures and exhibited a trend ($p < 0.11$) toward lower systolic pressures. Subjects in the stretching/control group showed no changes in blood pressure during the initial ten-week period of the study. Subsequently, the controls underwent training for ten weeks and showed a significant decrease in both systolic and diastolic blood pressure after training.

PHYSIOLOGIC MECHANISMS OF THE ANTIHYPERTENSIVE EFFECT OF EXERCISE

Several physiologic alterations have been observed after exercise training, which may explain the anti hypertensive effect of exercise. Perhaps the simplest explanation is the decrease in heart rate after exercise training.[51] Decreased peripheral resistance has also been observed in some studies after exercise training,[35,41,52,53] although other investigations failed to observe this effect.[46] A decreased blood volume after training has also been reported in a group of hypertensive men and women.[43]

Changes in catecholamine levels represent another intriguing possibility. The decreases in total catecholamine levels of hypertensive individuals who were described as hyperadrenergic before training are associated with the decreases observed in both systolic and diastolic blood pressure after training.[44,54] Catecholamine sensitivity has also been shown to increase after exercise training in both humans[55] and rats.[56] Plasma norepinephrine has consistently been shown to decrease in humans after exercise training.[34,41,43,48,49,53] This effect has also been shown in dogs.[57] The norepinephrine response to stress is also diminished in rats that have undergone exercise training.[58]

Changes in sodium metabolism may also be a factor, although changes in sodium excretion after exercise training do not appear to be important. In humans, either no change[43,48] or a decrease[34] in sodium excretion after training have been reported, although no change in rats[59] has been observed. However, a decrease in the intracellular erythrocyte sodium concentration has been noted,[59] possibly caused by alterations in the sodium/lithium countertransport system.[60,61]

Evidence for changes in glucose metabolism after exercise training, which may effect blood pressure, also appears in the literature. Hypertensive individuals have been reported to have impaired glucose tolerance and increased fasting levels of insulin not explained by body weight.[62] Insulin sensitivity has been shown to improve after exercise training in both obese individuals[63] and non-obese hypertensive patients.[41] One exercise intervention trial undertaken with a group of obese women reported a mean decrease in blood pressure from 134/87 mm Hg before training to 125/80 mm Hg after training; these decreases in blood pressure were not correlated with the decreases in body weight. However, patients with the greatest decreases in blood pressure had higher initial insulin and blood glucose levels.[64] Hypertensive persons have an increased cardiac output at rest.[65] DeFronzo has speculated that increased insulin levels may increase renal sodium reabsorption, leading to increased extracellular fluid volume, which could increase cardiac output and, in turn, increase blood pressure.[66] On the other hand, decreased blood pressure after exercise training with no change in fasting insulin levels has been reported.[48]

Other exercise associated differences that represent possible mechanisms for decreased blood pressure include increases in serum taurine[49] and prostaglandin E synthesis[34] and decreases in plasma viscosity.[67]

CONCLUSIONS

The preponderance of evidence appears to recommend exercise training as an effective treatment for mild and moderate hypertension. With few exceptions, studies of exercise training show that it decreases the resting blood pressure of hypertensive individuals. Many of the early investigations that observed a decrease in blood pressure after exercise training have been criticized for a variety of design flaws. More recent studies have addressed these shortcomings by using pre-training observation periods, appropriate control groups, single- and double-blind methodology, and by controlling for weight loss and diet. The better designed studies have found an even larger and more consistent decrease in resting blood pressure after training. Some reports indicate that medication may be unnecessary for many patients if they adhere to a regular exercise program. A variety of modes of training, including walking, running, cycling, and cross-country skiing appear to be effective. As few as two days per week and as many as seven have been reported to be effective in significantly reducing blood pressure. Exercise intensities as low as 40%–50% Vo$_{2\text{MAX}}$ and as high as approximately 80% Vo$_{2\text{MAX}}$ seem to be effective, although very high intensity (exhausting) exercise may not result in decreased blood pressure. Although more frequent and higher intensity programs have produced favorable results, the more moderate approach may be better tolerated and less likely to result in injury. Simple, unsupervised walking programs with a frequency of three sessions per week have been shown to produce results comparable to the more costly and inconvenient supervised programs. Additional benefits, such as improved insulin sensitivity, may be associated with the more moderate formats.

Changes in dietary habits may also be effective in treating hypertension.[68] Although diet and lifestyle modifications have often been derided as doomed to failure due to poor compliance, compliance with pharmacologic treatment is also poor and carries the added burdens of cost, decreased quality of life, and perhaps even increased mortality. Perhaps a 30-minute walk after dinner is not such a bad alternative, or as Paffenbarger[26] has written "life-style modification is regarded as a theoretical rather than a practical concept. In fact, physical exercise level can be modified and has direct influences on other life-style elements: reducing the desire to smoke, favoring reduction or avoidance of obesity, countering dietary impulses and psychological stresses, and inducing alteration of unfavorable environment or family circumstances."

REFERENCES

1. Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. Non-pharmacologic approaches to the control of high blood pressure: Final report of the sub-committee on non-pharmacologic therapy of the 1984 Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* 1986; 8:444–467.
2. Seals DR, Hagberg JM: The effect of exercise training on human hypertension: A review. *Med Sci Sports Exer* 1984; 16:207–215.
3. Chobanian AV, Alderman MH, De Quattro V, et al: The 1988 Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* 1988; 148:1023–1038.
4. Gifford RW: Management and treatment of essential hypertension, including malignant hypertension and emergencies, in Genest J, Kuchel O, Hamet P, Chantin M (eds): *Hypertension: Physiopathology and Treatment*, ed 2. New York, McGraw Hill, 1983, pp 1127–1170.
5. McCorvey E, Jackson TW, McKenney JM, et al: Does antihypertensive therapy influence quality of life. *Clin Pharm* 1989; 8:359–364.
6. Frohlich ED: Physiologic considerations in left ventricular hypertrophy. *Am J Med* 1983; 75:12–18.
7. Staessen J, Cattaert A, Fagard R, et al: Epidemiology of treated, compared to untreated hypertension, in Genest J, Kuchel O, Hamet P, Chantin M (eds): *Hypertension: Physiopathology and Treatment*, ed 2. New York, McGraw Hill, 1983, pp 1069–1093.
8. Genest J, Kuchel O, Hamet P, et al: Hypertension: *Physiopathology and Treatment*, ed 2. New York, McGraw Hill, 1983.
9. Lichtenstein MJ: Should we be treating high blood pressure in persons over 65? *Compr Ther* 1988; 14:54–59.
10. Perloff G: Hypertension in women. *Cardiovasc Clin* 1989; 19:207–241.
11. Multiple Risk Factor Intervention Group. Risk factor changes and mortality results. *JAMA* 1982; 248:1465–1477.
12. Kolata G: Heart study produces a surprising result. *Science* 1982; 218: 31–32.
13. Moser M: Less severe hypertension—when and how it should be treated. *Compr Ther* 1988; 14:11–18.
14. Alderman MH, Ooi WL, Madhaven S, et al: Treatment induced blood pressure reduction and the risk of myocardial infarction. *JAMA* 1989; 262:920–924.
15. Aberg H, Tibblin G: Addition of non-pharmacological methods of treatment in patients on antihypertensive drugs: Results of previous medication, laboratory tests and life quality. *J Intern Med* 1989; 226:39–46.
16. Miall WE, Oldham PD: Factors influencing arterial blood pressure in the general population. *Clin Sci* 1958; 17:409–444.
17. Morris JN, Crawford MD: Coronary heart disease and physical activity of work. *Brit Med J* 1958; 2:1485–1496.
18. Paffenbarger RS, Thorne MC, Wing AL: Chronic disease in former college students: VIII Characteristics in youth predisposing to hypertension in later years. *Am J Epidemiol* 1968; 88:25–32.
19. Paffenbarger RS, Wing AL, Hyde RT, et al: Physical activity, all-cause mortality, and longevity of college alumni. *N Engl J Med* 1986; 314:605–613.
20. Paffenbarger RS, Wing AL, Hyde RT, et al: Physical activity and incidence of hypertension in college alumni. *Am J Epidemiol* 1983; 117:245–257.
21. Blair SN, Goodyear NN, Gibbons LW, et al: Physical fitness and the incidence of hypertension in healthy normotensive men and women. *JAMA* 1984; 252:487–490.
22. Reaven PD, Barrett-Connor F, Edelstein S: Relationship between leisure-time physical activity and blood pressure in older women. *Circulation* 1991; 83: 559–565.
23. Pomrehn PR, Wallace RB, Burmeister LF: Ischemic heart disease mortality in Iowa farmers. The influence of lifestyle. *JAMA* 1982; 248:1073–1076.
24. Morris JN, Heady JA, Raffle PAB, et al: Coronary heart-disease and physical activity of work. *Lancet* 1953; 2:1053–1057.
25. Kannel WB, Belanger A, D'Agostino RD, et al: Physical activity and physical demand on the job and risk of cardiovascular disease and death: The Framingham Study. *Am Heart J* 1986; 112:820–836.
26. Paffenbarger RS, Hyde RT, Wing AL, et al: A natural history of athleticism and cardiovascular health. *JAMA* 1984; 252:491–495.
27. Garcia-Palmieri MR, Costas R, Cruz-Vidal M, et al: Increased physical activity: A protective factor against heart attacks in Puerto Rico. *Am J Cardiol* 1982; 50:749–755.
28. Ewing BJ, Irving JB, Kerr F, et al: Static exercise in untreated systemic hypertension. *Br Heart J* 1973; 35:413–421.
29. Morris JN, Chave SP, Adam C, et al: Vigorous exercise in leisure-time and the incidence of coronary heart disease. *Lancet* 1973; 1:333–339.
30. Pickering G: Hypertension: Definitions, natural histories and consequences. *Am J Med* 1972; 52:570–583.
31. WHO Expert Committee: World Health Organization Expert Committee: Arterial Hypertension, WHO Technical Report Series, 1978; 628:1.

32. Hagberg JM, Goldring D, Ehsani AA, et al: Effect of exercise training on the blood pressure and hemodynamic features of hypertensive adolescents. *Am J Cardiol* 1983; 52:763–768.

33. Johnson WP, Grover JA: Hemodynamic and metabolic effects of physical training in four patients with essential hypertension. *Can Med Assoc J* 1967; 96:842.

34. Kiyonaga, A, Arawaka K, Tanaka H, et al: Blood pressure and hormonal responses to aerobic exercise. *Hypertension* 1985; 7:125–131.

35. Hanson JS, Nedde WH: Preliminary observations on physical training for hypertensive males. *Circ Res* 1970; 27(suppl 1):I49–I53.

36. Choquette G, Ferguson RJ: Blood pressure reduction in "borderline" hypertensives following physical training. *Can Med Assoc J* 1973; 108:699–703.

37. Bonnano JA, Lies JE: Effects of physical training on coronary risk factors. *Am J Cardiol* 1974; 33:760–764.

38. DeVries HA: Physiological effects of an exercise training regimen upon men aged 52–88. *J Gerontol* 1970; 25:325–336.

39. Boyer JL, Kasch FW: Exercise therapy in hypertensive men. *JAMA* 1970; 211:1668–1671.

40. Ressl J, Chrastek J, Jandova R: Haemodynamic effects of physical training in essential hypertension. *Acta Cardiologica* 1977; 32:121–133.

41. Jennings G, Nelson L, Korner P, et al: The place of exercise in the long-term treatment of hypertension. *Nephron* 1987; 47(suppl 1):30–33.

42. Jennings GL, Nelson L, Nestel P, et al: The effect of changes in physical activity on major cardiovascular risk factors, haemodynamics, sympathetic function and glucose utilization in man: A controlled study of four levels of activity. *Circulation* 1986; 73:30–40.

43. Urata H, Tanabe Y, Kiyonaga A, et al: Antihypertensive and volume depleting effects of mild exercise on essential hypertension. *Hypertension* 1987; 9:245–252.

44. Duncan JJ, Farr JE, Upton SJ, et al: The effects of aerobic exercise on plasma catecholamines and blood pressure in patients with mild hypertension. *JAMA* 1985; 254:2609–2613.

45. Kukkonen K, Rauramaa R, Voutilainen E, et al: Physical training of middle-aged men with borderline hypertension. *Ann Clin Res* 1982; 14(suppl 34):139–145.

46. DePlaen JF, Detry JM: Hemodynamic effects of physical training in established arterial hypertension. *Acta Cardiol* (Brux) 1980; 35:179–188.

47. Roman O, Camuzzi AL, Villalon E, et al: Physical training program in arterial hypertension: A long-term prospective follow-up. *Cardiology* 1981; 67: 230–243.

48. Hagberg JM, Montain SJ, Martin WH, et al: Effect of exercise training in 60- to 69-year-old persons with essential hypertension. *Am J Cardiol* 1989; 64:348–353.

49. Tanabe Y, Urata H, Kiyonaga A, et al: Changes in serum concentrations of taurine and other amino acids in clinical antihypertensive exercise therapy. *Clin Exp Hypertens* 1989; 11:149–165.

50. Ades PA, Gunther PG, Meacham CP, et al: Hypertension, exercise, and beta-adrenergic blockade. *Ann Intern Med* 1988; 109:629–634.

51. Martin JF, Duhnert PM, Cushman WC: Controlled trial of aerobic exercise in hypertension. *Circulation* 1990; 81:1560–1567.

52. Frick MH, Konettinen A, Sarajas HS: Effects of physical training on circulation at rest and during exercise. *Am J Cardiol* 1963; 12:142–147.

53. Nelson L, Jennings G, Esler M, et al: The effect of changing levels of physical activity on blood pressure and haemodynamics in patients with essential hypertension. *Lancet* 1986; ii:474–476.

54. Duncan JJ, Hagan RD, Upton J, et al: The effects of an aerobic exercise program on sympathetic neural activity and blood pressure in mild hypertensive patients. (abstract) *Circulation* 1983; 68:III–285.

55. Bjorntorp P: Hypertension and exercise. *Hypertension* 1982; 4(suppl 3):III56–III59.

56. Askew EW, Dohm GL, Huston RL, et al: Response of rat tissue lipase to physical training and exercise. *Proc Soc Exp Biol* (NY) 1972; 141:123–129.

57. Bove AA, Dewey JD, Tyce GM: Increased conjugated dopamine in plasma after exercise training. *J Lab Clin Med* 1984; 104:77–85.

58. Cox RH, Hubbard JW, Lawler JE: Exercise training attenuates stress-induced hypertension in the rat. *Hypertension* 1985; 7:747–751.

59. Shepard RE, Kuehne MC, Kenno KA: Attenuation of blood pressure increases in Dahl salt-sensitive rats by exercise. *J Appl Physiol* 1982; 52:1608–1613.

60. Hespel P, Lijnen P, Fagard R, et al: Changes in erythrocyte sodium and plasma lipids associated with physical training. *J Hypertension* 1988; 6:159–166.

61. Adragna NC, Chang JL, Morey MC, et al: Effects of exercise on cation transport in human red cells. *Hypertension* 1985; 7:132–139.

62. Berglund G, Larsson B, Anderson O, et al: Body composition and glucose metabolism in hypertensive middle-aged males. *Acta Med Scand* 1976; 200:163–169.

63. Bjorntorp P, Holm G, Jacobsson B, et al: Physical training in human hyperplasic obesity. IV. Effects on the hormonal status. *Metabolism* 1977; 26: 319–328.

64. Krotkiewski M, Mandroukas K, Sjostrom L, et al: Effects of long-term physical training on body fat, metabolism and blood pressure in obesity. *Metabolism* 1979; 28:650–658.

65. Sannerstedt T: Hemodynamic findings at rest and during exercise in mild arterial hypertension. *Am J Med Sci* 1969; 258:70–79.

66. DeFronzo RA: Insulin and renal sodium handling: clinical implications. *Int J Obesity* 1981; 5(suppl 1):93–104.

67. Letcher RL, Pickering TG, Chen S, et al: Effects of exercise on plasma viscosity in athletes and sedentary normal subjects. *Clin Cardiol* 1981; 4:172–179.

68. Beilin LJ, Armstrong BK, Margetts BM, et al: Vegetarian diet and blood pressure. *Neph* 1987; 47(suppl 1):37–41.

Skin changes associated with hematologic and oncologic diseases in children

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The skin functions as a hematopoietic organ in the developing fetus. At the yolk stage, blood islands located within the endothelium produce blood cells; these subsequently regress. Extravascular or visceral hematopoiesis begins initially in the liver, followed by the skin, spleen, and lymph nodes by the ninth week of gestation.[1,2] Blood cell production in the viscera reaches its peak by the fifth and sixth fetal months. It then wanes, as hematopoiesis in the bone marrow, beginning in the fourth month, becomes the main source of blood cell production at birth.[1,2] At birth, all of the bone marrow is fully active, but the total marrow space is limited compared with that of the older child, because much of the newborn skeleton is still cartilaginous. In fact, at birth the bone marrow volume is equivalent to the volume of hematopoietic cells.[1] To increase blood cell production, hypertrophy of the marrow or reversion to extramedullary hematopoiesis must occur. As a consequence, the skin may revert back to production of blood cells.

The dermal mesenchyme initially produces megaloblastoid red cells at the 8 cm stage in the fetus. This is followed by production of normoblastic red cells; by the fifth month of gestation, granulocyte, and platelet production occurs. This extramedullary blood cell production by the skin disappears by the last two months of gestation. At that time, the hematopoietic cells are removed by phagocytic cells.[3] This hematopoietic function of the skin may return in the embryo or young infant and, less commonly, may also occur in older children and adults. Circumstances that favor a return to dermal hematopoiesis include viral infections, severe anemia, and malignancies.

In addition, the skin is an immunologic organ.[4] The Langerhans cells derived from bone marrow monocytic precursor cells are normal skin cells participating in the host's immune response. These cells and other monocytic cells are involved in both benign and malignant disorders included in the histiocytoses of infancy and childhood.[5]

VIRAL INFECTIONS

Dermal erythopoiesis occurs in infants with intrauterine viral infections. The earliest reports describing this were in infants with congenital rubella.[6–8] In these cases, the newborn infant is found to have dark blue and violaceous papules and macules, 2–10 mm in diameter, scattered over the body, and usually quite prominent on the face. When first described, the purpuric appearance of the lesions and the frequent concomitant thrombocytopenia caused them to be considered hemorrhagic papules. Biopsies of these skin lesions showed dermal infiltrates of erythropoietic cells and rarely myeloid precursors. The lesions usually fade over the first few weeks of life and are gone by the sixth week.

The clinical appearance of an infant with these skin lesions has given rise to the term blueberry muffin syndrome.[9] The dermatologic picture has been seen in infants suspected of having a TORCHES (eg, toxoplasma, rubella, cytomegalic inclusion disease [CID], herpes, or syphilis infection). In particular, this picture has been observed with congenital cytomegalovirus infection and in neonates with toxoplasmosis and rubella.[10,11] In one study, six infants with intrauterine viral infections with cytomegalovirus or rubella showed a macular "hemorrhagic purpuric rash."[12] Skin biopsies of the lesions revealed dermal erythropoiesis and no hemorrhage. Anemia was present in only one infant, and there was thrombocytopenia in all six infants. It was speculated that the dermal erythropoiesis was due to persistence of the potential of dermal mesenchyme to form erythroid percursors at the time of the viral infection.

NEONATAL ANEMIA

In rare instances of severe anemia occurring in utero, persistence of dermal erythropoiesis can be observed in the neonate. In these cases, the anemic newborn has multiple, small (2–8 mm), red, violaceous macular and papular skin lesions scattered over the entire body. Biopsy of the lesions reveals a diffuse infiltrate of erythroid precursors in the lower dermis. The lesions disappear within a short time after birth. These skin lesions have been observed in newborn infants with hereditary spherocytosis,[13] in the donor twin when there has been a twin-to-twin transfusion syndrome with a monochorial placenta,[14] and in infants born to mothers with whom there is Rh incompatibility.[15]

The "blueberry muffin" appearance indicating dermal erythropoiesis is not common in the anemic newborn; it is more common in the newborn infant with an intrauterine viral infection. In one case, a 38-week newborn had discrete, 2–6 mm, paleblue macules over the body.[13] The baby's initial hematocrit was 25%. The skin lesion began to fade by 72 hours and disappeared in one week. The baby was diagnosed as having hereditary spherocytosis at three months of age. It was postulated that the dermal erythropoiesis was provoked by intrauterine hypoxia resulting from the hemolysis.

Dermal erythropoiesis has been described in only one case report of RH hemolytic disease of the newborn.[14] The infant was born at 33 weeks and, in addition to the multiple

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macular skin lesions, he had massive hepatosplenomegaly. Biopsy of the skin lesions confirmed the clinical impression of dermal erythropoiesis. The investigators noted that severely anemic infants with Rh hemolytic disease usually have massive hepatosplenomegaly. Perhaps the erythropoietic response by the marrow in the liver and spleen is sufficient to respond to the hypoxic stress without reactivation of dermal erythropoiesis. The response of the fetus to hypoxia may depend on the timing of the insult in relation to differentiation of the hematopoietic system.

Finally, dermal erythropoiesis has been reported to occur in the anemic donor twin when there has been a significant intrauterine transfusion. Two cases of twin transfusion syndrome have been described.[15] There were significant weight difference and hemoglobin disparities in both twin pairs. In both cases, the anemic donor twin had bluish macular lesions scattered over the upper trunk and face. Pathologic confirmation of dermal erythropoiesis was obtained in both cases.

Dermal erythropoiesis occurring in the donor twin in a twin transfusion situation is uncommon when there has been vascular anastomosis between the two sets of fetal vessels in a monochorial placenta. In a series of 482 newborn twin pairs, there was 35 instances of twin-twin transfusion. No dermal erythropoiesis was observed in the donor twins.[16] Similarly, in a review of 19 cases of the twin transfusion syndrome, dermal erythropoiesis in the donor twin was not noted.[17] The mortality rate was high, and only in four cases did both twins survive. In ten cases, both twins died, and in five cases only one twin of the pair survived. The donor twins who survived were not significantly anemic. Thus, the phenomenon of dermal erythropoiesis in the anemic donor twin must depend on the duration and severity of the erythropoietic stress in utero. In this study, there was a significant difference in weight and hematocrit between the twins at birth.[15]

Leukemia

The infant with leukemia frequently has cutaneous signs of hematopoietic activity.[18–23] About half the infants with congenital leukemia will show skin nodules at presentation.[18,19] In the infant beyond the newborn period and in the young child, leukemic nodules are not as common. The nodules vary in size from 0.5 to 3.0 cm in diameter. They may be few to multiple in number and are found anywhere on the skin. The nodules are blue-purple and slate-grey in color. The hemorrhagic appearance of the nodules may be confused with the blueberry muffin appearance of the skin in the infant with a congenital viral infection.[24] Biopsy of the lesions reveals a dense dermal infiltration of leukemic cells. The skin nodules may accompany the diagnosis of leukemia on the basis of the findings of blood and bone marrow studies, or the appearance of the nodules may precede the diagnosis of leukemia by many months.[22,24,25] Figure 1 shows a localized leukemic nodule (one of three) in a three-month-old infant with acute myeloid leukemia.

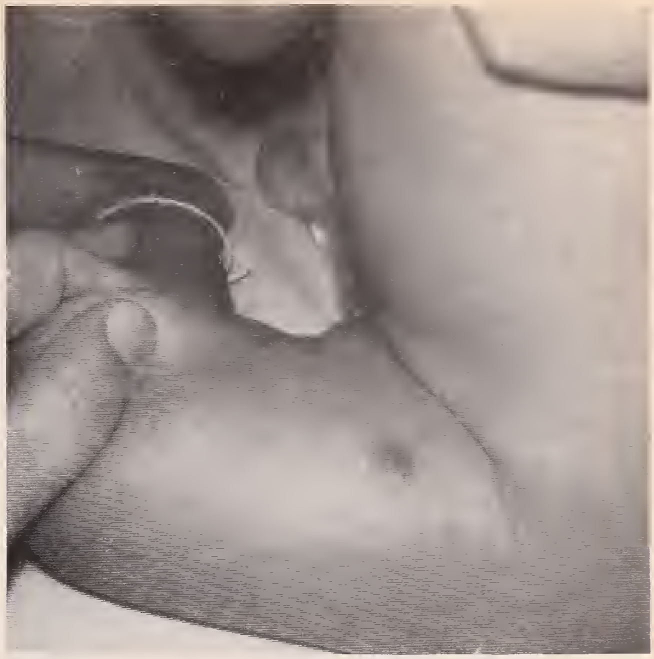

FIGURE 1. A leukemic nodule in an infant with acute meyloid leukemia.

These skin nodules have been observed in all types of acute leukemia seen in infants. They have also been noted in infants with Down's syndrome who have transient myelodysplasia, previously considered by some to be congenital leukemia.[26,27] Figure 2 shows a newborn with Down's syndrome with multiple macular and papular red-violaceous skin lesions. The infant had peripheral blood and bone marrow findings consistent with a transient myelodysplastic syndrome or congenital leukemia. Interestingly, these nodules have also been observed in newborn infants with normal karyotypes in whom there had been spontaneous remission of congenital leukemia.[27–29] As the leukemic blasts disappear from the bone marrow and from other extramedullary sites, the skin nodules also disappear. The increased incidence of dermal leukemic infiltrates in the newborn infant has been ascribed to the increased blood supply to the skin of the neonate as compared to the older infant.[21] One observer postulated that the leukemic skin nodules in the newborn may represent the early onset of intrauterine leukemia in the dermal mesenchyme.[18] Leukemic skin infiltrates are also seen in the older infant and child, particularly in those with acute monocytic leuke-

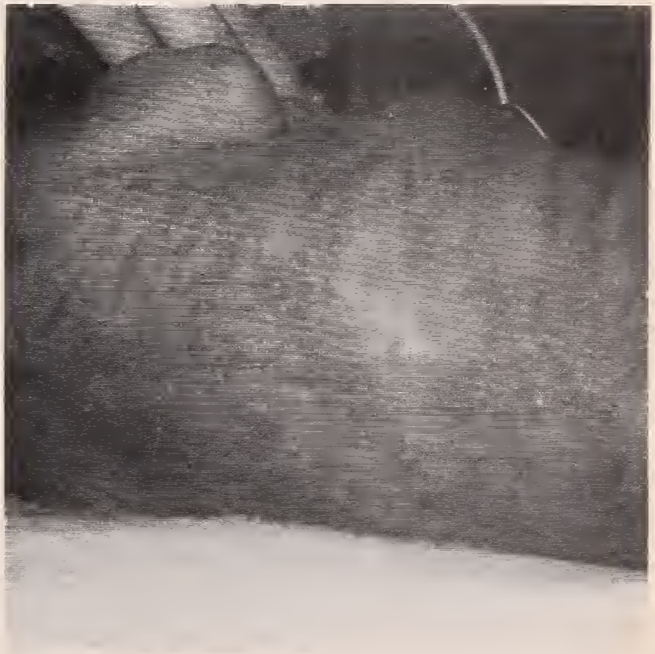

FIGURE 2. Multiple macular and papular leukemic skin lesions in a newborn with Down's syndrome.

mia,[20,30,31] as well as with acute lymphoblastic leukemia.[32] In adults, the dermal infiltrates of hematopoietic elements can be seen in the form of myeloid metaplasia.[32–35]

One study reported three patients with acute lymphoblastic leukemia who had very late recurrence of their disease at 9, 11, and 19 years after discontinuation of chemotherapy.[36] Two of the patients showed skin lesions at the time of relapse. At the time of bone marrow relapse, one patient had an isolated eyelid infiltration of leukemic cells, and another patient had multiple skin nodules. Another report described a 21-year-old women in whom acute lymphoblastic leukemia had been diagnosed at age 13 years. After 68 months off of chemotherapy, she presented with a large temporofacial mass, which biopsy showed to be a dermal and soft tissue tumor comprising lymphoblasts.[37]

LYMPHOMA

Lymphoma in childhood may present as isolated skin involvement although the skin is not a common site.[35–43] Lymphoma of the skin is more common in infants, but it can also occur in the older child. In a review of 42 cases of childhood lymphoma involving the head and neck, there were five cases of isolated cutaneous lymphoma.[44] The child most frequently presents with a small papular or mass lesion located on the scalp or less commonly in the preauricular area, but skin involvement by lymphoma can occur anywhere on the body as an isolated mass or as multiple nodules.[38–43.]

Biopsy of the skin lesion shows a dense dermal infiltrate of lymphoblasts. In a review of the immunophenotyping of 116 children with non-Hodgkin's lymphoma, the majority of the tumors were found to be either B cell or T cell in type.[41] However, in eight cases the tumors were typed as non-B, non-T CALLA (common acute lymphoblastic leukemia antigen) positive. Four of the patients had isolated skin lymphoma, and these patients were much younger than the mean age of 7 to 9 years, the age when childhood lymphoma is most common. Two of the patients were 18 months, another was 19 months, and one was 3 years old. In two cases of childhood lymphoma confined to the skin, both patients had scalp masses.[40] Immunophenotyping showed the tumors to be non-T, non-B, and CALLA-positive in type. It appears that most cases of childhood lymphoma confined to the skin may be of the pre-B or early pre-B cell immunophenotype.[38,40,41] Usually the histology is of the diffuse type, but the nodular type may also be seen.[42] In Figure 3, two different nodular skin lesions on the back of a 9-month-old infant are seen. Biopsy of one of the lesions showed non-B, non-T cell, CALLA-positive lymphoma confined to the dermis. Bone marrow examination was normal. Isolated skin lymphoma is more common in the infant and young child than in the older child.[41]

Although the B-cell phenotype is most frequently seen in lymphomas with involvement limited to the skin in children, cases with the T-cell type may also occur.[45,46] Another type of lymphoma, the Ki-1 lymphoma, is characterized by diffuse large cell histology and frequent involvement of the skin. In one report of six cases of Ki-1 lymphoma in children, all six patients had skin involvement at diagnosis.[46] In contrast to the bland-appearing masses seen with the pre-B type of non-Hodgkins' lymphoma, the skin lesions with Ki-1 lymphoma may appear as inflammatory papules. They may be initially diagnosed and treated as an abscess or as insect bites.

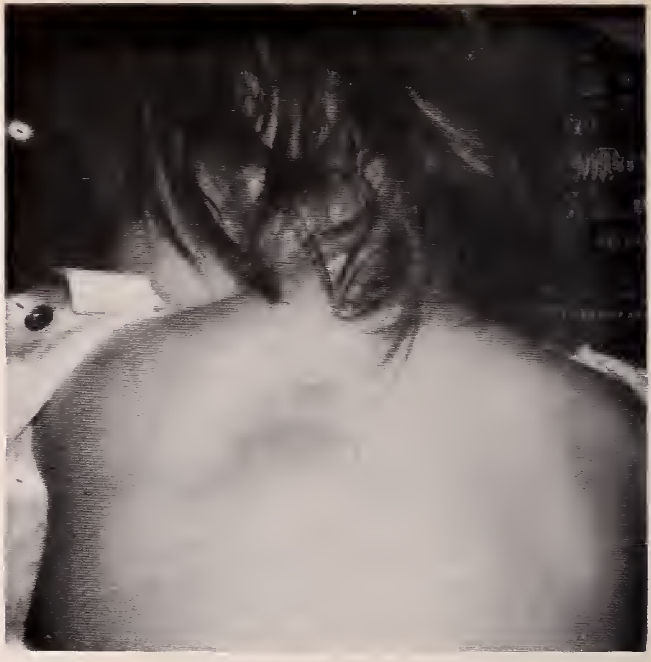

FIGURE 3. Lymphomatous skin inflitrates in a nine-month-old infant.

NEUROBLASTOMA

Skin nodules in the newborn and in the older infants may be due to metastatic neuroblastoma.[47] Such lesions are not seen in older children with neuroblastoma. The skin nodules in infants with neuroblastoma are firm, bluish in color, and may be few in number or multiple, occurring anywhere on the body.[48–54] In one case, blanching subcutaneous nodules were observed in an infant with neuroblastoma. The blanching was due to release of catecholamines from the tumor cells, resulting in cutaneous vasoconstriction.[54]

The skin nodules in an infant with neuroblastoma may continue to increase in number during the newborn period. If the child survives, the nodules may gradually involute or disappear completely.[50,51] Biopsy of the regressing nodules shows fibrosis, calcification, and generally, a small number of mature ganglion cells.[50,51] One report describes an infant who had many subcutaneous nodules and an abdominal mass at birth.[49] The diagnosis of neuroblastoma was made on the basis of a biopsy of one of the skin nodules and an elevated urinary vanillylmandelic acid (VMA). The patient was only observed and no treatment was given. The tumor originated in the adrenal gland and was metastatic to the skin and bone marrow. Maturation of the tumor was monitored by serial biopsies of the skin nodules. Neuroblastoma cells were seen at four and a half months, but the skin biopsy of the nodules at 16, 36, and 76 months showed only fibrosis and the presence of mature ganglion cells. When the patient was three years of age, the left adrenal gland was removed. Histopathologic examination showed no viable tumor.

The phenomenon, therefore, of maturation of neuroblastoma when the tumor is diagnosed in the new-born period may be observed by studying changes in the subcutaneous nodules.[53] In Figure 4, a large sacral mass in a 3-week-old infant is shown. This was a small nodule, 1.0 cm in diameter at birth, and it quickly increased in size to 3 × 4 cm. The infant had a pelvic neuroblastoma. A biopsy of the sacral mass showed a dermal infiltrate of neuroblastoma cells.

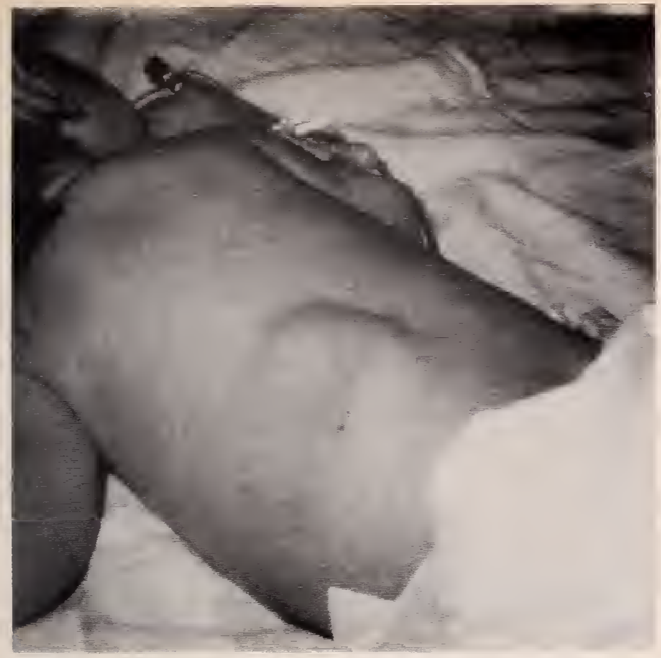

FIGURE 4. Sacral mass in a three-week-old infant with a pelvic neuroblastoma.

OTHER TUMORS

Besides leukemia, lymphoma, and neuroblastoma, there are isolated case reports of other tumors that have metastatic foci in the skin in the neonate and infant. One infant was noted to have multiple cherry red, slightly violaceous, nodular skin lesions scattered over the back, trunk, and neck.[55] A biopsy of a skin nodule revealed a dermal infiltrate of adrenocortical carcinoma. Computed tomography of the abdomen showed a right adrenal mass. No further treatment was given after adrenalectomy, and the skin tumors regressed over a four-month period. The infant at one year of age showed no signs of recurrent tumor. Another infant, two months of age, had two scalp masses and subsequent enlargement of cervical lymph nodes.[56] At five months of age, a lymph node biopsy yielded a diagnosis of adrenocortical carcinoma. The infant died from the tumor at 17 months of age. It is of interest that the mother of this infant had been treated during pregnancy with hydroxyprogesterone.

Finally, a three-month-old infant had pink papules on the cheek which, by 12 months of age, had developed into three red-bluish tumor nodules.[57] Biopsies of the skin nodules revealed tumor infiltration of the dermis and underlying muscle by rhabdomyosarcoma. At the time of diagnosis, no metastatic foci were found. Five weeks later the infant showed metastasis in an enlarged submandibular lymph node. Thus, it appears that rhabdomyosarcoma can occur in the skin as the primary site in an infant.

CONGENITAL SELF-HEALING RETICULOHISTIOCYTOSIS

Congenital self-healing reticulohistiocytosis is a recently described, rare dermatologic entity seen in the infant.[58–65] At birth, infants with this disease have red, violaceous papular lesions, sometimes vesicular, 0.5–1.5 cm in diameter, and scattered over the body.[58,59,61,62,64,65] Some cases have only a few scattered lesions at birth.[60,63] The lesions may increase in number for a short period after birth, but then spontaneous involution occurs. By three and a half months, the lesions are usually involuted, leaving a small hyperpigmentated area.[61] Biopsy reveals a dense dermal infiltration of large pale histiocytes with eosinophilic cytoplasm. Scattered eosinophils may also be found.[63] Electron microscopy of the histiocytes may show the Birbeck granules characteristic of Langerhans cells.[63]

Infants with this disease usually have involvement of the skin only, but a few cases have been reported with lung, bone, and skin involvement.[62,65] The entity of congenital self-healing reticulohistiocytosis in the infant is distinguished from the more common Letterer-Siwe disease by the lack of multiple organ involvement.[66] Infants and children with histiocytosis X usually have a red seborrheic rash with no nodular component; other organs may be involved such as liver, lung, lymph nodes, or bone. Biopsy of the skin shows more epidermal involvement by the histiocytic process. Letterer-Siwe disease as a form of histiocytosis X in the infant has a poor prognosis, in contrast with the self-healing reticulohistiocytosis.[65] Histiocytosis X has been reviewed in detail by Nezalof and Favara et al.[66,68]

A review of 33 reported cases of self-healing reticulohistiocytosis confirmed the uniformly good prognosis in these patients.[61] It is important to distinguish the skin rash of neonates with congenital self-healing retriculohistiocytosis from dermal manifestations of leukemia, sepsis, and neuroblastoma, with which it can be confused.[59] Self-healing reticulohistiocytosis represents a proliferation of dermal Langerhans cells, derived from the bone marrow precursors of this cell line.[67,68] This proliferation of Langerhans cells is seen at birth or, rarely, shortly after birth; the cells commonly involute within the first few months of life, occasionally presenting up to age six months.[61]

SKIN CHANGES CAUSED BY CHEMOTHERAPY

Dermatologic changes are not uncommon in patients who are being given chemotherapy. The more common dermatologic findings include acne associated with corticosteroids, Raynaud's phenomena, and skin pigmentation with bleomycin and adriamycin, hyperpigmentation of the nails with adriamycin, urticaria with adriamycin, and perioral eczematous rash with methotrexate.[69] Some of the more unusual skin changes seen in children during or after completing chemotherapy include acral erythema (Burgdorf's reaction), eczematous skin rash after completion of chemotherapy for childhood acute lymphoblastic leukemia, and the occurrence of increased numbers of melanocytic nevi after completing chemotherapy.

Burke et al[70] described painful acral erythema in a three-year-old child receiving high-dose methotrexate for acute lymphoblastic leukemia. Sharply demarcated areas of redness and swelling of the fingers and toe pads occurred two days after completion of a course of high-dose methotrexate. The lesions resolved after blister formation and desquamation. Painful acral erythema has been observed in adults receiving various chemotherapeutic agents, including cytosine arabinoside, adriamycin, and 5-fluorouracil, as well as after high-dose methotrexate.[71–74] This syndrome was observed in four out of 53 patients receiving high-dose cytosine arabinoside for acute myeloid leukemia. Acral erythema usually occurs within 14 days of receiving the drug and resolves over a seven to ten day period.[72] A similar skin reaction was observed in 17 patients receiving 5-fluorouracil and in one patient receiving adriamycin.[73] In three patients, acral erythema occurred after high-dose methotrexate therapy.[74] The syndrome occurs earlier with methotrexate in contrast with the other drugs, occurring within two days of drug administration.

In children who have completed chemotherapy, an increased number of benign nevi may be seen.[75,75] Thirty-two pediatric patients who had completed chemotherapy for different childhood cancers were compared with 32 age-matched controls. The mean number of moles in the cancer patients was significantly increased. This was true for moles both less than 3.0 mm and greater than 3 mm in size. Similarly, it was found that the mean number of moles in children who had received salvage chemotherapy six to 12 months after completing initial therapy was significantly more than in their siblings. The moles were benign in appearance. The authors of these reports emphasize the need for close follow-up of the moles.

A red papular and sometimes scalely facial rash has been reported to occur in pediatric patients after therapy with 6-mercaptopurine;[77,78] it has been reported to occur in half the patients shortly after completing 6-mercaptopurine therapy, resolving over a 3–4 week period. Shaw et al[77] described the rash on the trunk in a few of the patients.

GRAFT-VS-HOST DISEASE

Another aspect of the skin as an immunologic organ is graft-vs-host disease, wherein the skin is one of the target organs affected by transfusion-derived foreign lymphocytes. Graft-vs-host disease occurs after allogeneic bone marrow transplantation and, less frequently, after blood transfusion.[79–81]

The first reported cases after blood transfusion were in infants with immunodeficiency diseases. These infants received unirradiated blood products containing viable lymphocytes.[79] More recent reports of graft-vs-host disease after blood transfusions have been reported in premature infants; in infants requiring exchange transfusion in utero or after birth for erythroblastosis fetalis; and in patients on immunosuppressive chemotherapy for leukemia, lymphoma, and other tumors. Transfusion related graft-vs-host disease has also been reported to occur in adult patients after cardiac bypass surgery and in patients receiving blood transfusions from closely related relatives.[79,81–83]

The dermatologic changes associated with graft-vs-host disease consist of a red, macular papular rash distributed over the body. The rash may be more intense in the palms and soles. It is sometimes pruritic and may progress to bullous formation. The rash usually occurs from one week to two months after transfusion, with a mean time of three weeks.[81]

In bone marrow transplantation or in the post-transfusion setting, it may be difficult to distinguish the rash of acute graft-vs-host disease from a viral or drug reaction.[78] Skin rash is the most frequent manifestation of post-transfusion graft-vs-host disease. A skin biopsy is helpful for diagnosis

Histopathology of the skin rash in acute graft-vs-host disease shows degenerative changes and vascuolization of the keratinocytes in the basal layer of the epidermis along with a lymphocytic infiltration of the upper dermis extending into the epidermis.[80] If the patient progresses to chronic graft-vs-host disease, uncommon in the post-transfusion patient, skin atrophy will develop with hypo- or hyperpigmentatary changes.[79–81] Biopsy of the lesion shows thickening of the dermis with bundles of collagen and reduced numbers of lymphocytes compared with the acute form of the disease.[80] The mortality rate from transfusion related graft-vs-host disease has been reported to be as high as 95%, with infants having a higher mortality than older children.[79–81]

Acknowledgment. The author thanks Ms Karen Legois for her expert secretarial assistance in preparation of this manuscript.

REFERENCES

1. Miller DR: Origin and development of blood cells and coagulation factors: Maternal fetal interactions, in Miller DR (ed): *Blood Diseases of Infancy and Childhood,* ed 6, St Louis, Mo, CV Mosby Company, 1989, pp 1–25.
2. Oski FA, Naiman JL: Normal blood values in the newborn period, in Oski FA, Naiman JL (ed): *Hematologic Problemms in the Newborn,* ed 3, Philadelphia, WB Saunders Company, 1982, pp 1–36.
3. Popoff L, Popoff N: L'hematopoiese cutanee au course de la vie intrauterine. *Ann Dermatol Venerol* 1958; 85:137–176.
4. Shimada S, Katz SI: The skin as an immunologic organ. *Arch Pathol Lab Med* 1988; 112:231–234.
5. Chu T, D'Angio GJ, Favera B, et al: Histiocytosis syndromes in children. *Lancet* 1987; 1:208–209.
6. Horstmann DM, Banatvala JE, Riordan JT, et al: Maternal rubella and rubella syndrome in infants: Epidemiologic, clinical and virologic observations. *Am J Dis Child* 1965; 110:408–415.
7. Cooper LZ, Ziring PR, Ockerse AB, et al: Rubella: Clinical manifestations and mangement. *Am J Dis Child* 1969; 118:18–29.
8. Klein HZ, Marikarian M: Dermal erythropoiesis in congenital rubella. *Clin Pediatr* 1969; 8:604–607.
9. Hendricks WM, Hu CH: Blueberry muffin syndrome: Cutaneous erythropoiesis and possible intra-uterine viral infection. *Cutis* 1984; 34:549–554.
10. Groark SP, Jampel RM: Violaceous papules and macules in a newborn. Dermal erythropiesis associated with congenital cytomegalovirus infection. *Arch Dermatol* 1989; 125:114–117.
11. Fine JD, Arndt KA: The TORCH syndrome: A clinical review. *J Am Acad Dermatol* 1985; 12:697–706.
12. Brough AJ, Jones D, Page RH, et al: Dermal erythropoiesis in neonatal infants. *Pediatrics* 1967; 40:627–635.
13. Argyle JC, Zone JJ: Dermal erythropoiesis in a neonate. *Arch Dermatol* 1981; 117:491–494.
14. Hebert AA, Esterly NB, Gardner TH: Dermal erythropoiesis in Rh hemolytic disease of the newborn. *J Pediatr* 1985; 107:799–801.
15. Schwartz JL, Maniscalco WM, Lane AT, et al: Twin transfusion syndrome causing cutaneous erythopoiesis. *Pediatrics* 1984; 74:527–529.
16. Tan KL, Tan R, Tan SH, et al: The twin transfusion syndrome. *Clin Pediatr* 1979; 18:111–114.
17. Rausen AR, Seki M, Strauss L: Twin transfusion syndrome. *J Pediatr* 1965; 66:613–628.
18. Weinstein HJ: Congenital leukemia and the neonatal myeloproliferative disorders associated with Down's syndrome. *Clin Haematol* 1978; 7:147–154.
19. Pierce MI: Leukemia in the newborn infant. *J Pediatr* 1959; 54:691–706.
20. Seo IS, McGuire WA, Heerema NA, et al: Congenital monoblastic leukemia cutis: A case report with chromosomal abnormality. *Am J Pediatr Hematol Oncol* 1986; 8:158–162.
21. Zussman WA, Khan A, Shayesteh P: Congenital leukemia. *Cancer* 1967; 20:1227–1233.
22. Reinmann DL, Clemmens RK, Pillsbury WA: Congenital acute leukemia: Skin nodules, a first sign. *J Pediatr* 1955; 46:415–418.
23. Hoen RC: Congenital lymphatic leukemia. *Am J Dis Child* 1950; 80:800–802.
24. Gottesfeld E, Silverman RA, Coccia PF, et al: Transient blueberry muffin appearance of a newborn with congenital monoblastic leukemia. *J Am Acad Dermatol* 1989; 21:347–351.
25. Haidas S, Gratacos S, Ziva-Petropoulou M, et al: Transient skin nodules: First sign of congenital leukemia. *Eur Pediatr Haematol Oncol* 1985; 2: 221–223.
26. Engel RR, Hammond D, Eitzman DV, et al: Transient congenital leukemia in 7 infants with mongolism. *J Pediatr* 1964; 65:303–305.
27. Ganick DJ: Haematological changes in Down's syndrome. *Crit Rev Oncol Hematol* 1986; 6:55–69.
28. Lampkin BC, Bove KE, Peipon JJ, et al: Spontaneous remission of presumed congenital acute nonlymphoblastic leukemia in a karyotypically normal infant. *Am J Pediatr Hematol Oncol* 1985; 7:346–351.
29. Van Eys J, Flexner JM: Transient spontaneous remission in a case of untreated congenital leukemia. *Am J Dis Child* 1969; 118:507–514.
30. Shaw MT: Monocytic leukemia. *Hum Pathol* 1980; 11:215–227.
31. Darbyshire PJ, Smith JHF, Oakhill A, et al: Monocytic leukemia in infancy. *Cancer* 1985; 56:1584–1589.
32. Dunn NL, McWilliams NB, Mohanakumar T: Clinical and immunological correlates of leukemia cutis in childhood. *Cancer* 1982; 50:2049–2051.
33. Kuo T, Uhlemmann J, Reinhard EH: Cutaneous extramedullary hematopoiesis. *Arch Dermatol* 1976; 112:1302–1303.
34. Barton JC, Conrad ME, Poon MC: Pseudochloroma: Extramedullary hematopoietic nodules in chronic myelogenous leukemia. *Ann Intern Med* 1979;Z 91:735–738.
35. Tagami H, Tashima M, Uehara N: Myelofibrosis with skin lesions. *Br J Dermatol* 1980; 102:109–112.
36. Salloum E, Pico JL, Herait P, et al: Very late recurrence of childhood acute lymphoblastic leukemia treated with chemoimmunotherapy: A report of

three cases occuring 19, 11, and 9 years after discontinuation of chemotherapy. *Med Pediatr Oncol* 1989; 71:115–158.

37. Reaman GH, Poplack DG, Pizzo PA: Late soft tissue recurrence of acute lymphoblastic leukemia in a site of antecedent trauma. *J Pediatr* 1978; 92: 849–850.

38. Grumayer ER, Ladenstein RL, Slavc 1, et al: B-cell differentiation pattern of cutaneous lymphomas in infancy and childhood. *Cancer* 1988; 61:303–308.

39. Bhanot R, Brough J, Ravindranath Y, et al: Primary malignant lymphoma cutis in an infant: Case report and review of the literature. *Pediatrics* 1979; 64:478–482.

40. Link MP, Roper M, Dorfman RF, et al: Cutaneous lymphoblastic lymphoma with pre-B markers. *Blood* 1983; 61:838–841.

41. Bernard A, Murphy SB, Bowman WP, et al: Non-T non-B lymphomas are rare in childhood and associated with cutaneous tumor. *Blood* 1982; 59:549–554.

42. Cozzutto C, DeBernardi B, Cornelli A, et al: Primary cutaneous lymphoma with nodular pattern in infancy. *Cancer* 1980; 45:603–608.

43. Kukreja S, Zarkowsky H: Cutaneous lymphoma in an infant: Case Reprot. *Med Pediatr Oncol* 1979; 7:17–18.

44. Traggis D, Jaffe N, Vawter G, et al: Non-Hodgkin's lymphoma of the head and neck in childhood. *J Pediatr* 1975; 87:933–936.

45. Paller AS, Esterly NB, Baum ES, et al: Primary cutaneous T-cell lymphoma in a child. Pediatr Dermatol 1984; 2:131–135.

46. Kadin ME, Sako D, Berliner N, et al: Childhood Ki-1 lymphoma presenting with skin lesions and peripheral lymphadenopathy. *Blood* 1986; 68: 1042–1049.

47. Pochedly C: Neuroblastoma in infancy, in Pochedly C (ed): *Neuroblastoma*, ed 1. Acton, Publishing Sciences Group 1nc, 1976, pp 1–34.

48. Schneider KM, Becker JM, Krasna IH: Neonatal neuroblastoma. *Pediatrics* 1965; 36:359–366.

49. Hass D, Ablin AR, Miller C, et al: Complete pathologic maturation and regression of stage IVS neuroblastoma without treatment. *Cancer* 1988; 62:810–825.

50. Rangecroft L, Lauder 1, Wagget J: Spontaneous maturation of stage IV-S neuroblastoma. *Arch Dis Chld* 1978; 52:815–817.

51. Shown TE, Durfee MF: Blueberry muffin baby: Neonatal neuroblastoma with subcutaneous metastasis. *J Urol* 1970; 101:193–195.

52. Griffin ME, Bolande RP: Familial neuroblastoma with regression and maturation to ganglioneurofibroma. *Pediatrics* 1969; 43:377–382.

53. Schwartz AD, Dadash-Zadeh, Lee H, et al: Spontaneous regression of disseminated neuroblastoma. *J Pediatr* 1974; 85:760–763.

54. Hawthorne HC, Nelson JS, Witzleben CL, et al: Blanching subcutaneous nodules in neonatal neuroblastoma. *J Pediatr* 1970; 77:297–299.

55. Saracco S, Abramowsky C, Taylor S, et al: Spontaneously regressing adrenocortical carcinoma in a newborn. *Cancer* 1988; 62:507–511.

56. Mann JR, Cameron AH, Gornall P, et al: Transplacental carcinogenesis (adrenocortical carcinoma) associated with hydroxyprogresterone hexanoate. [letter] *Lancet* 1983; 2:580.

57. Wiss K, Solomon AR, Raimer SS, et al: Rhabdomyosarcoma presenting as a cutaneous nodule. *Arch Dermatol* 1988; 124:1687–1690.

58. Hashimoto K, Takahashi S, Lee RG, et al: Congenital self-healing reticulohistiocytosis. *J Am Acad Dermatol* 1984; 11:447–454.

59. Ornvold K, Jacobsen SV, Nielsen MH: Congenital self-healing reticulohistiocytoma. *Acta Paediatr Scand* 1985; 74:143–147.

60. Oranje AP, Vuzevski VD, DeGroot, et al: Congenital self-healing non-langerhans cell histiocytosis. *Eur J Pediatr* 1988; 148:19–31.

61. Kanitakis J, Zambruno G, Schmitt D, et al: Congenital self-healing histiocytosis (Hashimoto-Pritzker). *Cancer* 1988; 61:508–516.

62. Marsh WL, Lew SW, Heath VC, et al: Congenital self-healing histiocytosis. *Am J Pediatr Hematol Oncol* 1983; 5:227–233.

63. Dehner LP, Bamford JT, McDonald EC: Spontaneous regression of congenital cutaneous histiocytosis X: Report of a case with discussion of nosology and pathogenesis. *Pediatr Pathol* 1983; 1:99–106.

64. Hashimoto K, Griffin D, Kohsbaki M: Self healing reticulohistiocytosis. *Cancernm* 1982; 49:331–337.

65. Broadbent V, Davies EG, Heaf D: Spontaneous remission of multisystem histiocytosis X. *Lancet* 1984; 1:253–254.

66. Nezelof C: Histiocytosis X: A histological and histogenetic study. *Perspect Pediatr Pathol* 1979; 5:153–178.

67. Stingl G, Tamaki K, Katz SI: Origin and function of epidermal Langerhans cells. *Immunol Rev* 1980; 53:149–174.

68. Favara BE, McCarthy RC, Mierau GW: Histiocytosis X. *Hum Pathol* 1983; 14:663–676.

69. Delaunay M: Effects cutanes indesirable de la chimotherapie antitumorale. *Ann Dermatol Venereol* 1989; 116:347–361.

70. Burke MC, Bernhard JD, Michelson AD: Chemotherapy-induced painful acral erythema in childhood: Burgdorf's reaction. *Am J Pediastr Hematol Oncol* 1988; 11:44–45.

71. Burgdorf WC, Gilmore WA, Ganick RG: Peculiar acral erythema secondary to high-dose chemotherapy for acute myelogenous leukemia. *Ann Intern Med* 1982; 97:61–62.

72. Graves T, Hooks MA: Drug induced toxicities associated with high-dose cytosine arabinoside infusions. *Pharmacotherapy* 1989;Z 9:23–28.

73. Lokich JJ, Moore C: Chemotherapy-associated palmar-plantar erythrodysthesia syndrome. *Ann Intern Med* 1984; 101:798–800.

74. Doyle A, Berg C, Bottino G, et al: Erythema and desquamation after high-dose methotrexate. *Ann Intern Med* 1983; 98:611–612.

75. Hughes BR, Cunliffe WJ, Bailey CC: Excess benign melanocytic naevi after chemotherapy for malignancy in childhood. *Br Med J* 1989; 299:88–91.

76. de Wiut PJ, de Vann GM, de Boo TM, et al: Prevalence of naevocytic naevi after chemotherapy for childhood cancer. *Med Pediatr Oncol* 1990; 18: 336–338.

77. Shaw NJ, Eden OB: Skin rash after completion of therapy for leukemia in childhood. *Pediatr Hematol Oncol* 1989; 6:31–35.

78. Kirk JA, Rogers M, Menser MA, et al: Unusual skin rash following withdrawal of oral 6-mercaptopurine in children with leukemia. *Med Pediatr Oncol* 1987; 15:281–284.

79. Anderson KC, Weinstein JH: Current concepts: Tranfusion-associated graft-vs-host disease. *N Engl J Med* 1990; 323:315–321.

80. Hood AF, Soeter NA, Rappeport J: Graft-vs-host reaction. *Arch Dermatol* 1987; 1087–1091.

81. von Fliedor V, Highby DJ, Kim U: Graft-vs-host reaction following blood product transfusion. *Am J Med* 1982; 72:951–961.

82. Spitzer TR, Cahill R, Cottler-Fox M: Transfusion induced graft-vs-host disease in patients with malignant lymphoma. *Cancer* 1990; 66:2346–2349.

83. Funkhouser AW, Vogelsang G, Zehbauer B, et al: Graft-vs-host disease after blood transfusions in a premature infant. *Pediatrics* 1991; 87:247–249.

Waiting for the doctor

JACQUELINE GALLOWAY

When our health declines or because of having a chronic illness, we see our primary doctor regularly if the course of sickness progresses, we are often sent to other specialists for additional diagnoses and care. When being treated by doctors on a frequent basis as opposed to just periodic check-ups, it is not unusual for patients to become experts on doctors and their office patterns. Because of the high cost of medical care, patients rarely make ambivalent trips to the doctor, and I have noted from casual discussions in waiting rooms that even patients scheduled for just an annual check-up are more observant and sophisticated than in the past.

Over the past several years, as my systemic lupus erythematosus (SLE) manifested itself in ways beyond what my rheumatologist could treat, I have been directed to several other specialists. Recently, as I was waiting in a new doctor's office, I reviewed my pocket calendar and realized that I had been to ten different specialists within the year!

Since I am an author and journalist, it is natural for me to notice in detail aspects of experiences that cause a significant affect. However, I also believe that most patients with chronic or terminal illness are also greatly influenced by medical environments.

I realized almost at once that this particular waiting room was unique and that it strongly influenced me. What was it about this office that caused me to pause and analyze what was happening? That day, I gradually settled into a sense of calm and comfort that had previously eluded me in doctors' offices. Yet, I was in a foreboding place; the John Wayne Cancer Institute at Saint John's Medical Center in Santa Monica, California. It occupies an ingeniously designed new wing. I was at first apprehensive, fearing a new examination, a new physician and possibly surgery and/or a new course of treatment. I considered how alarming a disease can sound when we read about it in popular periodicals. Traditional medical stories are often based on the worst cases and complicated scenarios. Although I have learned to seek accurate data from scientific papers and journals, since advances in health care can rapidly change, even being fortified with information from trusted medical annals, does not always allay our deepest concerns.

My thoughts then changed to reviewing the procedure that had just taken place. The first stop was at an intake desk where I was directed to a small, private room to give my insurance information to the secretary, much like an admitting office in a hospital. I appreciated the seclusion and the confidentiality.

From there, I was escorted to a waiting room situated across the hall, quite apart from the sights and sounds of the intake desk or any other medical area. In fact, the large room was decorated like a library in a private home. It had thick carpeting, assuring excellent acoustics and softly lighted table lamps instead of harsh, overhead fluorescent lighting. I had the choice of sitting on a straight-back chair, at a desk, or on sofas situated in small groups throughout the spacious room. A coffee machine was kept warm, and the reading material was diverse. The time I spent in this room generated an optimal attitude within me and I began to relax.

Thus, the environment of the waiting room seemed to me to be as essential as the expertise of the physician I was about to see. The anxiety I had originally felt diminished, and what I realized in this distinctive setting was the absence of heavy, noisy doors opening and closing constantly, ringing phones, sliding glass partitions, harsh fluorescent lights (which cause a rash in many SLE patients), heavy file cabinets being opened and closed, typewriters, clicking computer keys, and most important, I was apart from the intake personnel, and I was also not being subjected to the name of each patient checking in. I realized that those seemingly ordinary sounds were obstacles that kept me in suspense and preventing me from relaxing in some doctors' offices. These typical things can be easily overlooked when one sees a doctor only a few times a year. However, when a patient is regularly in the office, perception becomes second nature, and one can almost sense the vibes the moment the door to the office is opened.

In certain adverse conditions, by the time my name was called and I did see the doctor, I was fatigued from all the activity to which I had been subjected. I discovered that I was tense and that I had lost my own sense of self and was so fatigued that I could not focus on my symptoms. At times, I was really agitated to the point when I saw the physician, I was almost "testing" the doctor because I had become exhausted and had lost patience. I even downplayed my problems to escape as soon as possible, surely defeating the purpose of my visit. I recall withdrawing at times and never could identify the reason until being in this serene library.

Perhaps, the reasoning behind the physical set-up of typical waiting rooms with glass petitions and bright lights is that they suggest a sense of professional and scientific proficiency. However, I have never looked forward to sitting in close proximity in chairs arranged almost elbow to elbow next to a stranger, sharing not only possible infections, but also the conversations of other family members or knowing each of their names and insurance companies from the audible signing-in. At times, I resented the glass barrier separating "us" from "them," as if "they" would be

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free of our germs or "they" were linebackers to the examining rooms. As I would stare at those glass windows, I found myself reflecting on the conversations of the appointment secretary. If I heard "when would you like to come in," I sensed a cooperative and not over-booked office. Whenever I heard "the only time the doctor can see you" (giving no choice), I decided it was an office that failed to recognize the value of the patient's time. It was almost as if the patient were the low priority.

My examination this particular day led to hospitalization and surgery. I was convinced that my stress was reduced to a minimum not only because the first encounter had set me up for an affirmed sense of self-confidence, but also because the ambiance of that waiting area reflected the attitude of the inner office, the nursing staff and the doctor. The climate of most waiting rooms, in fact, communicates the doctor's attitude and demeanor.

I acknowledge the need for certain stark areas such as Intensive Care Units, Emergency Rooms, Operating Rooms, and so forth. I suggest, however, that in this modern age of bringing more sensitivity to the doctor-patient relationship, the ideal first step is a pleasant, relaxing waiting room. Tranquility attained through the personal comfort felt in distinctively designed waiting rooms can be conducive to having a genuinely positive influence on the patient's state of mind. When we reach our destination, the examining room, we might arrive with less anxiety and bring an energetic enthusiasm to the examination instead of defeatism and exhaustion.

During a recent visit to another specialist's office who also has taken extraordinary measures to ensure the patient's comfort, I requested an interview to discover just why the office schedule was consistently on-time. Throughout my years of visits, I had never waited an unreasonable amount of time to see this doctor. What I discovered during my subsequent meeting was that the appointment secretary had a precise schedule and anticipated the times required for certain procedures. Then every five slots, one blank appointment was left for specific "catch-up-time" so that when visits exceeded the predicted time, the doctor would not fall far back behind schedule. That is the epitome of respect for patients and caring! It is the patient's responsibility to consider the doctor's time as precious. Emergencies and surgeries can occasionally delay the most precise calendar of appointments and most patients are very understanding. Physicians must also realize the natural anxiety that each patient brings to the office and give careful attention to the appointment schedule as well as to the environment of the office waiting room. Circumstances surrounding treatments can affect their results. These are not subtleties or luxuries but perceptions that many patients record after serious reflection.

We patients want our doctors to be as human as we are. Most of us are reasonable and seek both expertise in our physician and a fair degree of sensitivity that does not just begin with the examining process. When patients see their doctors on a regular basis, they become keenly aware of the tapestry created by the staff and the office setting. Medicines and surgeries can heal our bodies, but mental images have a powerful effect as well. An inviting office can suggest "I am at peace and I feel relaxed" regardless of the physical or emotional pain accompanying the wait.

The story of Lassa fever

Part II: Learning more about the disease

John D. Frame, MD

In late January 1970, the Sudan Interior Mission (SIM) office in New York notified me of fragmentary reports of a series of patients with high fever and a high case-fatality rate in their hospital in Jos, Nigeria; several members of the hospital staff were ill. Although the clinical information was sketchy, I wondered whether Dr Jeanette Troup might not be dealing with a second outbreak of Lassa fever (LF). It seemed improbable to me that yet another unknown agent of such virulence would have revealed itself in Nigeria within a year of the finding of Lassa virus (LV). Had animals, perhaps rodents, taken up infectious waste from the first cases and become a reservoir for the virus, to reinfect humans a year later?

Our wish for more information was largely unsatisfied because of the limitation of communications consequent on the civil strife in Nigeria. The use of short-wave radio was essentially forbidden because of military considerations; mails were slow, and even cables were often delayed. Communications within the country, poor at best, were unreliable; telephone conversations were hardly audible. Even after we in the United States had received more information, weeks were to pass before we fully realized the tragedy of this second outbreak of LF (Fig 1).

Lassa Fever in Jos Again

In the autumn of 1969, Dr Jeanette Troup was confronted in Nigeria with a flood of yellow fever cases. It was the Jos Plateau epidemic resulting in at least 252 cases with a case fatality rate in the hospitals of 40%.[1] Her resources were severely taxed with the influx of 27 yellow fever patients of whom 17 died. There was a respite in late December, and she returned to her usual tempo of life as superintendent of the Evangel Hospital in Jos.

Dr Troup, a graduate of Hahnemann Medical School, followed her brother, Dr Roger C. Troup, to Nigeria in 1954. Dr Roger had obtained his Diploma of Tropical Medicine and Hygiene from the Liverpool School of Tropical Medicine after graduating from Southwestern Medical College, and had gone to Nigeria in 1950. He had been assigned almost at once to the Bingham Memorial Nursing Home, earlier established to provide medical care for missionaries. By 1954, it was time for his furlough and once she had finished language school, Dr Jeanette Troup relieved him at the nursing home.

Subsequently, after her brother returned to Nigeria a year later, she worked in other mission hospitals. She relieved her brother again for a year in 1959 and finally took over the responsibility for the nursing home and the supervision of the health of missionaries in Nigeria when Dr Roger Troup left missionary service in 1962.

The SIM decided to build a general hospital in Jos; Evangel Hospital, as it was known, was completed in 1970, and the missionary Nursing Home responsibilities incorporated into it.

So it was at Evangel Hospital that patients with severe fever again began to seek medical care in mid-January 1970. At first Dr Troup believed she was dealing with new, atypical cases of yellow fever, but the symptoms were different, and hospital staff members, not affected in the yellow fever epidemic, were becoming ill; two had died. On January 27, she informed Dr Donald Carey of the Virus Research Laboratory (VRL) in Ibadan that the cases were reminiscent of LF, and began to submit paired sera for diagnosis by complement fixation (CF) tests. She was indeed in the midst of a major nosocomial outbreak of LF. Although the diagnosis could not yet be confirmed by laboratory means; the VRL in Ibadan had no antigen for CF tests.[2]

Although the diagnosis was not yet clear, the staff and the community of the church and mission did relate this outbreak to the experiences of the year before. The weekly prayer meeting on January 21 centered about Psalm 91:

> He that dwelleth in the secret place of the Most High shall abide under the shadow of the Almighty. I will say of the Lord, He is my refuge and my fortress: my God; in Him will I trust. . .Thou shalt not be afraid for the terror by night; nor for the arrow that flieth by day; nor for the pestilence that walketh in darkness.[3]

Asked to comment, Dr Troup, missionary as well as physician, noted how these words had always been a source of courage to her in the face of danger and trying circumstances. She also assured her colleagues that every possible care would be given for the sick while the staff attempted to understand the disease they were confronting.

Her African and expatriate colleagues were later to recall these comments, and a later statement of Dr Troup's, that the staff should do all they could professionally and then leave the rest to God's will.

In contrast to the circumstances of the first outbreak, it was not clear how the infection had entered the hospital nor how it was spread; this information would only be discovered in the epidemiological investigations conducted later. By January 25, 1970, she had treated 17 cases of whom four had died and seven were to die later. That day, to establish the cause of the outbreak in the event that other laboratory tests were not successful, Dr Troup decided to perform an autopsy. The necropsy was of an

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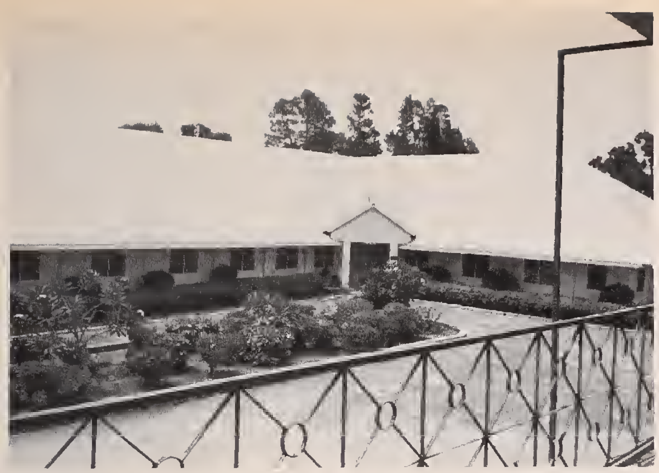

FIGURE 1. The Bingham Memorial Hospital where the first Lassa fever outbreak occurred (courtesy Lily Pinneo).

African staff member, one of the five workers who became ill and of three who died.

Dr Troup cut her finger during the course of the autopsy; she confided this information to her young colleague, Dr Harold White, and continued with her responsibilities in the hospital.

Dr Troup Becomes Ill

On February 4, the 10th day after the accident, she developed generalized muscle aching and chilling. Over the next three days, her myalgia increased; she experienced a fever of 101°F to 102°F. She vomited February 10, her fever rose to 103.8°F, and she was admitted to the hospital.

The following day her malaise increased, and she developed a large bulbar sub-conjunctival hemorrhage. She felt better the next day, but the hemorrhage increased in size, and a similar lesion developed in the other eye as well. Infection and white spots were seen in the pharynx, and the white spots grew to become patches. Cervical and submandibular lymphadenopathy, noted earlier, became more marked; respiration was somewhat labored, and infiltrations of the lungs were found on chest X-ray on February 15.

Her physicans were encouraged by a fall in her temperature; they had thought earlier of sending her to Ibadan, but with the apparent improvement in her status did not wish to subject her to the rigors of the trip there. However, the next day she coughed a small amount of rusty sputum. A cut-down was performed to ensure access for parenteral fluid administration; she began to ooze from the incision, and from sites of intramuscular injections. She was given plasma from a staff member who had recovered from a similar febrile illness with onset January 16; because of technical problems only 150 ml could be administered. On February 17, she vomited dark-brown material chemically positive for blood, and developed petechiae near her elbows. She showed signs of pulmonary edema; her urinary output was reduced, and her blood pressure was found to be 80/60. She was given another 80 ml of plasma. Her condition continued to deteriorate, and she died at 4:30 PM February 18, the 15th day of her illness, having followed the inexorable course she had described for the first two LF patients the year before. Hers was the last death in the outbreak (Fig 2).[4]

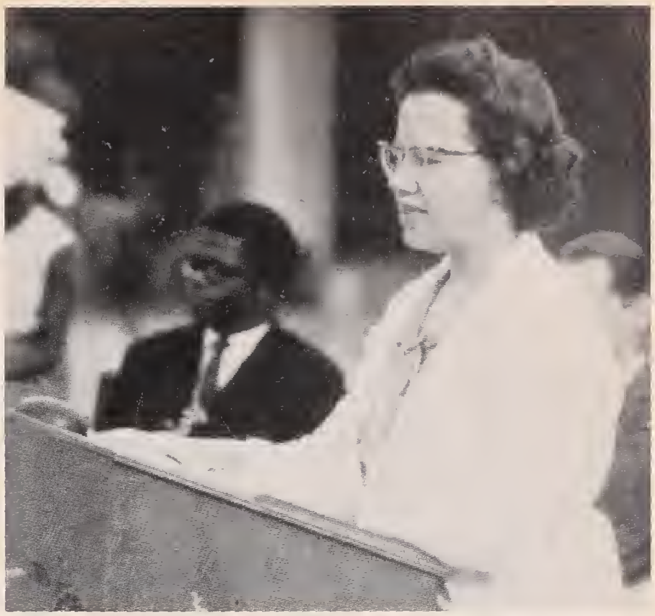

FIGURE 2. Dr Jeanette Troup, who first clinically described Lassa fever. This photograph was taken in Nigeria in January 1970 shortly before her death from Lassa fever (courtesy Lily Pinneo).

The inability of convalescent plasma to affect the course of Dr Troup's illness is now understood by information obtained in later studies of LF and the related Argentine hemorrhagic fever (AHF). From animal studies of LV infections, it has been found that whatever protection LF convalescent plasma offers is related to the level of neutralizing antibody (NA) titers, and that these antibodies reach appreciable levels only three to six months after the onset of infection.[5] Furthermore, the usual NA concentrations of convalescent plasma are such that two units (500 ml) of plasma are likely to be needed for effective treatment (PB Jarling, personal communication). In both LF and AHF plasma infusion may not be helpful in the late stage of the illness.[6,7]

In retrospect one may wonder whether Dr Troup's death could have been averted. One can always imagine that she might have survived with alternate programs of management. It must be born in mind, however, that at the time it seemed preposterous to many tropical disease experts that the outbreak was indeed LF; my suggestion then of the possibility of LF was viewed askance as being based in flimsy evidence. The facilities in Ibadan did not seem sufficiently superior over those in Jos to warrant the arduous trip there. There was not enough hard evidence at the time to justify the difficult trip to the United States at the beginning of Dr Troup's first symptoms of illness; it was not certain in her mind and in the minds of her colleagues that they were in fact dealing with a second outbreak of LF. Furthermore, there was every possibility that international transportation would have been refused her, in view of the alarm aroused by the earlier outbreak.

Dr Troup is to be honored for her devoted concern for her patients, and her clinical acumen. She demonstrated as well the scientific determination to discover, as completely as possible, the cause and pathological course of the disease she was facing. To her, we owe the first clinical descriptions of LF, descriptions based on clinical observation, examination and laboratory testing. Her descriptions, in the classical tradition of Addison and the other great clinicians, permit one to recognize severe LF; their significance is not diminished by the later discovery of milder forms of LV infection.

Miss Pinneo Returns to Nigeria

When word of the outbreak in Jos was received in New York, Miss Pinneo, the nurse who had recovered from LF in the 1969 outbreak, was determined to return to Nigeria, three months before the end of the convalescent leave given her by the SIM. She felt that, if the illnesses were indeed LF, with her immunity to LV she would be able to donate plasma for the treatment of patients. She believed the immunity would also make it possible for her to help unravel the course of the infections. Her wish to return to Nigeria became more urgent when she learned that Dr Troup was ill; she wanted to donate plasma for treatment of her friend and colleague.[8]

I recognized at the time that, although Miss Pinneo clearly could not provide plasma for all LF patients, treatment of Dr Troup might be feasible. Furthermore, in view of our ignorance of the source of contagion of the present outbreak and what risk there might be investigating it, her immune status could be an asset in the care of LF patients, as well as in investigations of the epidemiology of the outbreak. Although she still became fatigued easily and had residual nystagmus, mild ataxia, and intermittent tinnitus, it did appear that with some attention to her own health she could contribute to the needs of the Evangel Hospital. As it was, however, because of delays in the issuing of a visa to return to Nigeria, then in the midst of the Biafran war, she could not leave for Nigeria until near the end of February, after Dr Troup's death. She carried with her plasma units for treatment of patients, but the outbreak in Jos had subsided by the time of her arrival there.

Dr Jordi Casals of the Yale Arbovirus Research Unit (YARU) for lack of a Nigerian visa had also been delayed in his plan to proceed to Jos. He wished to help with elucidation of the course of the outbreak; he would also be able to donate plasma in case of emergency need. When news of Dr Troup's illness reached him from the VRL in Ibadan, he immediately shipped to the laboratory two units of his own plasma and a supply of LV antigen for CF testing. Again, communication problems limited the effectiveness of the steps he had taken. The garbled VRL message seemed to indicate that Dr Troup was being transferred to Ibadan for treatment. The plasma and antigen reached Ibadan on February 15 by a special courier of the US Agency for International Development. However, Dr Troup was still in the Evangel Hospital in Jos.

Dr Casals received his Nigerian visa soon after Miss Pinneo and proceeded to Ibadan. He, Dr Carey, Dr Graham Kemp, Dr George Stroh of the VRL proceeded to Jos, and with Dr White and Miss Pinneo began to trace the course of the outbreak by reviewing the clinical records of the suspected LF patients (Fig 3). It was finally found that all but one of the early cases identified in the outbreak had been admitted to Ward A of the hospital or had visited patients there. Infection in later cases had apparently been transmitted within families of those infected on Ward A. However, the initial presumptive case, the source of infection for the others, was not discovered easily. In reviewing, the hospital charts of patients treated at Evangel Hospital in the weeks before the first identified patients, Dr Kemp found the record of a woman whose medical history was compatible with LF. She was eventually traced to Lagos, the Nigerian capital, and subsequently found serologically positive for LV antibody.[9] In the hospital, she had occupied a bed near the window from which prevailing breezes swept across the rest of Ward A; airborne infection was possibly the means by which many fellow patients and visitors to the ward had been infected.

FIGURE 3. (L to R) Dr Jordi Casals, Dr Donald Carey, Dr Graham Kemp, and Dr Harold White, investigating the epidemiology of Lassa fever at Jos, Nigeria, March 1970 (courtesy Lily Pinneo).

In all, 28 LF cases were eventually identified at Jos, in the nearby Vom Christian Hospital and in the homes of the families of hospital cases; 13 died.

Serological Surveys

With the second outbreak of LF in Jos, it was clear that the newly described hemorrhagic fever was more than a tragic curiosity. Serological studies for NA to determine the earlier presence of LV infections in Africa were therefore conducted at the Atlanta laboratories of the US Centers for Disease Control (CDC).[10] Among specimens tested were those of missionaries.

Since 1967, I had been freezing and storing aliquots of sera obtained from missionaries returning to the United States from Africa. Of 712 missionaries in the group, 522 had lived in Central and West Africa. Five were found positive for LV NA: one from Nigeria and four from Telekoro, Guinea.

A review of the medical record of the missionary from Nigeria revealed that in December 1952, he had been severely ill. Dr Roger Troup had recorded the differential diagnosis of "atypical typhoid, atypical typhus or a viral disease not yet described." The patient had had only relatively mild fevers otherwise. In 1955, on his return from Nigeria on furlough, he was found to have a major hearing defect. In spite of this, he continued working in Nigeria until 1967, when he left mission service; the specimen tested had been obtained at that time, 15 years after his severe fever.

One of the three adult missionaries from Guinea positive for LV antibodies was a Mrs. Carrie Moore, a worker of the Christian and Missionary Alliance. She and her husband had served in Guinea for many years, and she was well-versed in the Kissi language. After her husband's death, Mrs. Moore was invited by her mission to return to Guinea to work on translating the Bible into Kissi. At the time, Guinea under the presidency of Sekou Toure had made things difficult for those from the West; an expatriate could obtain a visa to enter Guinea only if she or he were engaged in an occupation which for which no qualified Guinean could be found. The government recognized, however, that no Guinean was qualified to teach in a Christian seminary or translate the Bible. It therefore permitted foreign workers qualified for these tasks to enter the country if they had worked there previously or were replacing others. Carrie Moore fulfilled these criteria and was admitted.

Mrs. Moore was living in a small hut on the outskirts of Telekoro. Her interpreter and assistant was a young African named Jean who helped her with the nuances of his language. Each morning, Jean would prepare breakfast for the two of them, cleaning the table of mouse droppings as he did. He made a strong effort to clear the house of mice with traps and daily reported the evening's catch in what Mrs. Moore called the "mouse house" until he reached a total of 30; Mrs. Moore told him he need make no more reports!

In August 1965, Mrs. Moore became ill with a fever characterized by extreme lassitude, a prolonged course and a longer convalesence, and followed by total nerve deafness. Unable to carry out more than rudimentary translation after her recovery she stayed in Guinea until a replacement could be found for her, then returned to the United States to retire.

Both the other two adults from Guinea reported prolonged convalescence after illnesses characterized by fever, severe muscle pains, and prostration. Neither reported unusual bleeding nor a marked sore throat. None of the cases in Guinea was related temporally to another in such a way to suggest transmission of infection among them. The child of one of the patients was also seropositive; she had had a number of fevers during the four years in Guinea, none of them distinctive. No missionary colleagues had died of a febrile illness during the previous ten years.

The CDC laboratories also tested 458 sera obtained from a WHO Trypanosomiasis Survey conducted near the Benue River in Nigeria in 1965 and 1966. Ten neutralized LV on testing. There were, as well, 281 specimens from collections made by the VRL in Ibadan between 1965 and 1970. Of these, 25 neutralized LV. It is possible to estimate the approximate incidence of an illness from serological prevalence data by means of the following equation:

$$\frac{\text{number of cases}}{\text{population} \times \text{yrs of exposure}} \times 1{,}000 = \text{cases}/1{,}000/\text{yr}$$

One may use the ages of those who are tested as "the years of exposure," if one assumes that NA are present for a lifetime after infection (an assumption later demonstrated to be generally true). The ages of the donors in the VRL survey had been recorded; using the formula given above, the incidence of LV infections in the population may be calculated as shown in Table I.

TABLE I. Incidence of LV Infection

Age	Positive/Total	Number/1,000/Year
0–15	5/92	5.4
16–30	17/164	10.3
Over 30	1/25	40.0

It was now clear. LV infections had occurred in Nigeria before the discovery of the virus in 1969 and were not limited to that country, but this was present at least as far west as Guinea. The results of these findings were to bear fruit later.

It did seem that a broader picture of the nature of LF could be obtained if investigations were to be carried out in Guinea as well as in Nigeria. At the time research in Guinea was limited by the unwillingness of President Sekou Toure to open his country to Western scientists. When I studied the map of West Africa to find Telekoro, a suburb of Kissidougou in Guinea, I noted it was located in a portion of Guinea winding around Sierra Leone and Liberia. I proposed at the meeting of the American Society of Tropical Medicine and Hygiene in late 1970 that LF research in these two countries would likely be profitable. My suggestion evoked no response.

A second conclusion was that medical workers in Africa needed to be made aware of LF, and that the delays inherent in the normal means of scientific and medical communication by formal publication might risk many lives. I therefore prepared summaries of what was known of LF for the use of hospitals in Africa, and distributed them as widely as possible through mission and other agencies. It was a copy of this summary that helped identify the next known nosocomial outbreak of LF.

REFERENCES

1. Carey DE, Kemp GE, Troup JM, et al: Epidemiolical aspects of the 1969 yellow fever epidemic in Nigeria. *Bull WHO* 1972; 46:645–652.
2. Carey DE, Kemp GE, White HA, et al: Lassa fever: Epidemiolgical aspects of the 1970 epidemic, Jos, Nigeria. *Trans Roy Soc Trop Med Hyg* 1972; 66:402–408.
3. *The Bible (King James Version);* Psalm 91:vs1,5,6a.
4. White HA: Lassa fever: A study of 23 hospital cases. *Trans Roy Soc Trop Med Hyg* 1972; 66:390–401.
5. Jahrling PB, Frame JD, Rhoderick JB, et al: Endemic Lassa fever in Liberia. IV: Selection of optimally effective plasma for treatment by passive immunization. *Trans Roy Soc Trop Med Hyg* 1985; 79:380–384.
6. Frame JD, Verbrugge GP, Gill RG, et al: The use of Lassa fever convalescent plasma in Nigeria. *Trans Roy Soc Trop Med Hyg* 1984; 78:319–324.
7. Enria DA, Briggiler AM, Fernandez NJ, et al: Importance of dose of neutralizing antibodies in treatment of Argentine haemorrhagic fever with immune plasma. *Lancet* 1984; 2:255,256.
8. Altman LK: Nurse goes to Nigeria with plasma to fight Lassa fever virus. *NY Times* Feb 26, 1979.
9. Fuller JG: *Fever: The Hunt for a New Killer Virus.* The Reader's Digest Press, 1974, pp 240–249.
10. Henderson BE, Gary GW, Kissling RE, et al: Lassa fever: Virological and serological studies. *Trans Roy Soc Trop Med Hyg* 1970; 66:409–416.

Low-dose vincristine-associated bilateral vocal cord paralysis

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Vincristine causes several neurologic side effects; common manifestations are peripheral motor and sensory neuropathy, which may progress to wrist-drop, constipation, jaw pain, and rarely, cranial nerve palsies, including ophthalmoplegia.[1,2] Laryngeal nerve paralysis producing hoarseness and vocal cord paresis have been reported with higher doses,[3–8] but there are no reports of this complication at or below the currently recommended maximum dose level of 2.0 mg. We report a case of bilateral vocal cord paralysis secondary- to low-dose vincristine.

CASE REPORT

A 73-year-old woman noted right axillary swelling for one month. She did not have diabetes and had inactive rheumatoid arthritis with a history consistent with keratoconjunctivitis sicca. Physical examination showed large right axillary lymphadenopathy with enlarged bilateral supraclavicular lymph nodes and splenomegaly. An axillary node biopsy revealed malignant lymphoma of the large cleaved and non-cleaved cell type.

The patient received chemotherapy with vincristine, 1.0 mg intravenously, doxorubicin, 30 mg intravenously, cyclophosphamide, 600 mg intravenously, and prednisone, 100 mg orally each day for five days. A second treatment was given after six days with vincristine, 1.0 mg intravenously, doxorubicin, 30 mg intravenously, and cyclophosphamide, 600 mg intravenously, with good clinical response.

Six days after the second chemotherapeutic treatment, the patient experienced hoarseness, progressing to aphonia within 12 hours. She had no stridor. Direct laryngoscopy revealed bilateral vocal cord paralysis with a compromised airway. On the next day, pulmonary function tests showed fixed extrathoracic obstruction with decreased inspiratory and expiratory peak flows. A repeat laryngoscopy showed no vocal cord function. Mild stridor developed and, with imminent airway compromise, she underwent emergency tracheostomy. A neurologic evaluation confirmed isolated bilateral, recurrent laryngeal nerve paralysis. The patient did not have peripheral neuropathy or cranial nerve findings. A computed tomography (CT) examination of the neck and chest at this time was negative for lymphadenopathy or other masses. An esophagogram and thyroid scan were also negative.

Two weeks after the onset of aphonia, the patient was able to vocalize and speak by blocking the tracheostomy. Her condition continued to improve over the next week, and she regained full voice by the third week. Laryngoscopy confirmed improvement of the vocal cord functions; however, flow volume loops continued to suggest substantial upper airway obstruction. The patient was discharged with the tracheostomy tube in site. Four weeks after discharge the tracheostomy tube was removed. Six months later, the patient regained full vocal cord function. During this period she received chemotherapy with doxorubicin, 30 mg intravenously, cyclophosphamide, 600 mg intravenously, and prednisone, 100 mg orally each day for five days. Vincristine was deleted from the combination.

DISCUSSION

Non-function of the recurrent laryngeal nerve in this patient with lymphoma could be due to compression of the nerve by lymph nodes, central nervous system involvement, or the side effects of the chemotherapy, particularly vincristine. The negative CT scan of the neck and chest, the absence of other neurologic findings, and the rapid recovery on discontinuation of the medication, point to vincristine as the likely cause. In this patient, one might also consider crico-arytenoid joint involvement by rheumatoid arthritis. However, the sudden onset of vocal cord paralysis with no other active arthritis at the time of onset, and the rapid recovery on discontinuing vincristine make this possibility very unlikely. A paraneoplastic manifestation of lymphoma could be considered, but associated bilateral vocal cord paresis has not been reported. The lymphoma in this patient was in fact responding well to the chemotherapy when the paresis occurred, an observation that argues strongly against a paraneoplastic syndrome.

A review of the literature reveals 19 cases of recurrent laryngeal nerve paresis caused by vincristine[3,6–8] and two cases of vinblastine-induced paresis producing hoarseness.[5] All these patients received high-to-medium doses of vincristine (0.05 mg/kg/week to 0.36 mg/kg/week) for a longer period (4–32 weeks) before recurrent laryngeal nerve paralysis developed. Most of these patients (16 of 21) had associated peripheral neuropathy;[3,6–8] eight of the 21 patients had autonomic neuropathy as well.[3,6] Twenty patients had resolution of vocal cord paresis on discontinuing vincristine/vinblastine. One patient died of aspiration pneumonia.[3] Nine cases of paresis associated with vincristine were documented to be unilateral.[3,5,8] Bilateral vocal cord paresis was reported with vinblastine only.[5]

The patient presented here received very low doses of vincristine, 1 mg/wk for 2 weeks (0.6 mg/m^2). Six days after the second treatment, bilateral vocal cord paresis producing aphonia and imminent airway obstruction necessitating tracheostomy developed. She did not have any associated peripheral neuropathy or autonomic neuropathy. Moderate functional recovery occurred in four weeks and complete recovery in six months, while she continued to receive adriamycin and cytoxan.

We believe that the bilateral vocal cord paresis was the result of the use of vincristine and wish to draw attention to this potentially lethal complication. This patient's underlying autoimmune disorder may have enhanced her sensitivity to vincristine, but other patients may be subject to the same complication.

Although bilateral vocal cord paresis must be very rare, it is possible that unilateral vocal cord problems may be more common and are simply missed. Anecdotal comments from oncologists indicate that transient hoarseness after vincristine administration has been noted by other physicians. Careful observation of these patients, particularly with the help of head and neck physicians, may enhance knowledge of the true frequency of the effect of low-dose vincristine on recurrent laryngeal nerve function.

REFERENCES

1. Rosenthal S, Kaufman S: Vincristine neurotoxicity. *Ann Intern Med* 1974; 80:733–737.

2. Weiss HD, Walker MD, Wiernik PH: Neurotoxicity of commonly used antineoplastic agents M second of two parts. *New Engl J Med* 1974; 291:127–133.

3. Bohannon RA, Miller DG, Diamond HD:

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Vincristine in the treatment of lymphomas and leukemias, *Cancer Res* 1963; 23:613–621.

4. Bradley WG, Lassman LP, Pearce GW, et al: The neuromyopathy of vincristine in man: Clinical, electrophysiological and pathological studies. *J Neurol Sci* 1970; 10:107–131.

5. Brook J, Schreiber W: Vocal cord paralysis: A toxic reaction to vinblastine (NSC-49842) therapy. *Cancer Chemother Rep* 1971; 55:591–593.

6. Delaney P: Vincristine induced laryngeal nerve paralysis. *Neurology* 1982; 32:1285–1288.

7. Holland JF, Scharlau C, Gailani S, et al: Vincristine treatment of advanced cancer; A cooperative study of 392 cases. *Cancer Res* 1973; 33:1258–1264.

8. Whittaker JA, Griffith IP: Recurrent laryngeal nerve paralysis in patients receiving vincristine and vinblastine. *Br Med J* 1977; 1:1251–1252.

Tuberculous peritonitis as an initial manifestation of HIV infection

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***Mycobacterium tuberculosis* infection** frequently occurs in patients infected with the human immunodeficiency virus (HIV) or who manifest the acquired immunodeficiency syndrome (AIDS).[1–3] Tuberculosis usually precedes the diagnosis of other opportunistic infections, and extrapulmonary dissemination is quite common.[3] In HIV-infected patients, the lymph nodes, urogenital system, bone marrow, and liver are among the most common extrapulmonary sites, whereas peritoneal involvement is quite rare. We report an unusual case of tuberculous peritonitis as the initial manifestation of HIV infection.

CASE REPORT

A 25-year-old homosexual man was admitted to the hospital with the acute onset of severe abdominal pain, vomiting, and fever. The patient described vague abdominal pain over the preceding five months with weight loss of 30 pounds, but denied fever, chills, night sweats, cough, or sputum production. There was no known history of tuberculosis, exposure to patients with tuberculosis, or earlier pulmonary or gastrointestinal disease. The patient's purified protein derivative (PPD) status was unknown. He denied alcohol abuse, intravenous drug abuse, or a history of blood transfusion. The patient, born in El Salvador, was single and living alone in the United States for the last six years, and worked in a paint factory.

He appeared dehydrated and in pain. His blood pressure was 100/60 mm HG; oral temperature was 101.4°F; pulse rate was 96/min; and respiratory rate was 20/min. The physical examination was significant for the absence of lymphadenopathy, oral thrush, or skin lesions. No abnormalities were detected on examination of the heart and lungs.

The neurologic examination was within normal limits. Abdominal palpation revealed generalized rigidity and rebound tenderness with hypoactive bowel sounds, but no evidence of ascites. Laboratory assessment revealed hyponatremia, normal renal and hepatic function, and 7,900/μL white blood cells (82% neutrophils, 4% band forms, 3% lymphocytes, and 11% monocytes). The hematocrit was 33.8%, with 279,000/μL platelets. The amylase count was normal, urinalysis was unremarkable, and a radiograph of the chest was normal. An abdominal flat plate radiograph revealed partial small intestinal obstruction with no evidence of pneumoperitoneum.

Exploratory laparotomy disclosed a murky yellow peritoneal fluid. The liver, spleen, and peritoneal surfaces were studded with small (2.0 × 1.5 × 1.0 cm) raised whitish nodules, some of which were ruptured. Large necrotic mesenteric lymph nodes were present at the root of the small intestine. Biopsies of the lymph nodes and peritoneal nodules revealed caseating granulomas (Fig 1); special stains showed acid-fast bacilli, which on subsequent culture grew *Mycobacterium tuberculosis*.

The patient was started on isoniazide (300 mg/day), rifampin (600 mg daily), and pyrazinamide (1,500 mg daily). HIV antibodies were present on two enzyme-linked immunosorbent assays (ELISA) and confirmed by Western blot analysis. The absolute number of T-helper (CD^4) cells was diminished (267 cells/μL). A diagnosis of AIDS was established.[4]

After surgery, the patient was initially afebrile. However, ileus developed, and he required total parenteral nutrition. On the fifth postoperative day he developed a fever. Bone marrow and repeated blood cultures were sterile for bacteria (including mycobacteria). Cerebrospinal fluid examination was normal. Bronchoalveolar lavage was negative for acid-fast bacilli, viruses, and *Pneumocystis carinii.* Empirical treatment with broad spectrum intravenous antibiotics (ceftriaxone, 1 g every 12 hours, metronidazole 500 mg every 6 hours, vancomycin 1 gm every 12 hours) for two weeks did not result in any improvement.

Three weeks after surgery, blood cultures (as well as the tip of the central venous line) grew *Candida albicans,* and the patient was started on amphotericin B, 0.6 mg/kg/day, and 5-flucytosine, 1,500 mg every 6 hours. On antifungal therapy the patient's condition symptomatically and objectively improved, he became afebrile, Karnofsky scores increased, and he was able to tolerate normal feeding. Improvement continued for four weeks until the seventh postoperative week, when fever, hypotension, and respiratory distress suddenly developed. A chest film showed bilateral alveolar infiltrates, and he required intubation. Fever and the radiographic abnormalities persisted along with hypoxemia. There was no evidence of *Pneumocystis carinii* on sputum examination; repeated tracheal secretion cultures grew *Enterobacter cloacae*. On the basis of the above findings, nosocomial aspiration pneumonia was diagnosed, which did not response to two weeks of treatment with ceftriaxone, 2 g every 24 hours, and gentamycin, 80 mg every 8 hours. The patient's condition deteriorated further with the development of renal failure, liver failure, and disseminated intravascular coagulation, and he died ten weeks after admission. The absolute CD^4 count at this time was 8 cells/μL.

An autopsy was refused. The cause of death was thought to be unrelated to tuberculous peritonitis. He had resolution of the initial tuberculosis-related symptoms without isolation of mycobacteria from any other body sites, and he ultimately succumbed to complications of his prolonged hospital stay and debilitated state.

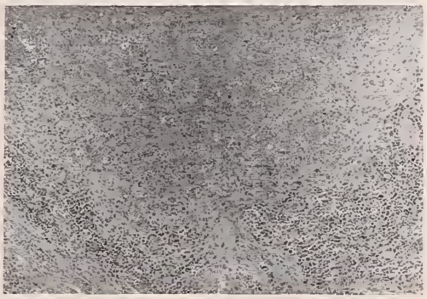

FIGURE 1. Caseating epithelioid cell granuloma in a peritoneal nodule. (hematoxylin and eosin stain; original magnification × 100).

DISCUSSION

To our knowledge, this is the first reported case of tuberculous peritonitis as the initial manifestation of HIV infection. A Medline computer search of the English language literature revealed only two other reported cases of tuberculous peritonitis in HIV-infected patients, both of whom had previously been diagnosed with AIDS. The first report described a homosexual patient with cutaneous and gastrointestinal Kaposi's sarcoma who developed abdominal pain and fever. Abdominal computed tomography demonstrated a 7-cm diameter heterog-

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enous mesenteric mass in the left lower quadrant, proven at laparotomy to be a tuberculous abscess.[5] The second report described a patient with AIDS, cerebral toxoplasmosis and end stage renal disease on continuous ambulatory peritoneal dialysis, who developed abdominal pain, vomiting, fever, and a cloudy dialysate. Investigation revealed acid-fast bacilli in the dialysate. Subsequent cultures grew *Mycobacterium tuberculosis*. His CD^4 count was 47 cells/µL.[6] Our case is consistent with studies that show that tuberculosis usually precedes and may be the initial marker of HIV infection.

There are at least three separate mechanisms by which the peritoneum may be involved with tuberculosis. The most common mechanism is hematogenous spread during primary pulmonary infection, resulting in formation of healed peritoneal tuberculosis that can later reactivate with or without evidence of pulmonary involvement.[7] CD^4 depletion, as in this case, may allow for easier reactivation of latent foci, as has been hypothesized elsewhere.[3] Alternatively, the peritoneum can be involved via direct extension from contiguous structures (ie, gastrointestinal or fallopian tubes).[7] Rarely, peritoneal involvement can develop during the bacteremic phase of military tuberculosis.[8] In this patient, a work-up failed to show evidence of pulmonary or disseminated tuberculosis, which is not at all unusual and in fact is the norm for patients with tuberculous peritonitis.[9]

Before the AIDS epidemic, the incidence of tuberculous peritonitis was estimated to be 0.1–0.7 of all tuberculosis cases.[9] Risk factors include alcoholism, ambulatory peritoneal dialysis, and immunosuppression. Typically, onset is insidious, with nonspecific constitutional symptoms predominating, such as anorexia, weight loss, and malaise. Increased abdominal girth (and ascites) is found in 53% of cases and abdominal pain in 60%.[10] The classic "doughy" abdomen described in various physical diagnosis texts is in fact rarely present. Although usually chronic, abdominal pain can be acute, mimicking acute disease in 6%.[11] Fever is present in 90% of patients, and 30% have an abdominal mass.[10]

An interesting aspect of this case is the acuteness of the presentation and the requirement for exploratory laparotomy, Barone et al[12] reported that abdominal pain occurred in 29 (12.3%) of 235 hospitalized patients with AIDS. The etiology of the abdominal pain included gastrointestinal infections (primarily *Cryptosporidium, Mycobacterium avium-intracellulare,* and *Cytomegalovirus*), malignancies (especially Kaposi's sarcoma), and miscellaneous causes (pentamidine-associated pancreatitis and ileus). Only four (14%) of these 29 patients required emergency laparotomy. Two of these were for pain associated with massive bleeding (Meckel's diverticulum and intestinal Kaposi's sarcoma), one was for a perforated duodenal ulcer, and one was for severe ileitis. Consideration of tuberculous peritonitis in an HIV-infected patient with an acute abdomen may mitigate against the need for laparotomy.

Before the advent of anti-tuberculosis chemotherapy, tuberculous peritonitis was frequently fatal, but because of early diagnosis and efficacious therapy, today it is curable.[10] Treatment recommendations for extrapulmonary tuberculosis in patients with HIV infections are not based on experience with large numbers of patients.[13] At least three drugs are advocated initially with the addition of a fourth drug in certain situations, such as central nervous system or disseminated disease, or when multi-drug resistance is suspected.[13] The optimal duration of treatment is also controversial. Therapy for at least nine months, or six months after culture conversion, has been recommended.[13] It is unknown if lifelong prophylaxis after treatment is beneficial.

Extrapulmonary (as well as pulmonary) tuberculosis is one of the earliest manifestations of HIV immunosuppression, especially in patients with earlier tuberculous exposure. Unusual presentations of common diseases, such as tuberculosis, should be carefully evaluated in patients with HIV infection.

REFERENCES

1. Chiasson RE, Schecter GF, Theuer CP, et al: Tuberculosis in patients with AIDS. *Am Rev Respir Dis* 1987; 136:570–579.
2. Centers for Disease Control: Tuberculosis and AIDS New York city. *MMWR* 1987; 36:785–795.
3. Shafer RW, Chirgwin KD, Glatt AE, et al: HIV prevalence, immunosuppression and drug resistance in patients with tuberculosis in an area endemic for AIDS. *AIDS* 1991; 5:399–405.
4. Centers for Disease Control: Revision of CDC surveillance case definition for AIDS. *MMWR* 1987; 36(suppl):3–9.
5. Barnes P, Leedom JM, Radin DR, et al: An unusual case of tuberculous peritonitis in a man with AIDS. *West J Med* 1986; 144:467–469.
6. Baumgartner DD, Arterbery VE, Hale AJ, et al: Peritoneal dialysis-associated tuberculous peritonitis in an intravenous drug user with AIDS. *Am J Kidney Dis* 1989; 14:154–157.
7. Singh MM, Bhargava AN, Jain KP: Tuberculous peritonitis: An evolution of pathogeentic mechanisms, diagnostic procedures and therapeutic measures. *N Engl J Med* 1969; 281:1091–1094.
8. Burack WR, Holister RM: Tuberculous peritonitis: A study of forty-seven proved cases encountered by a general medical unit in twenty-five years. *Am J Med* 1960; 28:510–523.
9. Dineen P, Homan WP, Grafe WR: Tuberculous peritonitis: 43 year experience in diagnosis and treatment. *Ann Surg* 1976; 184:717–722.
10. Karney WW, O'Donoghue JM, Ostrow JH, et al: The spectrum of tuberculous peritonitis. *Chest* 1977; 72:310–315.
11. Sochocky S: Tuberculous peritonitis: A review of 100 cases. *Am Rev Respir Dis* 1967; 95:398–401.
12. Barone JE, Gingold BS, Nealon TF, et jal: Abdominal pain in patients with AIDS. *Ann Surg* 1986; 204:619–623.
13. Centers for Disease Control: Tuberculosis and HIV infection: Recommendations of the advisory committee for elimination of tuberculosis. *MMWR* 1989; 38:236–250.

An unusual manifestation of testicular lymphoma

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Malignant lymphoma of the testis is a rare entity, constituting less than 2% of all extranodal lymphoma.[1,2] However, lymphoma accounts for 25%–50% of the testicular neoplasms seen among men older than 50 years of age.[3] Histologically, malignant lymphoma of the testis has been reported to be exclusively of the non-Hodgkin's large B-cell type.[4]

The main presenting symptom in testicular lymphoma is usually painless enlargement. The rate and duration of enlargement is variable.[5] Testicular lymphoma may spread to multiple regions, including the para-aortic, iliac, and inguinal lymph nodes, the contralateral testis, the mediastinum, the skin, the bone marrow, the lungs, the liver, the central nervous system (Waldeyer's ring), the oral pharynx, the cerebrospinal fluid, and the bone.[6,7]

We report a case of testicular lymphoma presenting in both testicles and the sphenoid sinus. This case represents the first known report of metastasis to this region, resulting in the unusual presenting symptoms of diplopia and nasal congestion in association with bilateral testicular masses.

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CASE REPORT

A 60-year-old man with no significant medical history presented with a three-day history of occipital headache, associated with a one-day history of blurry vision with diplopia, and a two-week history of nasal stuffiness. The patient had also noted bilateral scrotal swelling during the previous month. The physical examination was remarkable for ptosis of the right eyelid. The testicles were hard, nontender, and regular except for nodularity of the right epididymis. A neurologic examination was remarkable for dysmetria on the left side. Laboratory evaluation, including tests for testicular tumor markers, were within normal limits.

An ultrasound examination of the scrotum revealed enlarged testes with diffuse hypoechoic testicular parenchyma. There were no discrete masses. A magnetic resonance imaging (MRI) scan of the head showed a large, enhancing, soft tissue mass in the nasopharynx, sphenoid, and ethmoid sinuses with evidence of bony destruction of the mass of the sphenoid sinus, the floor of the sella turcia, and the clivus. The mass encased both carotids in their cavernous portion (Fig 1).

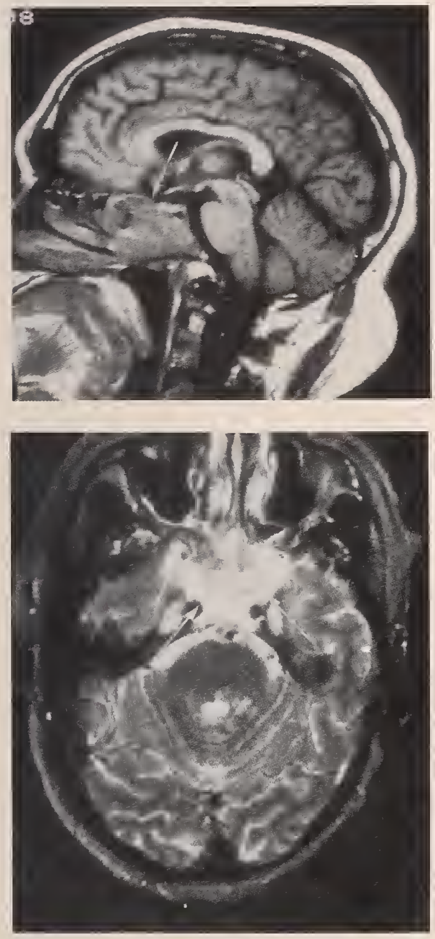
FIGURE 1. T_1 sagital (top) and T_2 (bottom) MRI images illustrating a large soft tissue mass involving the nasopharynx and extending into the sphenoid and ethnoid sinuses. There is evidence of bony destruction involving the wall of the sphenoid sinus, the floor of the sella turcia and clivus. The mass surrounds both cavernous portions of the carotid arteries that are present.

A right radical inguinal orchiectomy was performed. The pathology report was consistent with a diffuse non-Hodgkin's lymphoma of the large, cleaved cell type with abundant immunoblastic cells (Fig 2). Subsequently, a left radical orchiectomy was performed with identical pathologic findings. A computed tomography (CT) scan of the chest, abdomen, and pelvis was performed. There were extensive bilateral pleural calcifications but no evidence of adenopathy.

An abdominal CT scan obtained one week after surgery demonstrated significant retrocrural and retroperitoneal adenopathy. A nasopharyngeal skinny needle biopsy was performed to obtain a tissue diagnosis of the central nervous system (CNS) mass; this confirmed metastatic lymphoma. The CNS fluids showed no evidence of malignant cells. Cytologic evaluation of bone marrow aspirate was within normal limits.

The patient began chemotherapeutic prophylaxis to the central nervous system with cytarabine (ARA C), 50 mg intrathecally, for three courses. Radiation was administered to the region of the metastatic lymphoma (Waldeyer's ring) with a boost radiation dose of 4,400 cGY delivered over four and a half weeks. In addition, prophylaxis of the CNS was carried out with whole brain radiation (3,000 cGY over three weeks.) Systemic chemotherapy with cyclophosphamide and etoposide was also administered.

A follow-up MRI of the head revealed dramatic improvement with decrease in the size of the previous mass (Fig 3). He is currently free of any visual or nasal symptoms.

DISCUSSION

Testicular lymphoma is fortunately a rare cancer. It accounts for 1%–7% of all testicular neoplasms. Malignant lymphoma with primary clinical manifestations in the testes may occur at any age but is found most frequently in the fifth through eighth decades. Bilateral testicular lymphoma is quite common, with bilateral metachronous involvement approaching 38%, and bilateral synchronous involvement in the range of 3%–5%.[6,8]

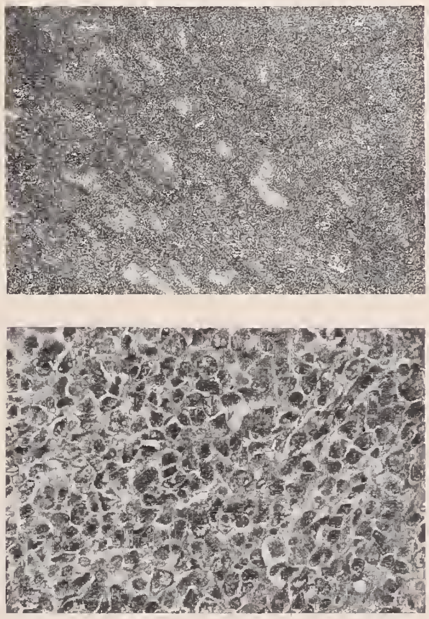
FIGURE 2. Photomicrographs showing details of lymphoid population. Most cells have large vesicular nuclei showing prominent indentation. (hematoxylin & eosin stain; original magnification × 450).

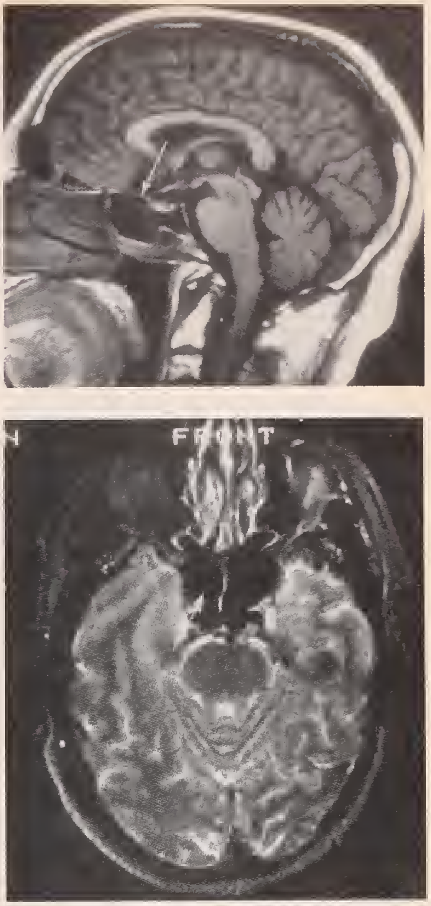
FIGURE 3. T_1 sagital (top) and T_2 coronal (bottom) MRI images after radiation treatment and intrathecal chemotherapy. A marked reduction in the size of the soft tissue mass is evident, correlating with resolution of the patient's symptoms.

This finding is in marked contrast to the frequency of bilateral germ cell testicular tumors, which is about 2.3%. Malignant lymphoma of the testes carries a grim prognosis. In spite of aggressive therapy, two-year and five-year survival rates are 10%–30% and 10%–20%, respectively.[5,8,9] In this case, involvement of Waldeyer's ring or adjacent structures does not in itself carry a poorer prognosis than similar stage II lymphomas.[6,7]

Although the usual presenting symptom of testicular lymphoma is painless testicular swelling, central nervous system (ophthalmic/olfactory) complaints are not unusual. It has been reported that the lymphoid tissue in Waldeyer's ring and adjacent structures in the oro- and nasopharynx are subsequently involved in from 22% to 45% of cases of testicular non-Hodgkin's lymphoma.[6,7] Therefore, careful evaluation of these areas should be performed as part of the routine staging procedure. CNS involvement is treated primarily with radiation therapy, 4,000–4,500 rads, and prophylactic intrathecal chemotherapy to prevent CNS recurrence.[6]

References

1. Freeman C, Berg JW, Cutler SJ: Occurrence and prognosis of extranodal lymphomas. *Cancer* 1972; 29:252–258.
2. Aozasa K, Tsujimoto M, Sakurai M, et al: Hodgkin's lymphoma in Osaka, Japan. *Eur J Cancer Clin Oncol* 1985; 21:487–499.
3. Eckert H, Smith JP: Malignant lymphoma of the testis. *Br Med J* 1963; 2:891–894.
4. Nonomura N, Aozasa K, Ueda T, et al: Malignant lymphoma of the testis: Histological and immunohistological study of 28 cases. *J Urol* 1989; 141:1368–1371.
5. Talerman A: Primary malignant lymphoma of the testis. *J Urol* 1977; 118:783–786.
6. Buskirk SJ, Evans RG, Banks PM, et al: Primary lymphoma of the testis. *Int J Radiat Oncol Biol Phys* 1982; 5:1699–1703.
7. Duncan RR, Checa F, Gowing NFC, et al: Extranodal non-Hodgkin's lymphoma presenting in the testicle. *Cancer* 1980; 45:1578–1583.
8. Sussman EB, Hajdu SI, Lieberman PH, et al: Malignant lymphoma of the testses: A clinicopathologic study of 37 cases. *J Urol* 1977; 118:1004–1007.
9. Mostofi FK: Testicular tumors. Epidemiologic etiologic and pathologic features. *Cancer* 1973; 32:1186–1201.

Intralymphatic immunotherapy of glioblastoma

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Patients with solid tumors have been reported to undergo remission with immunotherapy, either after treatment with autologous tumor cell vaccine or after administration of lymphokines.[1] Another example of immune rejection of tumors occurs in renal transplant recipients bearing non-major histocompatibility complex-matched tumors. Cessation of immunosuppression leads to immune regression of the tumor in the majority of these patients.[2] In the case of glioblastoma, an extract of *Serratia marcescens* was found to induce remission in 3 of 19 patients with recurrent tumors.[3] This report describes a patient with recurrent glioblastoma who underwent more or less complete remission of a brain tumor after immunotherapy; however, spinal cord metastasis developed.

Case Report

A 46-year-old man was admitted to University Hospital of Brooklyn for evaluation of recurrent glioblastoma. He had undergone undergone subtotal removal of a right temporal lobe tumor four and a half months previously. Because of early recurrence at the end of radiotherapy (5,000 cGy), he underwent a second subtotal removal 60 days after the first operation. Pathologic findings confirmed the presence of recurrent glioblastoma. He was given an additional 1,000 cGy, and four weeks after completion of the second course of radiotherapy or 139 days after the first operation, he was started on immunotherapy, at which time he had a second relapse confirmed by clinical and computed tomography (CT) criteria.

Informed consent was obtained, and all procedures were approved by the hospital's Institutional Review Board. The patient received three treatments (days 0, 14, and 42; average dose of 6.3×10^6 cells) of vaccine prepared from autologous tumor cells grown in tissue culture, inactivated with mitomycin, and linked sequentially to a glycylglycylcystamide spacer and the adjuvant muramyl dipeptide, as according to the methods of Eggers et al.[4] The vaccine was administered intralymphatically via dorsal pedal lymphatics, as described previously by Wiseman et al.[5] The patient's only other medication was phenytoin. He was not on steroids.

Within two weeks of starting treatment, the patient became more alert, and the bulging free bone plate covering his craniotomy site began to recede into place. A CT scan taken at nine weeks showed significant decrease in the size of the tumor; the ring-enhancing lesion having collapsed into a small nubbin of residual contrast-enhancement at the site of surgical debulking on the medial aspect of the middle fossa (Fig 1). A repeat scan at 14 weeks showed no change in this pattern, with the patient remaining alert and interactive. No side effects of treatment were seen.

During the course of immunotherapy, progressive spinal cord compression developed. A magnetic resonance imaging (MRI) scan of the spinal cord showed a very faintly gadolinium-enhancing tumor extending from C6 to T2 (Fig 1). Surgical exploration disclosed an intramedullary tumor with a large exophytic component, which was debulked; pathologic assessment revealed it to be glioblastoma. This was interpreted as a metastasis, although a second primary tumor could not be ruled out. The patient succumbed to pulmonary complications of the progressively growing spinal cord tumor shortly after the 14-week scan.

Discussion

Immunotherapy of glioblastoma has been attempted previously in numerous cases with limited success, as reviewed by Couldwell et al.[6] Bloom et al[7] described a randomized controlled study with irradiated autologous tumor cells injected subcutaneously, which led to negative results. There are apparently no reports of glioblastoma regression induced by active specific immunization.

The approach to immunotherapy illustrated in this case report is based on work in a murine fibrosarcoma model, in which animals were immunized against syngeneic tumor cells linked covalently through a spacer molecule to the adjuvant muramyl dipeptide, a simplified form of the monomer of the mycobacterial cell wall.[8] Cytotoxic lymphocytes were detected with a short-term, chromium release assay, and the growth of small tumors could be slowed or reversed with immunotherapy. Preliminary data from human glioblastoma patients innoculated subcutaneously with a similar vaccine have demonstrated the ability to immunize patients against tumor-associated antigens, as detected with a short-term, chromium-release assay performed with peripheral blood lymphocytes and autologous tumor cell targets, but no definite therapeutic effect.[4] Cytotoxic lymphocytes appear to have T-cell markers and require major histocompatibility complex (MHC) matching at the sensitization but not effector stage of immunity. The presence of T-cell markers does not prove that cytolysis occurs via the T-cell receptor. This kind of adjuvant-induced immunity resembles the MHC-unrestricted "promiscuous" killing by T cells described by other workers.[9] No toxicities have been associated with treatment except occasional urticaria around the injection site, which has been treated successfully with diphenhydramine. The putative advantage of intralymphatic versus subcutaneous administration of vaccine is probably a dose effect because injection into dorsal pedal lymphatics directly accesses the largest lymph node chain in the body. Intralymphatic injection of tumor vaccine has been reported to induce occasional remission of solid tumors outside of the central nervous system.[5]

An important issue in this case is whether the observed partial regression (ie, the regression of the brain tumor only) can be explained as a delayed effect of radiotherapy of the brain. Graeb et al[10] reported that three out of 47 glioma patients undergoing radiotherapy experienced transient CT worsening on CT scan followed by gradual improvement occurring over a period of

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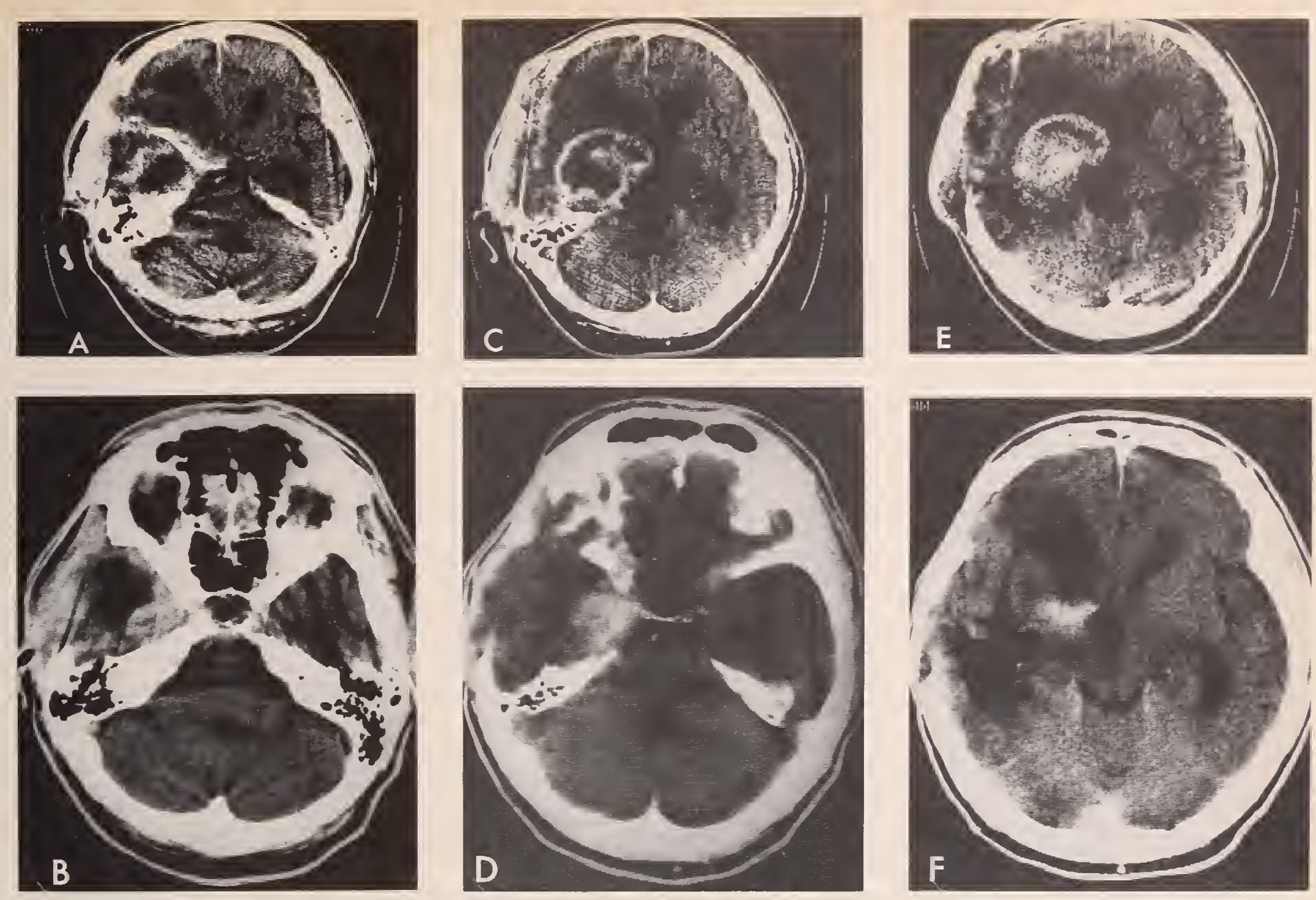

FIGURE 1. CT scans of brain tumor. Sections of scan taken four weeks after completion of second course of radiotherapy (1,000 cGy), at the time of starting immunotherapy (A,C,E). Corresponding sections of scan taken nine weeks later (B,D,F).

seven to 18 months. This kind of delayed radiation effect cannot be ruled out in this case, but it seems unlikely because of surgical documentation of early tumor recurrence, which implied radioresistance, and because of the rapidity of clinical improvement after the initiation of immunotherapy. On the other hand, it cannot be stated on the basis of one case that this is an efficacious treatment for glioblastoma, and further studies are warranted before any claims can be made in this regard.

An hypothetical alternative explanation for the mixed clinical response in this case may relate to the concept of "immunologic privilege." The central nervous system is said to be an "immunologically privileged site," a place where graft or tumor rejection is impeded, where lymphatic drainage is poor, and where lymphocytes do not normally circulate.[11,12] Lymphocyte access is thought to be regulated by the blood-brain barrier, which is maintained by tight junctions between endothelial cells, as well as by special physiologic properties of the endothelial cells and surrounding pericytes and astrocytic end-feet.[12,13] In this patient, the primary tumor, which showed marked contrast enhancement on neuroimaging, signifying breakdown of the blood-brain barrier, regressed simultaneously with growth of a spinal cord metastasis, which retained an intact blood-brain barrier. One can hypothesize that breakdown of the blood-brain barrier on neuroimaging correlates with improved lymphocyte access, although the latter may require more than just opening of tight junctions. Why endothelial cells in different parts of the central nervous system should have reacted differently to the same tumor is unknown. The alternative explanation that the spinal tumor was a second primary with different tumor-specific antigens that were not expressed on vaccine cells prepared from the first tumor is unlikely because immunity in this system has been shown to have little specificity, at least as measured by in vitro assays.[4]

Another noteworthy feature of this case was the spinal cord lesion. Clinically apparent spinal cord metastasis is a rare complication of glioblastoma[14]—tumor spread via cerebrospinal fluid being more characteristic of medulloblastoma and pinealoma.[15] Clinical spinal cord involvement has been reported in four out of 85 patients with intracranial glioblastoma, all of these cases having involved a primary tumor in the posterior fossa.[16] However, it can be argued that this patient outlived the natural history of his disease and that if immunotherapy had not been effective against the primary tumor, the spinal cord lesion would not have had time to develop.

REFERENCES

1. Marx JL: Cancer vaccines show promise at last. *Science* 1989; 245:813–815.

2. Harvey L, Fox M: Transferral of malignancy as a complication of organ transplantation: an insuperable problem? *J Clin Pathol* 1981; 34:116–122.

3. Jaeckle KA, Mittelman A, Hill FH: Phase II trial of *Serratia Marcescens* extract in recurrent malignant astrocytoma. *J Clin Oncol* 1990; 8:1408–1418.

4. Eggers AE, Tarmin L, Gamboa ET: In vivo immunization against autologous glioblastoma-associated antigens. *Cancer Immunol Immunother* 1985; 9:43–45.

5. Wiseman CL, Rao VS, R, Kennedy PS, et al: Clinical responses with active specific intralymphatic immunotherapy for cancer a phase I-II trial. *West J Med* 1989; 151:283–288.

6. Couldwell WT, Mitchell MS, Mazumder A, et al: Immunology and immunotherapy of intrinsic glial tumors, in Apuzzo MJ (ed): *Malignant Cerebral Glioma*. Park Ridge, American Association of Neurological Surgery, 1990, pp 41–57.

7. Bloom HG, Peckham MJ, Richardson AE, et al: Glioblastoma multiforme: A controlled trial to assess the value of specific active immunotherapy in patients treated by radical surgery and radiotherapy. *Br J Cancer* 1973; 27:253–267.

8. Eggers AE, Tarmin L, Ginsburg S: Use of covalently-bound adjuvant peptide to increase tumor immunogenicity. *Cancer Immunol Immunother* 1982; 12:167–172.

9. Thiele DL, Lipsky PE: The role of cell surface recognition structures in the initiation of MHC-unrestricted "promiscuous" killing by T cells. *Immunol Today* 1989; 10:375–381.

10. Graeb DA, Steinbol P, Robertson WD: Transient early computed tomographic changes mimicking tumor progression after brain tumor irradiation. *Radiology* 1982; 144:813–817.

11. Medawar PB: Immunity to homologous grafted skin, III. The fate of skin homografts transplanted to brain, to subcutaneous tissue, and to the anterior chamber of the eye. *Br J Exp Pathol* 1948; 29:58–69.

12. Wekerle H, Linington C, Lassman H, et al: Cellular immune reactivity with the CNS. *Trends Neurosci* 1986; 27:271–277.

13. Crone J: The normal barrier, in Suckling AJ, Rumsby MG, Bradbury MWB (eds): *The Blood-Brain Barrier in Health and Disease.* Chichester, Ellis Horwood Co, 1986, p 20.

14. Cobb CA, Youmans JR: Glial and neural tumors of the brain in adults, in Youmans JR (ed): *Neurological Surgery,* Philadelphia, WB Saunders Co, 1982, ed 3, p 2805.

15. Zulch KJ: *Brain Tumors, Their Biology and Pathology,* ed 3. Berlin, Springer-Verlag Co, 1986, p 184.

16. Salazar OM, Rubin P, McDonald JV, et al: Patterns of failure in intracranial astrocytomas after irradiation: Analysis of dose and field factors. *Am J Roentgenol* 1976; 126:279–292.

The Laurance D. Redway Award

The Laurance D. Redway Award for Excellence in Medical Writing was awarded to two recipients on March 19, 1992 at the annual House of Delegates meeting of the Society. The award was presented to Adrian W. Zorgniotti, MD, and Peter B. Farnsworth, MD.

Dr Zorgniotti was honored for his article, "Caruso and His Doctors," which was published in the August 1991 issue of the *Journal*. A graduate of Princeton University and the State University of New York Health Science Center at Brooklyn, he is Professor of Clinical Urology at New York University School of Medicine. Since 1979, he has served as Medical Director of the Metropolitan Opera Association.

Dr Farnsworth was honored for his article, "Triplicate Prescription in New York: History and Review," which appeared in a special supplement to the *Journal* published in November 1991. He is a graduate of Williams College and received his medical degree from McGill University School of Medicine. Dr Farnsworth is a Diplomate of the American Board of Pediatrics and Professor of Clinical Pediatrics at New York Medical College. He is currently Director of the Society's Division of Public Health and Education.

The Laurance D. Redway Award is named in honor of Laurance D. Redway, MD, who was the editor of the *Journal* in 1952–1960.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Nocardia brasiliensis soft tissue infection in an immunocompromised host

TO THE EDITOR: Nocardia species are aerobic gram-positive, soil-borne, filamentous higher bacteria. Members potentially pathogenic in humans are *Nocardia asteroides*, *N brasiliensis*, *N caviae*, and *N madurae* . Acute, subacute, or chronic, and localized or disseminated infection with frequent remissions and exacerbations may occur.

N asteroides typically produces pleuropulmonary disease with hematogenous spread to distant sites. In the United States, *N brasiliensis* is the second most common cause of nocardosis in humans. It typically causes localized cutaneous and soft tissue infection, mimicking pyogenic infections. Such infections are often managed by incision and drainage followed by empirical broad-spectrum antibiotics. Without diagnostic microbiologic studies, the diagnosis of *N brasiliensis* infection may be missed.

Case Report. A 56-year-old, white male presented to the emergency room with painful swelling and impaired mobility of his ring finger for a five-day duration. He had experienced fever and chills at home. He denied direct trauma to his finger, but he did have a cat at home which licked his fingers. The patient had been treated for large-cell lymphoma, diagnosed three years before admission, with 14-day courses of 75 mg of cyclophosphamide on alternate fortnights. He had completed a course of cyclophosphamide 10 days before his symptoms developed. The patient was married, a printer by profession, a non-smoker, and a tee-totaler. He had no significant family history.

On admission, he appeared to be a well-preserved, middle-aged, comfortable, alert, and well-oriented white man. His temperature was 36°C. His left finger had a 2-cm × 2-cm abscess with surrounding erythema on the dorsum of his middle phalanx without regional adenopathy. The other extremities were normal. His lungs were clear, his heart sounds were normal without a murmur, and his abdomen was benign. He lacked meningeal signs, and his neurological examination was normal. His hematocrit was 43.7%; peripheral white blood cell count was 9,600/mm^3 with 67% polymorphs, and 28% lymphocytes; his platelet count was 282,000/mm^3. His coagulation profile, chemistries, chest X-ray, electrocardiogram, and urinalysis were normal. Initially, samples of the pus were not studied. The patient was admitted to the hospital and was empirically treated with 2-gm nafcillin intravenously every four hours. He subsequently developed worsening cellulitis of his ring finger distal to his proximal interphalangeal joint. Ascending lymphangitis and fluctuant pyophlebitis up to his left antecubital fossa evolved within 36 hours of admission. He remained afebrile and hemodynamically stable. Because of the extent of infection, 2-gm ceftriaxone intraveneously daily was added to the regimen, and he underwent an incision and drainage of the pyophlebitis of his left forearm. Microbiologic studies were requested on the material drained at surgery. The patient remained afebrile with resolution of his cellulitis after surgical drainage. He was discharged on oral dicloxacillin after the sixth day of hospitalization. He was seen regularly by his private physician and had no recurrence in 18-months of follow-up.

Microscopic examination of a gram-stained sample of the pus showed a sea of white cells without any organisms. After several days of incubation, colonies were detected on sheep blood agar as white mold with a strong earthy odor, suggesting Streptomyces. The organism was transferred to Sabouraud dextrose agar and mycobiotic agar, and speciation was attempted. The strain was acid fast by modified acid fast stain. It hydrolyzed casein and tyrosine; and it did not decompose xanthine and was urease positive. It was also resistant to lysozyme. It was subsequently identified as *N brasiliensis.* The identification was confirmed by Dr I. Weitzman at the New York City Mycology Unit and by the New York State Mycology Laboratory in Albany, New York.

Discussion. Nocardiosis is an acute, subacute, or chronic infection often arising in the lung, caused by members of the family *Nocardiaceae*. *N brasiliensis* is the second most common cause of human nocardiosis in the United States. It causes cutaneous soft-tissue infection often mimicking pyogenic infections. Infection usually arises after traumatic implantation resulting in localized self-limited infection that rarely disseminates. Infection typically occurs in debilitated patients with compromised cellular and humoral immunity. This is particularly so in patients with lymphoreticular neoplasia; chronic obstructive, restrictive and granulomatous lung diseases; prolonged corticosteroid and cytotoxic therapy; and chronic bowel disease, sarcoidosis, vasculitis, Paget's disease, and immunosuppression after organ transplantation.

Typically, *N brasiliensis* infection presents as solitary or multiple subcutaneous abscesses. It may be spread via the hematogenous route or the lymphatic route mimicking pyogenic bacteria or sporotrichosis.[1,2]

Before the the advent of effective antibiotics, nocardiosis was managed with local drainage and supportive care. Currently, the antimicrobial therapy of choice is combination trimethoprim and sulfamethoxazole. A minimum of six weeks therapy is generally recommended to treat systemic disease.

N brasiliensis is a consistent β-lactamase producer. The genetics of this β-lactamase have not been elucidated. It is sensitive to amoxicillin-clavulanic acid, erythromycin, gentamicin, and possibly to third generation cephalosporins. *N brasiliensis* strains are generally resistant to imipenem and many other β-lactam antibiotics.[3] Antituberculous and antifungal agents are not effective in nocardiosis. Concomitant use of immunosuppressive agents may impair the therapeutic response. When required, surgical drainage or excision of the localized focus is important in the management of diseases caused by the *Nocardia* species.

Our patient presented with a purulent lymphocutaneous limb infection. Diagnostic studies were not requested at the time of his first incision and drainage. His initial infection progressed after treatment with nafcillin. He subsequently improved with incision and drainage. Delayed diagnosis was made on cultures of the surgical specimen. He was treated with ceftriaxone, a β-lactamase-stable agent, which might have had some efficacy against *N brasiliensis.* In spite of the lack of specific and prolonged therapy, neither recurrent nor systemic nocardiosis occurred.

This case illustrates the need for a high index of suspicion for nocardial infection in immunocompromised patients with pyogenic skin and soft-tissue infection. It also highlights the need for further studies into alternative antibiotic regimens for nocardiosis.

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1. Smego RA, Gallis HA: The clinical spectrum of *Nocardia brasiliensis* infection in the United States. *Rev Inf Dis* 1984; 6:164–80.

2. Padron S, Domingues M, Drosd R, et al: Lymphocutaneous *Nocardia brasiliensis* infection mimicking sporotrichosis. *South Med J* 1973; 66:609–612.

3. Wallace RJ, Nash RD, Johnson WK, et al: B-Lactam resistance in *Nocardia brasiliensis* mediated by B-lactamase and reversed in the presence of clavulanic acid. *J Inf Dis* 1987; 156:956–966.

Low technology and the prevention of nosocomial AIDS

TO THE EDITOR: A recent conference was devoted largely to a discussion of research evaluating devices to minimize the risk of needlestick and other accidents that might expose workers to nosocomial human immunodeficiency virus (HIV). It seems clear that a variety of manufacturers have become interested in this area because of the large potential market. Technological solutions to eliminate sharp devices may be most readily achievable in settings where they are being used for convenience as connectors, such as in piggyback connections for intravenous solutions. More complex strategies minimizing the risk of exposure to needles are also being investigated for use by phlebotomists or those giving injections. It seems unlikely, however, that use of sharps can be eliminated in many settings, most notably in the operating room.[1]

It is my impression that the research and expenditures on the prevention of nosocomial transmission of HIV have focused only on some links in the chain of transmission. Certainly there has been consideration by the Centers for Disease Control (CDC) and others of precautions to decrease exposure to potentially infectious material,[2,3] efforts which may now be complemented by technological devices to decrease the risk of penetrating injuries. At the other end in the chain of transmission, there has been some attention to the management of health care workers who sustain a needlestick or similar injury, with ongoing discussion of the still uncertain role of AZT postexposure prophylaxis.[4] It seems, however, that relatively little attention has been paid to the issue of decreasing the inoculum size and virulence of any material that may be transmitted at the time that a needlestick injury occurs. It is my impression that this stage in the process of nosocomial disease transmission might well be susceptible to relatively simple and economical infection control measures.

One particular strategy worthy of exploration is trying to increase the chance that virucidal material may be present at the site of any needlestick or similar injury at the actual time it occurs. When needles are used to start an intravenous line for example, would it be feasible to have the bore of the needle pre-filled with betadine or similar substance? This would decrease the dead space in the needle for blood to enter should a needlestick accident occur during the procedure. It also would provide a small amount of local antiseptic, that would nevertheless be large in comparison to the microliter quantities of blood that might be carried within the needle tip, in the event of a misadventure during cannulation of the patient's vein.[5]

Some similar considerations might apply in the operative arena. If the surgeon is going to double glove for a procedure, perhaps the first set of gloves once in place could be washed in antiseptic solution before the second set is put on.[6] In this way, a suture needle that might inadvertently transverse both sets of gloves would also come in contact with some residual antiseptic solution. The actual amount of disinfectant might be small but the same would likely be true of the amount of infectious material being carried. Even a transient interaction of disinfectant and HIV might allow significant virucidal activity to occur.[7]

An incidental advantage of this approach might be that if a colored solution were used as part of the antiseptic it would be easier to monitor glove integrity during the procedure. If the indicator material were observed to leak onto the operative field, this would be an indication that one set of gloves might be defective and that all of the operative team should re-glove.

It may be difficult to evaluate the effects of these measures in reducing the already relatively inefficient nosocomial transmission of HIV. It might be that these sorts of measures would have a more readily apparent effect on transmission of viral hepatitis. This would not only be a useful marker or likely efficacy against HIV transmission, but also a worthy goal in itself.

A disinfectant agent can be used, not simply as a cleansing agent to remove earlier contamination, but also to prevent future contamination from transmitting disease, and this is an experiment that may in fact already be in progress. Intravenous drug addicts who re-use or share their equipment are often advised to clean their utensils with bleach.[8,9,10] The main goal of this effort is to prevent the addict from being infected from the blood of an earlier user. Given the variability with which such advice is implemented by the drug abuser, it seems likely that in many cases residual antiseptic will be present at the time of the subsequent injection.

One can only speculate whether this carryover has a subsequent effect in reducing the infectious risk to the next user of the syringe whether or not it is again washed.

In conclusion, there is a whole chain of events involved in the process by which a health care worker may acquire HIV from a patient. There are substantial economic incentives for developing new products to prevent nosocomial penetrating injuries. Although these efforts are worthwhile, it seems equally important to try to devise simple strategies to decrease the infectious risk of those penetrating injuries, which may still occur.

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1. Panlilio AL, Foy DR, Edwards JR, et al: Blood contacts during surgical procedures. *JAMA* 1991; 265:1533–1537.

2. Doebbeling BN, Wenzel RP: The direct costs of universal precautions in a teaching hospital. *JAMA* 1990; 264:2083–2087.

3. Zuck TF: Low technology in a high technology era. *Transfusion* 1991; 31:390–391.

4. Jeffries DJ: Zidovudine after occupational exposure to HIV. *British Med J* 1991; 302:1349–1351.

5. Napoli VM, McGowan JE: How much blood is in a needlestick? *J Inf Dis* 1987; 155:828.

6. Walker LG: Protective garb. *N Engl J Med* 1988; 318:1333–1334.

7. Hanson PJ, Gor D, Jeffries DJ, et al: Elimination of high titre HIV from fibreoptic endoscopes. *Gut* 1990; 31:657–659.

8. Chaisson RE, Osmond D, Moss AR, et al: HIV, bleach, and needle sharing. *Lancet* 1987; i:1430.

9. Division of Health Promotion, New York City Department of Health. Shooting up and AIDS. Promotional pamphlet, 1987.

10. Navarro M: Yale study reports clean needle project reduces AIDS cases. *NY Times* August 1, 1991; p A1.

Illegal drug use

TO THE EDITOR: I must object strenuously to the form and content of the article "Illicit drugs should not be legalized" that appeared in the February issue of the *Journal*.[1] To begin with, it is full of moralizing, which does not belong in a medical journal, to wit, ". . .Society does not like addicts. . ." and ". . .even most advocates of legalization agree that drug use is grossly immoral. . ." Where are the data for such statements? Who asked whom? In any case, such statements are judgmental.

The two expert witnesses who spoke against legalization are part of the present set up and they have pre-set partisan views on the subject. In a truly unbiased panel, neither speaker should have been asked to appear, and should they have asked to be heard, they should not have been given the opportunity to speak, again, because of their present partisan position.

Most of the statements about drug use before and after legalization are meaningless because as long as drug use is illegal there are no reliable means of obtaining hard data. The statement ". . .in countries where such drugs have been legalized their use has increased. . ." is meaningless; what most likely has happened is that it is no longer dangerous to declare oneself a drug user. Thus, the users have come out of the closet, and the present figure is the correct one for drug use. In any case, the Dutch experience would indicate that ". . .the policy of normalization is rather successful and does not produce an increase in drug use. . ."

As for providing help ". . .to those who need and want it. . ." these are of course laudable goals. However, present experience indicates a tendency toward compulsion in the sense that rehabilitation is offered as an alternative to prison terms. Experiences with similar programs in the jails notoriously do not accomplish the objective.

The problems created by drug use can be divided into two categories. Those that affect individual and constitutional freedoms should not be dismissed through moralistic compulsion by a vocal minority. Then there are those that affect society as a whole. These include diversion of police efforts and police corruption, occurring because of the enormous amounts of easy money available. In my view, these issues should not be discussed in a medical journal.

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1. Rosner F, Wolpaw J, Sechzer PH, et al: Illicit drug use should not be legalized. *NY State J Med* 1992; 92:59–62.

TO THE EDITOR: The committee report prepared by Rosner et al entitled "Illicit drug use should not be legalized,"[1] makes a point that I believe has been overlooked by both sides of the drug legalization/decriminalization issue.

Rosner et al include a statement by Wilson who argues that, if heroin had been legalized in 1972, "We almost certainly would have had today a permanent population of several million, not several thousand, heroin addicts. If we yield now, we will have a far more serious problem with cocaine."[2] One popular American folk saying states that "locks are for honest people." The moral of this saying is that anybody with a little bit of creativity or brute force can get past a lock. However, the very existence of the lock provides just enough disincentive for people, who normally would not steal something, to not steal. That is—locks help keep honest people honest. This concept can be applied to illicit drug legislation. Laws banning illicit drugs are enough of a disincentive to prevent large numbers of Americans from trying drugs, and perhaps, from becoming addicts.

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1. Rosner F, Wolpaw J, Sechzer PH, et al: Illicit drug use should not be legalized. *NY State J Med* 1992; 92:59–62.
2. Wilson JQ: Against the legalization of drugs. *Commentary*, Feb 1990, pp 21–28.

TO THE EDITOR: I read with interest the report of the Committee on Bioethical Issues entitled "Illicit drug use should not be legalized."[1] I found it thoughtful and even-handed but flawed:

The committee did not differentiate illicit drug commerce from illicit drug use. The medical community has at best an advisory capacity relative to the former while there is an ethical role for itself for the latter.

If society asks, "Is there a medical benefit to reducing the use of drugs such as cocaine or heroin?" the medical community should clearly answer in the affirmative. How society implements this—legalization, regulation, interdiction, or some combination—is primarily a political question. It is not one of medical ethics and is not an area where physicians' expertise is needed.

The ethical question for physicians relates to the patient's question, "Doctor should I use crack or heroin?" The answer is a clear No! for reasons admirably summarized by the committee. This is no different than the physician's role in advising against the use of tobacco or alcohol. The next question is harder: "OK, doctor, how do I stop?" Resources for rehabilitation are grossly inadequate. If the committee wishes to delve into an ethical question with direct political implications this would be the one. The medical community should be committed ethically to increasing the availability of drug rehabilitation services.

Today, in the most urban areas illicit drug commerce is functionally, if not legislatively, legal: Drugs are sold for legal tender in the open. Sales are freely made. Drug commerce enjoys the support of such a large part of the populace that enforcement is impractical. It is a general tenet of the law that a law that is unenforceable is no law. It would be difficult to see the committee taking the same stand on tobacco sales which in toto are more dangerous to the individual and costly to society. Drug commerce differs from tobacco commerce principally by the absence of regulation and taxation.

The committee seems to accept the thesis that legalization would increase the use of cocaine/heroin as happened with alcohol after the repeal of prohibition. The committee ignores the fact that both total and per capita ingestion of alcohol in the United States has been declining. Although the reasons are diverse, education of the public to the dangers of alcohol must be afforded some weight. The decline has occurred in spite of generations of use and the incorporation of alcohol use into the fabric of daily life. Much of the same can be said of tobacco. It is well to note that scientific substantiation of the deleterious effects of tobacco and alcohol has been relatively recent compared to the many generations of their use. However, the deleterious effects of drugs are well accepted. Thus, the evidence supports the idea that the public will respond positively to enlightened, well-substantiated, rational appeals to appropriate behavior.

For these reasons, I believe educational campaigns, financed by taxing drug sales at the source, would minimize the spread of addiction. Data quoted editorially in the *New York Times* show that 85% of homeless drug users would enter rehabilitation programs if available. Furthermore, illicit drugs should be made available at low cost while interdiction is maintained. This would shift the risk/reward ratio against drug entrepeneurs and decrease pressures to increase the size of the market.

Finally, the committee's stand on non-legalization does not allow for controlled trials of various strategies that might provide data needed to make rational decisions in this difficult area.

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1. Rosner F, Wolpaw J, Sechzer PH, et al: Illicit drug use should not be legalized. *NY State J Med* 1992; 92:59–62.

In reply. We agree fully with Dr. Bloom that "the medical community should be committed to increasing the availability of drug rehabilitation services." In fact, the concluding sentence of our paper clearly states that "the committee strongly urges society to provide public education and treatment for all drug addicts who request it."

We do not necessarily accept "the thesis that legalization would increase the use of cocaine/heroin as happened with alcohol after the repeal of prohibition." We point out, however, that in countries where such drugs have been legalized, their use has increased.

We disagree with Dr. Bloom's contention that "scientific substantiation of the deleterious effect of tobacco and alcohol has been relatively recent." Tobacco was first implicated as a cause of cancer in 1761.[1] The overwhelming medical evidence that proved that cigarette smoking is associated with a shortened life expectancy was summarized in the Surgeons General's classic 1964 report on the relationship between smoking and health.[2] The deleterious effects of alcohol have been known since biblical and talmudic times.[3]

We agree with Dr Bloom that "the public will respond positively to enlightened, well-substantiated rational appeals to appropriate behavior" and we support such efforts. Public education and readily available treatment for drug users should be the major goals in combating the public health problem of illicit drug use.

Dr Boba's comments suggest that he ignored the fact that our article on illicit drug use was authored by the members of the Committee on Bioethical Issues of the Medical Society of the State of New York as an educational and informational report. Our committee deals specifically with moral and ethical issues. We attempted to present the various arguments in favor and against the legalization of illicit drug use as well as philosophical considerations and results of physicians' surveys on this matter. The committee, by an overwhelming majority, concluded that illicit drug use should not be legalized. Dr Boba is entitled to disagree. That is his "individual and constitutional freedom." It is unfortunate that he expresses his view in such an angry and aggressive manner.

The topic of illicit drug use involves not only moral, but also political, sociological and public policy issues. The topic is timely and of importance to the medical community. Hence, the validity of publishing our article in a medical journal.

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1. Redmond DE Jr: Tobacco and cancer: The first clinical report, 1761. *N Engl J Med* 1970; 282: 18–23.

2. *Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service.* Washington, DC. US Gov't Printing Office, 1964.

3. Preuss J: *Biblical and Talmudic Medicine* (F. Rosner, trans), New York, Hebrew Publ. Co, 1978.

ERRATA

In the December 1991 issue of the *Journal*, Figures 1 and 2 (pp 526, 527) were mistakenly reversed. Figure 1 is the graph for New York State, and Figure 2 is the graph for New York City. In the May 1991 issue, the second column head in Table I on page 180 should read "Number" only. In the April 1992 issue, on page 131, lines 17 and 18 have typographical errors; both should read "cardiopul*m*onary resusci*t*ation.

The William Hammond Award

The William Hammond Award for Distinguished Service to the *New York State Journal of Medicine* was presented on March 19, 1992 at the Annual House of Delegates meeting to Charles D. Gerson, MD, Associate Clinical Professor of Medicine at Mt. Sinai School of Medicine. Dr Gerson was cited for his many years of conscientious and dedicated service to the *Journal* as both a peer reviewer and as a member of the Associate Editorial Board. A 1958 graduate of Cornell University, Dr Gerson received his MD degree in 1962 from the State University of New York Health Science Center at Brooklyn. He is Chief of the Gastroenterology Clinic at Mt. Sinai Hospital where he is also an Associate Attending Physician. Dr Gerson is the author of numerous papers published in peer-reviewed scientific medical journals. He served in the Peace Corps in Nigeria in 1965–1967 and has since served as a volunteer in other third world countries.

The William Hammond Award is named in honor of William Hammond, MD, who was the editor of the *Journal* in 1960–1972.

The roots of the modern medical malpractice crisis

Medical Malpractice in Nineteenth Century America: Origins and Legacy. By Kenneth Allen DeVille. 319 pp, illustrated. New York University Press, 1990. $34.95 (hardcover)

Paul Johnson, in his book *The Birth of the Modern*, argues that the world as we know it had its foundation built between 1815 and 1830. He describes a time of explosive breakthroughs in technology, thinking, and politics. Kenneth DeVille, in the book under review here, *Medical Malpractice in Nineteenth Century America*, describes the first crisis in medical malpractice litigation as beginning around 1835. It was apparently caused by the very same developments described by Johnson.

Suits for medical malpractice were not a new phenomenon, even in the early 19th century. The fundamental legal principles had been established at least a hundred years before. As DeVille explains, there had been some legal thought that the claims against physicians should arise out of a theory of contract law; however, that was always a minority view. The long-standing, common law concept was that the relationship between physician and patient was one whereby a professional must provide ordinary diligence, care, and skill. Originally, views included the concept that the service was provided without pay. There was always the built-in safeguard that the test would be that of the average physician not the most expert.

It was somewhat unsettling for this reviewer to realize that the landmark New York case of *Pike v. Honsinger*, which was a decision of the New York Court of Appeals in 1898, was not a breakthrough, but merely a restatement of a law that had existed for more than 100 years.

DeVille makes no argument as to whether the law that has governed and still governs medical malpractice is ideal, but clearly he correctly states that it does favor the physician. It is difficult to quarrel with a test that requires ordinary diligence from a professional that amounts to a standard no higher than the average in the professional community. That means that the medical profession has always had the right to establish its own standard.

There are some strong themes from DeVille's analysis of 19th century medical malpractice claims, and one is the impact of Jacksonian democracy. He explains that there was an explosion of medical malpractice claims in western New York State starting in 1840 which he traces, in large part, to the political atmosphere of intense Jacksonian democracy in that area. The core of this political philosophy was that everyone was equal, leading to systematic attacks on anything that resembled privilege. Physicians as a privileged class were easy targets of this new sense of total equality. The political philosophy went so far as to oppose the licensing of physicians on the grounds that it interfered with competition.

To emphasize the importance of the impact of this Jacksonian mentality, the author shows that there was never a medical malpractice crisis in the South. He attributes this to the fact that traditional values continued in the South, somehow insulated from the upheaval in the North and Midwest.

DeVille notes that Jacksonian energy was not simply anti-physician. Shortly after the Civil War, he points out, there began bitter labor-management wars, and corporations and railroads were under constant litigation pressure.

Through a variety of methods (most of which made very good reading), the author details that there were reasons other than political philosophy that caused the first medical malpractice crisis which lasted from 1835 to 1865. Probably the most important issue was the poor quality of medicine being practiced. Although there was some thoughtful work being done in the care of fractures, medicine was not scientific. Formal medical education was skimpy at best. It was disappointing to read "heroic medicine" consisted of bleeding and purging the patient by having him vomit for hours, if not days.

One of the most startling revelations from DeVille is that another cause was that so many suits were instigated by fellow physicians. The physicians who practiced in the 19th century were competing not only with other physicians, but also with a variety of pseudo-medical practitioners called Thomasonians and Homeopaths. Consequently, suggesting a lawsuit against the other doctor in town was an early marketing technique. In addition, the availability of experts to testify for and against physicians was endless.

Lest one jumped to the conclusion that the medical community should protect itself by closing ranks, DeVille notes that after the well-reported case of *Smith v. Goodyear* (New York, 1840) it appeared to the public that organized medicine conspired against physicians who spoke against the establishment. It not only appeared, but was also probably the case, that the profession in ". . .desperate efforts to establish a united front sometimes encouraged physicians to accept substandard performances from their colleagues and implicitly support outdated and dangerous treatments" (p154). Even then there was a backlash against a conspiracy of silence.

The last driving force in the 19th century was the concept of unrealistic expectations held by both the patient and the physician. The best of DeVille's examples deals with the treatment of fractures. In the 1850s, a Dr. Hamilton of Buffalo, New York, pioneered a statistical table to demonstrate that the treatment of most fractures and dislocations did not produce perfect results. Although the Hamilton tables probably aided the defense in fracture cases, by the 1870s, with the introduction of plaster of Paris, the expectations were revised. The concept was promoted that all fracture treatments should produce perfect results. This notion, promoted by physicians of course, led to disastrous results in the courthouse.

DeVille concludes with a warning: the attitudes of the late 19th century have not abated. Liability in tort law has been moving away from proven fault to absolute liability. The expectations from medicine, generated by the medical community, continue to rise. As the author points out, the cultural trends that gave birth to large numbers of medical malpractice suits continue to mature. The courts are bursting, and medical malpractice is just a small part of it.

DeVille's work is recommended reading for anyone who wants to know the roots of the modern malpractice crisis.

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Reprinted from the September/October 1991 issue of Epidemiology Notes (*Vol 6, No 8*), *published by the Division of Epidemiology, New York State Department of Health, Albany, NY.*

Evaluation and clinical management of children with HIV infection

Recent advances have been made in the evaluation and clinical management of children with HIV infection. The early identification of potentially infected children is essential for optimal medical care. New York State leads the nation in the number of reported cases of AIDS in women and children. Recent HIV seroprevalence studies indicate approximately 1,900 infants in New York State are born to women with HIV infection each year and up to 30% of these children may be HIV-infected.

Clinical Management

The US Centers for Disease Control and an expert panel convened at the State Department of Health's AIDS Institute have published guidelines for prophylaxis against *Pneumocystis carinii* (PCP) in children with HIV infection. PCP is frequently the first clinical indication of HIV infection in children and is associated with substantial morbidity and mortality. PCP prophylaxis is now recommended as early as one month of age for seropositive infants with immunologic, laboratory, or clinical indications of HIV infection. CD^4 lymphocyte counts may be used as a guideline for the initiation of prophylaxis.

In the absence of knowledge of the infant's HIV serostatus, the child's physician should be cognizant of other potentially HIV-related symptoms, which may precede PCP in infants and would allow for timely introduction of prophylaxis. Treatment modalities for children with HIV-related symptoms or immunologic abnormalities include intravenous immunoglobulin and antiretroviral therapy. Guidance on the initiation of these therapies and other aspects of care, including immunizations and prophylaxis for significant exposures to communicable diseases, may be obtained by contacting the medical centers listed in Table I.

Early Identification

Although routine health care should include HIV counseling and active encouragement of testing, these services are particularly important for women of childbearing age and should be provided before pregnancy or as early in pregnancy as possible. Any known information regarding the mother's HIV status should be documented in the labor and delivery record and in the newborn's medical record.

Infants born to HIV-infected mothers initially test seropositive and need to be monitored closely to determine

TABLE I. HIV/AIDS Clinical Resource Directory

Hospital	Adult Medicine	Obstetrics	Pediatrics
Manhattan			
Beth Israel Medical Center	Dr. Usha Mathur (212) 420-2620	Dr. Jane Stein (212) 420-4236	Dr. Steve Heaton (212) 420-3288
Cabrini Medical Center	Dr. Michael Mullen (212) 995-6871		
Lenox Hill Hospital	Dr. Michael Tapper (212) 439-3440		
Mount Sinai Medical Center	Dr. David Rose (212) 241-7834	Dr. Rhoda Sperling (212) 241-6718	Dr. Vicki Peters (212) 241-6930
New York Hospital	Dr. Jonathan Jacobs (212) 746-1483	Dr. K. Laguardia (212) 746-3088	Dr. Paul J. Edelson (212) 746-3326
Presbyterian Hospital	Dr. Jay Dobkin (212) 305-8507	Dr. Harold Fox (212) 305-2362	Dr. Jane Pitt (212) 305-5885
St. Claire's Hospital	Dr. Victoria L. Sharp (212) 459-8409		
St. Luke's-Roosevelt Hospital	Dr. Linda Attoe or Dr. Minamoto (212) 523-7434 or 2470	Dr. Amos Grunebaum Dr. Mark Weinrod (212) 523-7750	Dr. Saroja Bakshi or Dr. Ram Kairam (215) 523-5688 or 5818
St. Vincent's Hospital	Dr. William Say or Dr. R. Feldman (212) 790-8034		
Harlem Hospital	Dr. Wafaa El Sadr (212) 491-4035	Dr. Janet Mitchell (212) 491-4421	Dr. Elaine Abrams or Dr. S. Nicholas (212) 928-2228
Metropolitan Hospital	Dr. Eileen Sacharski (212) 230-6771	Dr. Sari Kaminsky (212) 230-6796	Dr. Mahrukh Bamji (212) 230-6841
Bellevue Hospital	Dr. Mindell Seidlin (212) 561-3906	Dr. Machelle Allen (212) 263-6337	Dr. Keith Krasinski (212) 561-3612 or (212) 263-6427

TABLE I. HIV/AIDS Clinical Resource Directory (Continued)

Hospital	Adult Medicine	Obstetrics	Pediatrics
Brooklyn			
Interfaith Medical Center	Dr. Johanna Pindyck (718) 935-7953	Dr. R. Ossowski (718) 604-6520	Dr. Alita Arroyo (718) 604-6260
Luthern Medical Center	Dr. P. de Caprariis (718) 630-8232	Dr. Luz Chengte (718) 630-7239	
University Hospital (SUNY)	Dr. Martin Kramer (718) 270-1432	Dr. Howard Minkoff (718) 270-2860	Dr. Senih Fikrig (718) 270-1908
Kings County Hospital	Dr. Cathy Rones (718) 245-3448	Dr. Louis Camilien (718) 245-4582	Dr. Hermann Mendez (718) 245-4485
Woodhull Hospital	Dr. Alijah Morgan or Dr. C. Ramis (718) 963-8193		Dr. John W. Moohr (718) 963-7958
Brookdale Hospital	Dr. Richard O. Loeb (718) 240-5011	Dr. R. O. Loeb (718) 240-5011	Dr. Norman Klein (718) 240-6357
Brooklyn Hospital (Brooklyn/Caledronian			Dr. Sung Chung (718) 403-8965
Bronx			
Bronx-Lebanon Hospital	Dr. Katherine Anastos (212) 901-8933	Dr. Laurie Solomon (212) 518-5088	Dr. Andrew Wiznea (212) 518-5760
Montefiore Medical Center Center	Dr. Robert S. Klein (212) 920-2199 or (212) 920-6363		
Lincoln Hospital Center	Dr. Dial Hewlett (212) 579-5874	Dr. J. Ayromlooi (212) 579-5830	Dr. Kiran Shan or Dr. H. Sloan (212) 579-5141
Albert Einstein Col. of Medicine	Dr. Carol Harris (212) 918-3670	Dr. D. Jay Gloeb (212) 904-2842	Dr. Arye Rubinstein or Dr. Larry Bernstein (212) 430-2940
North Central Bronx Hospital	Dr. Catherine Small (212) 519-3970	Dr. Joanna Shulman (212) 519-3880	Dr. Marguerite Mayers (212) 519-3345
Queens			
Elmhurst Hospital (718) 830-1323	Dr. Joseph Masci (718) 830-1323	Dr. Romina Kee or Dr. Jasmin Moshirpor (718) 830-1340	Dr. Lila Shah (718) 830-1381
Long Island Jewish Medical Center	Dr. Fred Siegal (718) 470-7698	Dr. Joseph Rovinsky (718) 470-7661	Dr. Vincent Bonagura (718) 470-3300
Queens Hospital Center	Dr. Fred Rosner (718) 883-4050	Dr. Vicki Seltzer (718) 883-4035	Dr. Vincent Bonagura (718) 883-4500
Staten Island			
Staten Island Hospital	Dr. Jordon Glaser (718) 226-9137 or (718) 273-4199	Dr. Hamid Mootabar (718) 226-9850	Dr. Jose Rementeria (718) 226-9360
Long Island			
Nassau County Medical Center	Dr. Aaron Glatt (516) 542-2506	Dr. Peter Hong (516) 542-2256	Dr. Howard Balbi (516) 542-3611
North Shore University Hosp.	Dr. Jonathan Samuels (516) 562-4280	Dr. Solan Chao (516) 562-4440	Dr. Savita Pahwa (516) 562-4641
University Hospital (Stony Brook)	Dr. Jack Fuhrer (516) 444-1660		Dr. Sharon Nachtman (516) 444-2700
Upstate New York			
Westchester County Medical Center	Dr. Harold Horowitz (914) 285-8865	Dr. Nancy Kirshenbaum (914) 285-7689	Dr. Asha Gupta (914) 285-7898
Albany Medical Center	Dr. Steven Szebenyi (518) 445-4439	Dr. Renee Samuelson (518) 445-5015	Dr. Nancy Wade (518) 432-1501
Upstate Medical Center (Syracuse)	Dr. Donald Blair (315) 464-5533	Dr. John Hagen (315) 464-4732	Dr. Leonard Weiner (315) 464-6331
Strong Memorial Hospital (Rochester)	Provider Hotline (for all three disciplines): (716) 275-8418		
Erie County Medical Center	Dr. Ross Hewitt (718) 898-4419	Dr. Laurence Gugino (716) 898-3800 or (716) 898-7737	Dr. Howard Faden or Dr. Maureen Montgomery (716) 898-7737

HIV Laboratory Services Directory

For further information about specialized laboratory evaluation of infants born to HIV-infected mothers, contact the New York State Department of Health Wadsworth Center for Laboratories and Research.

- For information about available tests, their accuracy and diagnostic value, contact: Dr. James Conroy, Retroviral Immunology Laboratory, (518) 474-7281.
- For infomation about submitting samples, contact: Ms. Tina Charbonneau, Retroviral Immunology Laboratory, (518) 474-8579.

whether or not they are actually infected. Specialized laboratory evaluation is necessary to establish their immunologic and infection status. Information on available diagnostic tests may be obtained from the Department of Health's Wadsworth Center for Laboratories and Research.

When the mother's HIV serologic status has not been determined, the infant's doctor should routinely counsel the mother regarding the importance of knowing her own and therefore her infant's serostatus and should encourage maternal HIV testing. The physician of a child in foster care may request consent for HIV testing from the local Department of Social Services. In cases where the mother is HIV-infected, other family members may also be HIV-infected. Evaluation, treatment, and coordination of services should be provided to all family members as appropriate.

Care Consultation

Consultation on clinical evaluation and management of HIV-infected women and children is available through medical centers experienced in treating HIV-infected women and children. A list of these centers around New York State is included in this paper.

Clinical protocols developed by the AIDS Institute's Pediatric HIV Medical Criteria Committee may be obtained by contacting the Pediatric/Maternal HIV Unit at (518) 486-6048.

BOOK REVIEWS

DRUG-RELATED PROBLEMS IN GERIATRIC NURSING HOME PATIENTS

By James W. Cooper, PhD. 148 pp. Binghamton, NY, Haworth Press, Inc, 1991. $10.95 (softcover)

Any practitioner who cares for older adults has experienced clinical problems related to adverse drug reactions caused by polypharmacy. Because of the multitude of conditions afflicting the frail elderly person, pharmaceutical management of these problems often occurs in the setting of an extended care facility. Unique to this setting is that there are a large number of health care professionals assessing the institutionalized adult on a daily basis, and some of this assessment includes dispensing medications that have been ordered in some cases on a "prn" basis.

Unique to this setting, also is that the frail sometimes medically unstable patient is examined by the physician only on a monthly basis, unless the nursing staff contacts the doctor with an acute problem. In an ambulatory or acute-care setting, this evaluation and treatment often occurs with medication management being more frequently assessed.

It is from this framework that Dr. Cooper's book reviewed many of the common pharmaceutical problems related to nursing home care. The author's purpose in writing this book was to emphasize the magnitude of drug-related problems that occur very frequently in this setting. Dr. Cooper attempted to breakdown and catalog the various types of problems that do occur and then present practical solutions to prevent them. The author illustrates the all-too-common catastrophic events relating to medications leading to serious morbidity and mortality in this patient population.

The author is well qualified to write on this subject, being professor and head of the Department of Pharmacy Practice at the University of Georgia, College of Pharmacy, Athens, Georgia. He has authored over 200 articles on geriatric pharmacology and is editor of the *Journal of Geriatric Drug Therapy and Clinical Consult.* He is involved in teaching and research in clinical pharmacy in ambulatory and long-term care settings.

Drug-related Problems in Geriatric Nursing Home Patients covers diverse topics concerning drug-related problems commonly encountered in a long-term care institution. Much of what is written can be applied to other settings as well. Dr Cooper provides an overview of drug-related problems pertaining to medication errors, adverse-drug interactions, and drug reduplication. He also includes chapters on the nutritional aspects related to this area and the use of laboratory testing. I found his comments on the morbidity and mortality, as well as the economic impact, of adverse reactions very interesting and revealing of the significance of this aspect of geriatric care.

Dr Cooper presents his material from the viewpoint of a pharmacist. As a result, some of his comments could be construed as making the physician the "bad guy" and a significant contributor to this very real problem. On very close review of the text of each chapter, I found that his comments were valid and that he did attempt to give a balanced overview. He also contends that all persons involved in the care of the patient have an equal responsibility to preventing adverse drug reactions.

Because many nursing homes are now using the services of a consulting pharmacist to review each patient's chart on a regular basis, the consultant should be an important part of the health care team. The author constructs various scenarios of how the pharmacist could interact and be actively involved in the care of each patient.

This book will act as an excellent primer for students in the health professions exploring this area of care. I do not believe it is the intention of the author to present an exhaustive text on geriatric pharmacology and for this reason it will not act as a reference text to be used in the nursing home setting. For medical educators, it can be incorporated into a curriculum in geriatric medicine and ambulatory care.

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EVALUATION AND TREATMENT OF THE PSYCHOGERIATRIC PATIENT

Edited by Diane Gibson, MS, OTR. 111 pp. Binghamton, NY, Haworth Press, 1990. $22.95 (hardcover)

This book has also been published under the title *Occupational Therapy in Mental Health.* With that more accurate title it probably would not have been given to a psychiatrist for review. It is a slim volume composed of six papers by different authors. It suffers the unfortunate fate of some edited works, namely the repetition of the all too familiar geriatric statistics. Although the book improves as one goes along, it never achieves what it purports to do. It does not read well, partially because of the distracting citations provided within the text.

The chapter written by a psychiatrist on learned helplessness in the elderly was scholarly but overly focused on theory with insufficient clinical material. Such a presentation would be more appropriate for a psychology text.

Only after completing half the book is one presented with some assessment batteries and scales. However, they are embedded within "work-up" protocols and a profusion of abbreviations that only serve to distract and confuse the reader.

Toward the end of the book, we are actually presented with clinical material replete with a full occupational therapy assessment summary that clearly defines appropriate treatment interventions and by itself demonstrates how a thorough multidimensional assessment leads easily and logically to intervention strategies.

In the penultimate chapter, there is a marvelously lucid description of a transitional program assisting psychogeriatric patients in negotiating different levels of care. It is the best chapter in the book.

On the whole, the book is disappointing. The overuse of abbreviations was distracting and annoying, and the writing was stilted, unclear, and difficult to read. The title implies that the reader will learn specific evaluative tools and specialized treatment techniques for this population. Would that be true. As a clinician, I did not feel that my armamentarium had been enhanced.

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PSYCHIATRY IN THE NURSING HOME: ASSESSMENT, EVALUATION, AND INTERVENTION

By D. Peter Birkett, MD. 175 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

It is my opinion that you can tell whether the author of an article or book is or has been a practicing clinician because of the tenor and practical material included in the publication. Dr Birkett has written a text that reflects this background and clinical experience in his practical approaches and insights into the problems of caring for the mentally impaired adult in the nursing home setting. His credentials, which also verify his clinical experience, include being the medical director of the Riverside Nursing Home in New York City, a faculty member at Columbia University, and having a private practice in psychiatry. Although the major emphasis of this book is on psychiatric conditions, much of the information pertains to the general care of patients and would be of interest to clinicians in any medical field.

The author has organized the book in an interesting fashion. He begins the text with a history of federal legislation that deals with the care of the mentally impaired person. He then follows with a description of the various types of personnel staffing a skilled and intermediate nursing care facility; this is important for a person not familiar with this type of health care setting. Much of the book deals with a description of the common types of mental illness seen in older adults and problems of management in this care setting. Both pharmaceutical and "non-medical" approaches to treating these conditions are also included. He closes the book with several interesting sections concerning medical aspects of care, including mention of problems of caring for AIDS-victims and policy issues of institutions in dealing with chronically ill patients.

I feel that even though the author did not write exhaustively on any of his topics, his attempt to address the complex issues confronting the physician, nursing home staff, and most importantly, the patient deserves honorable mention. The sections dealing with psychiatric conditions were naturally the most complete. I congratulate the author for not indicating that the very common problem of depression seen in the older adult and in particular in this health care population is not part of normal aging. For the demented patient, the author offers practical approaches to this frequently encountered problem in skilled nursing facilities. Other severe psychiatric conditions, not as common, are also addressed.

In addition to psychiatric diseases, the book also covers areas rarely addressed in the geriatric texts of any clinical specialty. Dr Birkett touches on issues such as violence, sexual behavior, toileting problems, hoarding, the noisy patient, and issues that frequently arise between the patient, staff, and family member. These issues are often considered academically "soft," but often consume much of our energy and time. Unfortunately, it is not possible to study these problems in any statistical fashion. Rather, one must rely on anecdotal descriptions.

The author does not use technical language that would preclude the non-psychiatrist from understanding and using the contents of this text. His presentation of material is often humorous, when appropriate. His recognition of the sometimes tragic dilemmas patients often find themselves in when institutionalized reflects the type of sensitivity that is unfortunately lacking in most geriatric text.

The production, print quality, and layout of the book are adequate. There is a reference section after each chapter and an easily followed index.

I have rarely enjoyed reviewing a text as much as I have this one. My only criticism is that many important areas are only mentioned in passing. I look at this book more as a primer that introduces non-psychiatrists to the field of consultative psychiatry in the nursing home setting. Certainly, students of any clinical area and administrators of long-term care institutions would benefit from reading this book. I applaud this sensitive and intuitive work.

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GERIATRIC RADIOLOGY

Edited by Manuel Viamonte, Jr., MD. 228 pp, illustrated. Baltimore, MD, Williams & Wilkins, 1991. $65.00 (hardcover)

This book is a multiple authored text covering a select group of important geriatric disorders and their imaging manifestations. All of the authors are well qualified to write their chapters. To my knowledge, this is the first book written that covers the imaging aspects of aging and age-related diseases.

The topics include an overview of geriatric medicine, care of the geriatric patient in the radiology department, arthropathies, osteoporosis, respiratory diseases, cardiovascular problems, alimentary tract diseases, genitourinary diseases, cancer, and brain disorders of the elderly. The book is well written, with good illustrations and charts. In each chapter, clinical and imaging aspects of the disorders are presented.

The chapter on the care of the geriatric patient in the radiology department describes problems that are unique to an aged and debilitated patient. The importance of the patient understanding the test that is to be performed, and the informed consent required is stressed. The care of the patient and avoiding harm while obtaining the best possible imaging study are covered. The relative merits of the various types of intravascular contrast media, and the indications for their use are described. A table of drugs useful in the treatment of contrast media reactions is also included.

The common arthropathies of the elderly and their differential diagnoses are presented. The role of advanced imaging methods (CT and MRI) in these disorders is described.

The clinical epidemiology and the detection of postmenopausal and senile osteoporosis are discussed. Radiological aspects are presented as well. The different techniques for assessing osteoporosis and quantitative measurement of bone density are reported.

The radiographic changes that occur with aging, in the thoracic cage, mediastinum, and lungs are described. The imaging options for evaluating the respiratory system, including common neoplasms of the elderly, chest wall lesions, airway disease, infection, diffuse lung disease, adult respiratory distress syndrome, pneumoconiosis, pleural disease, and mediastinal disease are presented. The evaluation of cardiovascular problems of the elderly by means of echocardiography, electrocardiography, and radionuclide studies is discussed.

The incidence and manifestations of gastrointestinal disease in the elderly is different than in younger patients. Neuromuscular degeneration, ischemia, and neoplasia are more common mechanisms. Dysphagia is a common symptom that requires a careful fluroscopic study to define its causes. Other alimentary diseases covered are gastrointestinal polyps, cancer, malabsorption syndromes, ischemic bowel disease, intestinal obstruction, bleeding, diverticular disease, and anorectal dysfunction.

The genitourinary disorders described include benign prostatic hyperplasia, prostate cancer, incontinence, and urological malignancies.

The final chapter is a review of the normal brain and abnormal brain in the elderly. The role of various imaging procedures in the diagnosis of dementing disorders (Alzheimer's disease, Parkinson's disease, alcoholic dementia, multiple-infarct dementia [Binswanger's disease], Pick's disease, progressive supranuclear palsy, normal pressure hydrocephalus, Huntington's disease, and Creutzfeldt-Jacob disease) is analyzed.

This book will be of value to the radiologist and to the physician caring for the elderly. It not only describes many of the important geriatric disorders, but also presents guidelines for their evaluation and for the determination of which method of imaging should be used.

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DEFEATING PAIN: THE WAR AGAINST A SILENT EPIDEMIC

By Patrick D. Wall, MD, and Mervyn Jones. 285 pp. New York, Plenum Publishing Corp, 1991. $24.95 (hardcover)

If I were browsing through the medical section of my favorite bookstore and saw this book, I would be attracted to the subject matter since I live with chronic pain. However, on closer inspection I would search for the credentials of the authors, Dr Patrick D. Wall and Mervyn Jones. My immediate reaction would be "Oh no, a chiropractor or another osteopath has found a ghost writer and is about to present another one of those trendy pop methods to a quick cure for all pain."

I am committed to the traditional medical profession's expertise. Why did the publisher elect to omit the letters MD after the senior author's name? American readers are not accustomed to identifying renowned medical experts as "doctor." I suggest that the publisher create a new book jacket for the American market or qualify in detail, within the biography on the jacket, that Dr Wall is indeed a medical doctor with impressive qualifications.

Whereas traditionally we have looked to the nurse or doctor as the guardians and controllers of pain management, the qualified authors of this no-holds-barred informative, hand-sized encyclopedia suggest that effective pain management is a multidisciplinary responsibility, including both professionals and the patient. The reader is offered a journey through an expert, concise, definitive, and practical understanding of the sources of pain and the realistic treatments available.

Wall and Jones openly admit the past, old-fashioned medical approaches that invited ridicule when pain was not easily explained. Using modern scientific data, they bridge the gap from the past to the exciting modalities of the 1990s, bringing pain control to the forefront where it rightly belongs. No longer is pain seen as only a symptom that is treated by discovering the cause. The empowerment to treat the variations of pain described in detail is not limited to strict classifications but includes the person in pain for which there has been no diagnosis on the general list of known diseases. This treatise offers resources and delivers hope as the reader discovers that the problem of pain can be more social than medical.

The good news is that although science seemingly perpetuates the "silent epidemic" (pain) and overlooks pain management while enthusiastic research continues for the popular enemies, cancer, heart disease, and AIDS, Wall and Jones assure the reader that intellectual progress in this area is now evident (or being made) and many fine medical schools are recognizing the need to alleviate pain as extended life has become fact. Patients, their nurses, and their doctors now seek to restore as well as extend. By necessity, this must include high-tech pain management.

This constructive manual, should be required reading for all medical and nursing students. The informed, sophisticated patient will find it a welcome handbook for the home library. The practicing physician who was not been trained in the new methods of pain control, such as patient-controlled-analgesia (PCA), will be challenged by the haunting philosophies offered regarding the many pain categories explored.

Pain is a universal experience, yet who can reliably measure pain, and how can the mystery of phantom pain be understood by one who has not lost a limb? Pain is a vital reporting system, and the aim of modern medicine is to diagnose and treat disease. The responsibility for effective pain control can be orchestrated among health care providers, family, and patients. The authors deliver a powerful message about patients and physicians in the this exciting era who are increasingly open to eliminating the usual parental role (of the doctor) to that of advisor, with the patient as partner.

This book dispels the social conditioning we have been heir to and brings the reader to the healthy vanguard of world class pain care.

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DISINFECTION, STERILIZATION, AND PRESERVATION

By Seymour S. Block, PhD. 1,162 pp, illustrated. Malvern, Pa, Lea & Febiger, 1991. $175.00 (hardcover)

The fourth edition of this book provides an up-to-date, comprehensive reference source that will certainly appeal to a multidisciplinary audience of infection control practitioners, microbiologists, and other health care facilities who function as consultants on questions of appropriate disinfection and sterilization. The book is organized into nine sections, the chapters within each section being written by one of seventy-nine expert authors. Some sections could easily qualify as complete publications. The introduction is followed by sections on the fundamental principals of the activity of various disinfectants and antiseptics. The agents are characterized by chemical and physical properties, target organisms, and their uses in sterilization, disinfection, food sanitation and preservation. A final section outlines methods of testing for the efficacy of these agents. The tables and graphs are extremely useful in helping to summarize the many segments of complex, technical materials contained herein.

The introduction contains a fascinating historical review. Biblical references to such practices as the use of sulphur as a disinfectant circa 800 BC to events of the middle ages and then to accomplishments of the 19th and 20th centuries gives one a perspective of the developments in this field over time. The many interesting and delightful anecdotes set the tone for the wealth of knowledge that follows. The introductory section also contains a chapter of term definitions required in the lexicon of anyone interested in disinfection, sterilization, and preservation.

The section on fundamental principles of activity gives extensive coverage of the topics of thermal destruction of microorganisms, antimicrobial processes of both a physical and chemical nature as well as mechanisms of resistance to antimicrobial agents. This section contains in-depth coverage of mathematical modeling techniques with supporting formulas and graphs. A thorough understanding of this section requires a background in calculus and statistics.

The third section contains fifteen chapters, each concerned with a different chemical disinfectant or antiseptic compound. Persons involved in infection control, such as this reviewer, will be particularly interested in the chapters covering chlorine compounds, iodine compounds, alcohols, phenolic compounds, chlorhexidine, and the very extensive chapter on quaternary ammonium antimicrobial compounds. Included among this section's chapter is one on the disinfection and control of the human immunodeficiency virus (HIV). It covers what is currently known about the nosocomial transmission of HIV, including the recent dental procedure associated cases.

Another section covers sterilization by heat, filtration, ultraviolet (UV) irradiation, ionizing irradiation, ethylene oxide, and glutaraldehyde. The chapter on sterilization by UV irradiation is particularly timely given the current epidemic occurrence of tuberculosis in New York City and the Centers for Disease Control's recommendation for the use of UV lights in acid fast bacillus (AFB) isolation.

Given the current emphasis on infection control, the section on health-related applications will appeal to an audience ranging from health care planners to health care providers. One chapter is devoted to disinfection and sterilization procedures for dentistry practices. Other topics covered by the text but not reviewed here are food, medications, cosmetics, and veterinary associated concerns. This review cannot begin to touch on the vast numbers of topics contained in this 1,275 page book. The book is comprehensive in nature and will be useful as a resource to persons in infection control and others who need timely information on disinfection, sterilization, and preservation. It should therefore be viewed as a welcome addition to our current armamentarium of reference material.

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CONTEMPORARY INTERNAL MEDICINE: CLINICAL CASE STUDIES

Edited by Juan M. Bowen, MD, and Ernest L. Mazzaferri, MD. 286 pp, illustrated. New York, Plenum Publishing Corp, 1991. $75.00 (hardcover)

Teaching by case study is not new, but is still a very effective way of instructing students. It reminds me of the case study method used at the Harvard Business School and the Harvard School of Public Health. Although it is case oriented, it allows for a broad discussion of diagnosis and discriminative diagnostic techniques, as well as therapeutic options. Perhaps most important of all, it keeps the focus of attention on the patient, the person who so often is lost sight of in the maze of laboratory results. The editors note that "the cases presented in this book are discussed in a format that shows the art of medicine in a framework of science." They emphasize that the discrimination of tests and procedures is a part of the art of medicine rather than its science.

Twenty-five cases are presented by 31 authors, all collected from the Ohio State University Hospitals and discussed by faculty members of the Department of Internal Medicine. The cases vary from such common diagnoses as mitral regurgitation, asthma, and thyrotoxicosis to more unusual conditions, such as thrombotic thrombocytopenic purpura, solitary pulmonary nodule, and temporal arteritis. Each case begins with a summary of the history and pertinent physical findings, along with the laboratory findings necessary to formulate a diagnosis. The discussion varies somewhat from case to case, but generally includes clinical features, differential diagnosis, pathophysiology, treatment, clinical course, and prognosis. The pathophysiological discussions are clear, yet detailed, and appropriately illustrated with drawings and tables. The references used for the discussions contain some of the most recent knowledge about the subject, an aspect that adds considerable value to the presentations. However, with relatively few photographic illustrations, one wonders about the cost of the volume ($75.00).

The case orientation of this volume is relatively unique among current medical monographs. Most are subject oriented, using statistical approaches to cases, more the science of medicine rather than its art. The authors of the case discussions are a distinguished group of professors of the Department of Internal Medicine of Ohio State University Medical School, which speaks to the quality of the discussions.

If one is looking strictly for information, probably a textbook of medicine, dollar for dollar, would be more valuable. However, if one is looking for a clinical approach to patients, this volume would fit to a T. However, if I were a medical student on a restricted budget, I would find this book in the medical library and use it there.

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POSITIVE AND NEGATIVE SYNDROMES IN SCHIZOPHRENIA: ASSESSMENT AND RESEARCH

By Stanley R. Kay, PhD. 283 pp. New York, Brunner/Mazel Publishers, 1991. $39.95 (hardcover)

In the late 1970s, mental health professionals working with schizophrenia began to review critically the etiological, clinical, and prognostic significance of the Kraepelinian typology with regards to schizophrenia. Tying schizophrenics into paranoid, hebephrenic, catatonic, and simple types, could be deceptively helpful, but did not help in the understanding of etiology, change clinical treatment, or affect prognosis. Therefore, interest developed in grouping symptoms together in a more helpful fashion. In the early 1980s, emphasis began to be placed on positive versus negative schizophrenia. Positive schizophrenia reflects a prominence of hallucinations, delusions, formal thought disorder, bizarre, or disorganized behavior. Negative schizophrenia reflects a prominence of affective flattening, anhedonia, a sociality, apathy, attentional impairment, and poverty of speech. This book documents Dr Kay's work in developing the concept of positive and negative syndromes as a helpful way of understanding schizophrenia.

Stanley Kay, PhD, was Chief of the schizophrenia research program and Associate Clinical Professor in the Department of Psychiatry, Albert Einstein College of Medicine. He was also the Director of Psychological Research at Bronx Psychiatric Center. He published numerous papers on schizophrenia in the 1970s and 1980s. He was clearly qualified to give an authoritative discussion of this subject. Of note, Dr Kay died suddenly in 1990, this book being among his last completed written works.

Since the 1950s, there has been a major effort to better understand and treat schizophrenia. In spite of this major effort, there remains a lack of clarity in the understanding of this disorder. This book is a helpful addition to the body of knowledge about schizophrenia. It presents a different way of classifying schizophrenia and documents the potential effects of a better understanding of the etiology, treatment, and prognosis of this chronic disorder.

The book begins by discussing the need for a better way of sub-typing schizophrenia and introduces the concept of positive and negative syndromes. Dr Kay then proceeds to discuss his method of assessing positive and negative syndromes using the positive and negative syndrome scale (PANSS). He argues the usefulness of this instrument and discusses how to implement it. A chapter discusses the Spanish adaptation of the PANSS. The book then discusses research done by Dr Kay in regard to etiology, treatment, and prognosis, using the concept of positive and negative syndromes. The book ends with a discussion of how the positive and negative model provokes new theories about schizophrenia.

Dr Kay writes in a clear, readable style. The book is well organized enabling the reader to focus either on the PANSS diagnostic instrument or on the more theoretical concerns of the book. The appendix gives a detailed documentation of how to use the PANSS. A weakness of the book is that the majority of the text is devoted to the development, validity, and use of the PANSS. I would have wished a more detailed discussion of research around the positive-negative syndrome model, and the implications to the understanding of schizophrenia. A majority of studies Dr. Kay used to validate theories regarding the usefulness of this model employed small numbers of subjects, making conclusions drawn from then tentative. However, Dr Kay clearly states that the concepts presented in this book are not definitive. He means this book to be an introduction of new models and methods for understanding schizophrenia and proposes that future investigation will validate the usefulness of positive and negative syndrome model.

Schizophrenia continues to be one of the greatest challenges in the field of mental health. This common disorder has a profound impact on individuals, families, and societies around the world. In this book, Dr Kay has developed a model for better understanding and treatment this disorder. Although not the final word, I believe that this book is important reading for both the clinician and researcher working with schizophrenia.

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OBITUARY LISTINGS

Gaspar Mathew Adamo, MD, Long Island. Died February 14, 1992; age 88. Dr Adamo was a 1933 graduate of Rush Medical College of Chicago. He was a Fellow of the American College of Surgeons and Fellow of the International College of Surgeons. His memberships included the Medical Society of the County of Queens and the Medical Society of the State of New York.

Maurice Adler, MD, New York. Died February 18, 1992; age 79. Dr Adler was a 1951 graduate of the Universitat Basel Medizinische Fakultat, Switzerland. He was a member of the New York County Medical Society and the Medical Society of the State of New York.

Edward M. Auringer, MD, Hornell. Died February 1, 1992; age 72. Dr Auringer was a 1945 graduate of the Albany Medical College of Union University. He was a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the Radiological Society of North America, Inc, the Steuben County Medical Society, and the Medical Society of the State of New York.

Frank Berchenko, MD, New York. Died August 28, 1991; age 81. Dr Berchenko was a 1935 graduate of the Universitat Zurich Medizinische Fakultat, Switzerland. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a Fellow of the American Psychiatric Association. His memberships included the American Psychoanalytic Association, the Medical Society of the County of New York, and the Medical Society of the State of New York.

Z. John Betlejeski, MD, Altamont. Died August 30, 1991; age 78. Dr Betlejeski was a 1943 graduate of the State University of New York Health Science Center at Brooklyn, New York. He was a Diplomate of the American Board of Surgery. He was a the Fellow of American College of Surgeons. He was a member of the Albany County Medical Society and the Medical Society of the State of New York.

Charles Joseph Bivona, MD, Newburgh. Died November 7, 1991; age 80. Dr Bivona was a 1941 graduate of the New York Medical College, Flower and Fifth Avenue Hospitals, New York. He was a Fellow of the American Academy of Family Practice. He was a member of the Orange County Medical Society and the Medical Society of the State of New York.

Oliver John Blaber, MD, Sayville. Died March 21, 1992; age 69. Dr Blaber was a 1948 graduate of the State University of New York Health Science Center at Brooklyn, New York. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologist. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

William Herbert Burwig, MD, Tonawanda. Died March 7, 1992; age 93. Dr Burwig was a 1923 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the International College of Surgeons, the American College of Surgeons, the American College of Obstetricians and Gynecologist. His memberships include the Academy of Medicine, the Erie County Medical Society, and the Medical Society of the State of New York.

Victor Louis Cohen, MD, Buffalo. Died March 16, 1992; age 88. Dr Cohen was a 1929 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Allergists. His memberships included the American Academy of Allergy, the New York State Society of Internal Medicine, the Erie County Medical Society, and the Medical Society of the State of New York.

Patrick Rudolph Coseglia, MD, Niagara Falls. Died March 10, 1992; age 64. Dr Coseglia was a 1953 graduate of the Georgetown University School of Medicine, Washington. His memberships include the Erie County Medical Society and the Medical Society of the State of New York.

Edward Victor Denneen, MD, Annapolis, Maryland. Died June 4, 1991; age 92. Dr Denneen was a 1925 graduate of Cornell University Medical College, New York. He was a Diplomate of the American Board of Surgery. He was a Fellow of the American College of Surgeons. His memberships included the New York State Surgical Society, the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Carlo E. DeSantis, MD, Pittsford. Died February 25, 1992; age 60. Dr DeSantis was a 1961, graduate of the University of the Buffalo School of Medicine. He was a Diplomate of the American Board of Ophthalmology. He was a Fellow of the American College of Surgeons. His memberships included the American Opthalmological Society, the American Academy of Ophthalmology and Otolaryngology, the Academy of Medicine, the New York Ophthalmology Society, the Medical Society of the County of Monroe, and the Medical Society of the State of New York.

Jacob B. Dranitzke, MD, Patchogue. Died December 19, 1991; age 89. Dr Dranitzke was a 1924 graduate of the Columbia University of the College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

John W. Draper, MD, San Mateo, California. Died October 12, 1991; age 87. Dr Draper was a 1931 graduate of Cornell University Medical College, New York. He was a Diplomate of the American Board of Urology. He was a Fellow of the American College of Surgeons. His memberships included the American Geriatrics Society, the American Urological Assocation, the New York State Urological Society, the Academy of Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Harry Russell Faller, MD, Fort Jervis. Died February 22, 1992; age 82. Dr Faller was a 1936 graduate of the St. Louis University School of Medicine. He was a Fellow of International College of Surgeons. His memberships included the American College of Occupational Medicine, the Orange County Medical Society, and the Medical Society of the State of New York.

Bertold B. Feldman, MD, New City. Died February 6, 1992; age 89. Dr Feldman was a 1929 graduate of the Universita Karlova Fakulta Lekarska, Praha. He was a Fellow of the American College of Gastroenterology. His memberships included the American Gastro-Enterological Association, the New York Academy of Gastroenterology, and the Rockland County Medical Society, and the Medical Society of the State of New York.

Joseph Ernest Fracasso, MD, Buffalo. Died March 13, 1992; age 61. Dr Fracasso was a 1962 graduate of Marquette University School of Medicine. He was a Diplomate of the American Board of Internal Medicine. He was a member of the Erie County Medical Society and the Medical Society of the State of New York.

Robert Parker Freeman, MD, Worton, Maryland. Died January 16, 1992; age 87. Dr Freeman was a 1939 graduate of Universite de Lausanne Faculte De Medecine. He was a member of the American Academy of Family Practice, the Kings County Medical Society, and the Medical Society of the State of New York.

Murry Nathan Friedman, MD, New Hyde Park. Died February 25, 1992; age 73. Dr

Friedman was a 1943 graduate of the New York University College of Medicine, New York. He was a Diplomate of the American Board of Surgery. He was a Fellow of American College of Surgeons. He was a member of the Nassau County Medical Society and the Medical Society of the State of New York.

Benjamin Futernick, MD, Great Neck. Died March 10, 1992; age 76. Dr Futernick was a 1945 graduate of Chicago Medical School. He was a member of the Queens County Medical Society and the Medical Society of the State of New York.

Donald David Goldbert, MD, Jamaica. Died March 5, 1992; age 71. Dr Goldbert was a 1955 graduate of Rijks-Universiteit Te Leiden Faculteit Der Geneeskunde. He was a Diplomate of the American Board of Orthopedic Surgery. He was a Fellow of the International College of Surgeons. His memberships included the Pan American Medical Association, the American Academy of Orthopaedic Surgeons, the Queens County Medical Society, and the Medical Society of the State of New York.

Sidney Saul Greenberg, MD, New York. Died April 22, 1991; age 80. Dr Greenberg was a 1937 graduate of the State University of New York Health Science Center at Brooklyn, New York. He was a Fellow of the American College of Cardiology. His memberships included the American Society of Addition Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Orman Gregersen, MD, Jamesport. Died March 10, 1992; age 99. Dr Gregersen was a 1921 graduate of the Jefferson Medical College of Philadelphia. He was a member of the Queens County Medical Society and the Medical Society of the State of New York.

Edmund Roet Heenan, MD, Setauket. Died April 13, 1992; age 82. Dr Heenan was a 1936 graduate of the State University of New York Health Science Center at Brooklyn, New York. He was a member of the Suffolk County Medical Society and the Medical Society of the State of New York.

Hyman Dave Kachalsky, MD, Rye. Died March 30, 1992; age 63. Dr Kachalsky was a 1955 graduate of the State University of New York Health Science Center at Brooklyn, New York. He was a Diplomate of the American Board of Psychiatry and Neurology. He was a Fellow of the American Psychiatric Association. His memberships included the American Society Adolescent Psychiatry, the American Academy Child & Adolescent Pyschology, the Westchester County Medical Society, and the Medical Society of the State of New York.

Hayk Kalenderian, MD, Queens Village. Died March 1, 1992; age 77. Dr Kalenderian was a 1940 graduate of the Istanbul Universitesi Tip Fakultesi. He was a member of the Queens County Medical Society and the Medical Society of the State of New York.

James S. Kime, MD, Buffalo. Died November 16, 1991; age 82. Dr Kime was a 1934 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Surgeons. His memberships included the Academy of Medicine, the Erie County Medical Society, and the Medical Society of the State of New York.

Theodor Kron, MD, New York. Died November 8, 1991; age 90. Dr Kron was a 1927 graduate of the Julius-Maximilians-Universitat Medizinische Fakultat, Wurzburg, Bavaria. He was a member of the Bronx County Medical Society and the Medical Society of the State of New York.

Augustus T. Legato, MD, New York. Died January 31, 1992; age 38. Dr Legato was a 1980 graduate of the Chicago Medical School. He was a Diplomate of the American Board of Anesthesiology. His memberships included the American Society of Anesthesiologists, Inc, the New York State Society of Anesthesiologists, the New York County Medical Society, and the Medical Society of the State of New York.

Carmine Jos Loffredo, MD, Saratoga Springs. Died March 19, 1992; age 86. Dr Loffredo was a 1930 graduate of the New York Medical College, Flower and Fifth Avenue Hospitals, New York. He was a member of the Schenectady County Medical Society and the Medical Society of the State of New York.

Charles L. Mache, MD, Buffalo. Died February 2, 1992; age 68. Dr Mache was a 1947 graduate of Yale University School of Medicine, New Haven. He was a Diplomate of the American Board of Pediatrics. He was a Fellow of the American Academy of Pediatrics. He was a member of the Erie County Medical Society and the Medical Society of the State of New York.

George Martin Masotti, MD, Williamsburg, Virginia. Died February 12, 1992; age 82. Dr Masotti was a 1933 graduate of University of Buffalo School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Psychiatric Association, the Academy of Medicine, the Erie County Medical Society, and the Medical Society of the State of New York.

Melvin Moore, MD, Brooklyn. Died March 22, 1992; age 68. Dr Moore was a 1947 graduate of the Chicago Medical School. He was a Diplomate of the American Board of Radiology. He was a Fellow of the American College of Radiology. His memberships included the New York Roentgen Society, the Kings County Medical Society, and the Medical Society of the State of New York.

Morris Moskowitz, MD, Waterbury, Connecticut. Died March 28, 1992; age 88. Dr Moskowitz was a 1938 graduate of the University of St. Andrews Conjoint Medical School, St. Andrews and Dundee. His memberships included the American Academy of Family Practice, the Queens County Medical Society, and the Medical Society of the State of New York.

Daniel Benjamin Perry, MD, Dublin, Georgia. Died December 3, 1991; age 84. Dr Perry was a 1948 graduate of Meharry Medical College, Nashville. He was a Diplomate of the American Board of Anesthesiology. He was a Fellow of the American College of Anesthesiologists. His memberships included the American Society of Anesthesiologists, Inc, the New York State Society of Anesthesiologists, the New York County Medical Society, and the Medical Society of the State of New York.

Hans Pollak, MD, Gloversville. Died March 11, 1992; age 79. Dr Pollak was a 1939 graduate of the Universitat Basel Medizinische Fakultat, Switzerland. His memberships included the American Society of Anesthesiologists, Inc, the Fulton County Medical Society, and the Medical Society of the State of New York.

Dominick P. Scagnelli, MD, Phoenicia. Died March 16, 1992; age 79. Dr Scagnelli was a 1941 graduate of Universita Degli Studi Di Bologna, Facolta Di Medicina E Chirurgia. His memberships included the American Academy Family Practice, the Ulster County Medical Society, and the Medical Society of the State of New York.

Samuel Schechter, MD, Briarcliff Manor. Died January 6, 1992; age 96. Dr Schechter was a 1923 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of North America, Inc, the Westchester County Medical Society, and the Medical Society of the State of New York.

Sigmund Schwarzer, MD, New Hyde Park. Died April 1, 1992; age 71. Dr Schwarzer was a 1949 graduate of the Universitat Heidelberg Medizinische Fakultat, Baden. He was a Diplomate of the American Board of Pediatrics. He was a Fellow of the American Academy of Pediatrics. He was a member of the Queens County Medical Society and the Medical Society of the State of New York.

Maurice N. Sholtzow, MD, Brooklyn. Died May 24, 1991; age 79. Dr Sholtzow was a 1940 graduate of the Licentiate of the Royal College of Physicians of Edinburgh.

His memberships included the Academy of Psychosomatic Medicine, the Aerospace Medical Association, the American Geriatrics Society, the Pan American Medical Association, the American Academy Family Practice, the Clinical Society, New York Diabetes Association, the New York Cardiological Society, the Kings County Medical Society, and the Medical Society of the State of New York.

A. Irving Swiller, MD, Tamarac, Florida. Died March 22, 1992; age 79. Dr Swiller was a 1940 graduate of Licentiate of the Royal College of Physicians of Edinburgh, Scotland. His memberships included the International Society of Hematology, the New York State Society of Internal Medicine, the Kings County Medical Society, and the Medical Society of the State of New York.

Byron Calvin Tillotson, MD, Fort Edward. Died March 19, 1992; age 97. Dr Tillotson was a 1921 graduate of the University of Vermont College of Medicine, Burlington. He was a Fellow of the American College of Obstetricians and Gynecologist. His memberships included the Academy of Medicine, the Warren County Medical Society, and the Medical Society of the State of New York.

Joseph A. Valvo, MD, Pinehurst, NC. Died March 13, 1992; age 74. Dr Valvo was a 1944 graduate of the University of Buffalo School of Medicine. His memberships included the Academy of Medicine, the Oneida County Medical Society, and the Medical Society of the State of New York.

Paul M. Walczak, MD, Buffalo. Died March 3, 1992; age 68. Dr Walczak was a 1946 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Surgery. He was a Fellow of the American College of Gastroenterology and the American College of Surgeons. His memberships included the American Geriatrics Society, the New York State Surgical Society, the Erie County Medical Society, and the Medical Society of the State of New York.

Herman Herbert Willner, MD, Montclaire, New Jersey. Died January 8, 1992; age 82. Dr Willner was a 1935 graduate of Medizinische Fakultat Der Universitat, Wien. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the Association for Advancement of Psychotherapy, the Association for Research in Nervous and Mental Disease, the American Academy of Neurology, the American Psychiatric Association, the Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Stuart A. Winning, MD, Canton. Died March 12, 1992; age 93. Dr Winning was a 1926 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Fellow of the American College of Surgeons. His memberships included the Academy of Medicine, the St. Lawrence County Medical Society, and the Medical Society of the State of New York.

Anthony J. Pisani, MD
1911–1992

Dr Anthony J. Pisani, a former Chief of Fracture and Orthopedic Surgery at St Vincent's Hospital and Medical Center in New York City, passed away on January 24, 1992. Dr Pisani was a 1931 graduate of Fordham University and received his MD degree from New York University School of Medicine in 1935. He did a two-year surgical internship at St Vincent's, and a two-year residency in Orthopedic Surgery at Bellevue Hospital. During World War II, he served for four years as Chief of Orthopedic Surgery at several US Army hospitals. Returning to St. Vincent's, Dr Pisani specialized in surgery of the hip, knee, ankles, and shoulders. He also had a special interest in pediatric orthopedics and was consultant and Chief of Orthopedic Surgery at the New York State Hospital for Crippled Children, now the Helen Hayes Hospital in Haverstraw, New York.

Dr Pisani was a leading pioneer in sports medicine. He was the team physician for the Brooklyn Dodgers from 1946 to 1951 and served as the official team surgeon for the New York Giants football team for 18 years. He was also Clinical Professor of Orthopedic Surgery at New York University School of Medicine. Over the years, his patients included not only the famous, but also the poor. He served as physician to New York State governors Alfred E. Smith, Malcolm Wilson, and Hugh Carey.

After his retirement in 1983, Dr Pisani continued to be active in many areas of medicine. Over the years, he was of great help to the editorial staff of the *Journal* as a peer reviewer and in providing much valuable advice. He was a gentle, kind, and thoughtful man who was always ready to encourage young physicians in their publication efforts. We at the *Journal* were always touched by his concern for these young authors and by his desire to see them succeed. We will miss his wise counsel.

Dr Pisani was married in 1944 to Angela McElligot, a graduate of St Vincent's Hospital School of Nursing. She died in 1985. He is survived by his wife, Eugenia Wise Pisani, seven children, thirteen grandchildren, and two brothers, Dr Bernard J. Pisani, who served as President of the Medical Society of the State of New York from 1983–84 and Dr Joseph M. Pisani. A sister, Dr Josephine M. Brown, died in 1987. He was a member of the American Medical Association, the Medical Society of the State of New York, New York County Medical Society, and the Society of Alumni of Bellevue Hospital.

He was a Fellow of the New York Academy of Medicine, the American College of Surgeons, and the American Academy of Orthopedic Surgeons.

Pascal James Imperato, MD
Editor

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1992; 92:122–125). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

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An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

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REFERENCES

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Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Samples references:

1. Bowen J: Autoimmune paraneoplastic diseases of the nervous system. *NY State J Med* 1991; 91:2–3.
2. Cotran RS, Kumar V, Robbins SL: *Robbins Pathologic Basis of Disease,* ed 4. Philadelphia, WB Saunders Co, 1989, pp 842–845.

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All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

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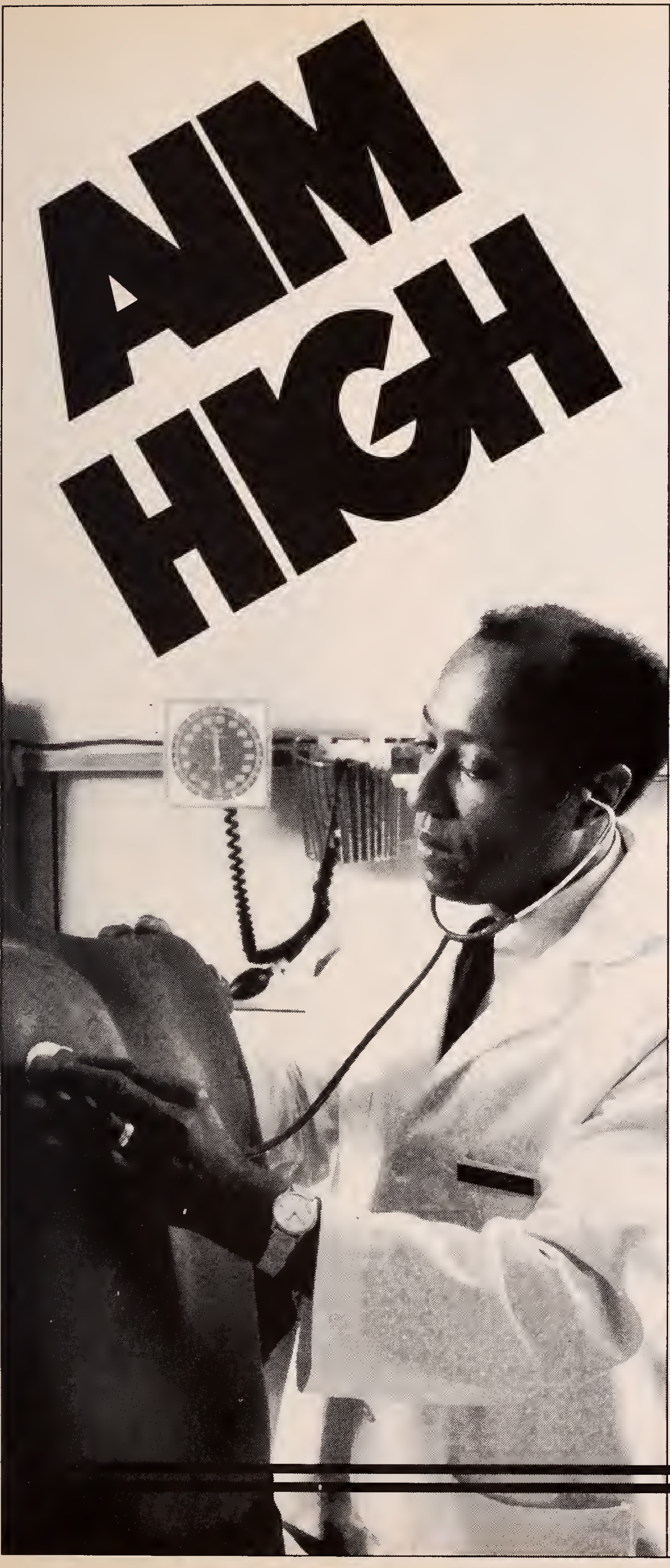

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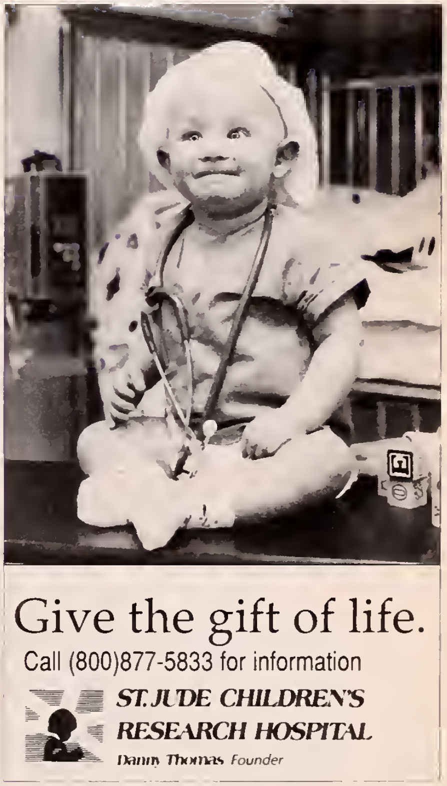

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(*Continued from p 6A*)

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Jul 24–28. **Thirteenth International Congress on Child and Adolescent Psychiatry.** Fairmont Hotel. Contact: University of California, Extended Programs in Medical Education, Room LS-105, San Francisco, CA 94143. Tel: (415) 476-4251.

FLORIDA

Jul 31–Aug 2. **Cape Cod Conference on Pediatrics.** Tara Hyannis Hotel and Resort, Cape Cod. Contact: Paula Kehrli, Program Coordinator, Nemours Children's Clinic, PO Box 5485, Jacksonville, FL 32247. Tel: (904) 390-3638.

NEW MEXICO

Jul 13–17. **Imaging 1992: Ultrasound, CT, MR, and Mammography.** Eldorado Hotel, Santa Fe. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773

SOUTH CAROLINA

Jul 30–Aug 2. **Adolescent Medicine.** Hilton Head Island Hilton Resort. Contact: Debbie Shealy, South Carolina Chapter of the American Academy of Pediatrics, PO Box 11188, Columbia, SC 29211. Tel: (803) 798-6207.

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Jul 8. **Update for General Practice: Neurological Disorders.** Sheraton Hotel & Conference Center, Burlington. Contact: Mary Lynn Duranleau, Course Coordinator, University of Vermont, Department of Surgery, Burlington, VT 05405. Tel: (802) 656-2563. ☐

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†Constipation, which is easily managed in most patients, is the most commonly reported side effect of Calan SR.

‡Verapamil should be administered cautiously to patients with impaired renal function.

References: 1. Data on file, Searle. 2. Edmonds D, Würth JP, Baumgart P, et al. Twenty-four-hour monitoring of blood pressure during calcium antagonist therapy. In: Fleckenstein A, Laragh SH, eds. *Hypertension—the Next Decade: Verapamil in Focus*. New York, NY: Churchill Livingstone; 1987:94-100. 3. Midtbø KA. Effects of long-term verapamil therapy on serum lipids and other metabolic parameters. *Am J Cardiol*. 1990;66:13I-15I. 4. Fagher B, Henningsen N, Hulthén L, et al. Antihypertensive and renal effects of enalapril and slow-release verapamil in essential hypertension. *Eur J Clin Pharmacol*. 1990;39(suppl 1):S41-S43. 5. Schmieder RE, Messerli FH, Garavaglia GE, et al. Cardiovascular effects of verapamil in patients with essential hypertension. *Circulation*. 1987;75:1030-1036. 6. Midtbø K, Lauve O, Hals O. No metabolic side effects of long-term treatment with verapamil in hypertension. *Angiology*. 1988;39:1025-1029.

BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol and propranolol clearance may occur when either drug is administered concomitantly with verapamil. A variable effect has been seen with combined use of atenolol. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digitoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration. Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels or increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Verapamil may inhibit the clearance and increase the plasma levels of theophylline. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. There was no evidence of a carcinogenic potential of verapamil administered to rats for 2 years. A study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°, 2°, 3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes, reversible non-obstructive paralytic ileus. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rash, exanthema, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, galactorrhea/hyperprolactinemia, increased urination, spotty menstruation, impotence.

4/11/91 • P91CA6277V

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New York State Journal of Medicine

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VOL 92
NO 7
JULY
1992

THE CHALLENGE OF CONTROLLING TUBERCULOSIS IN NYC

TUBERCULOSIS IN NY: RAPID RISE OF A PREVENTABLE DISEASE

AN UPDATE ON THE *NEW YORK STATE JOURNAL OF MEDICINE*

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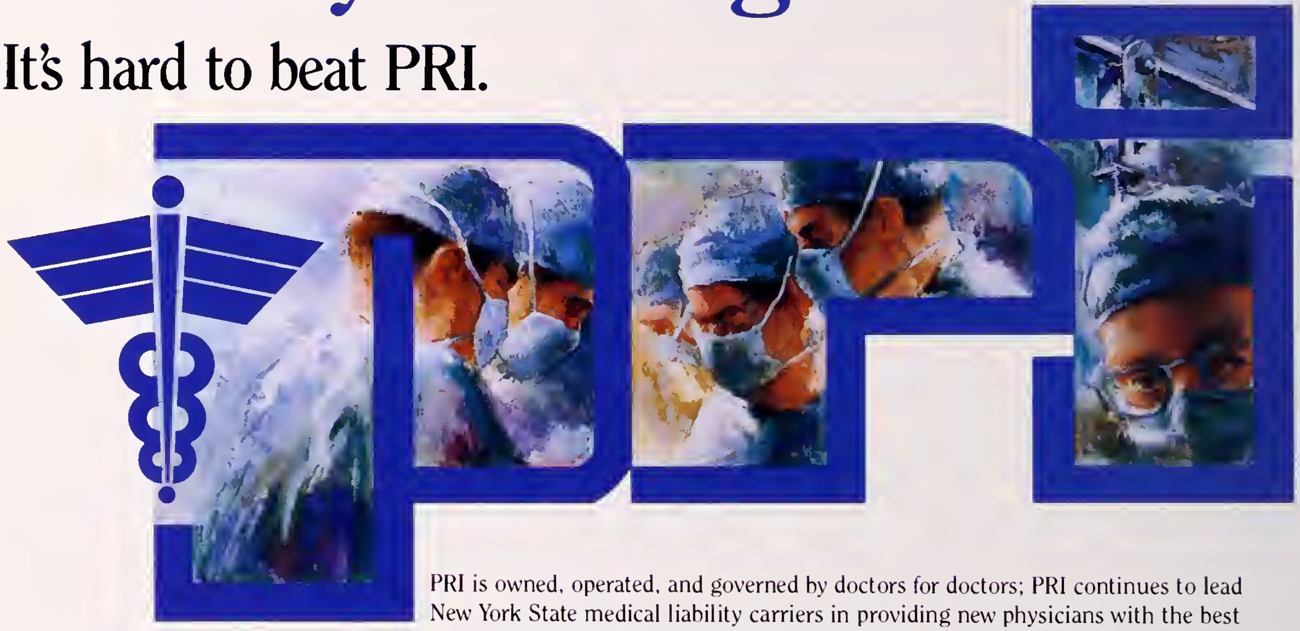

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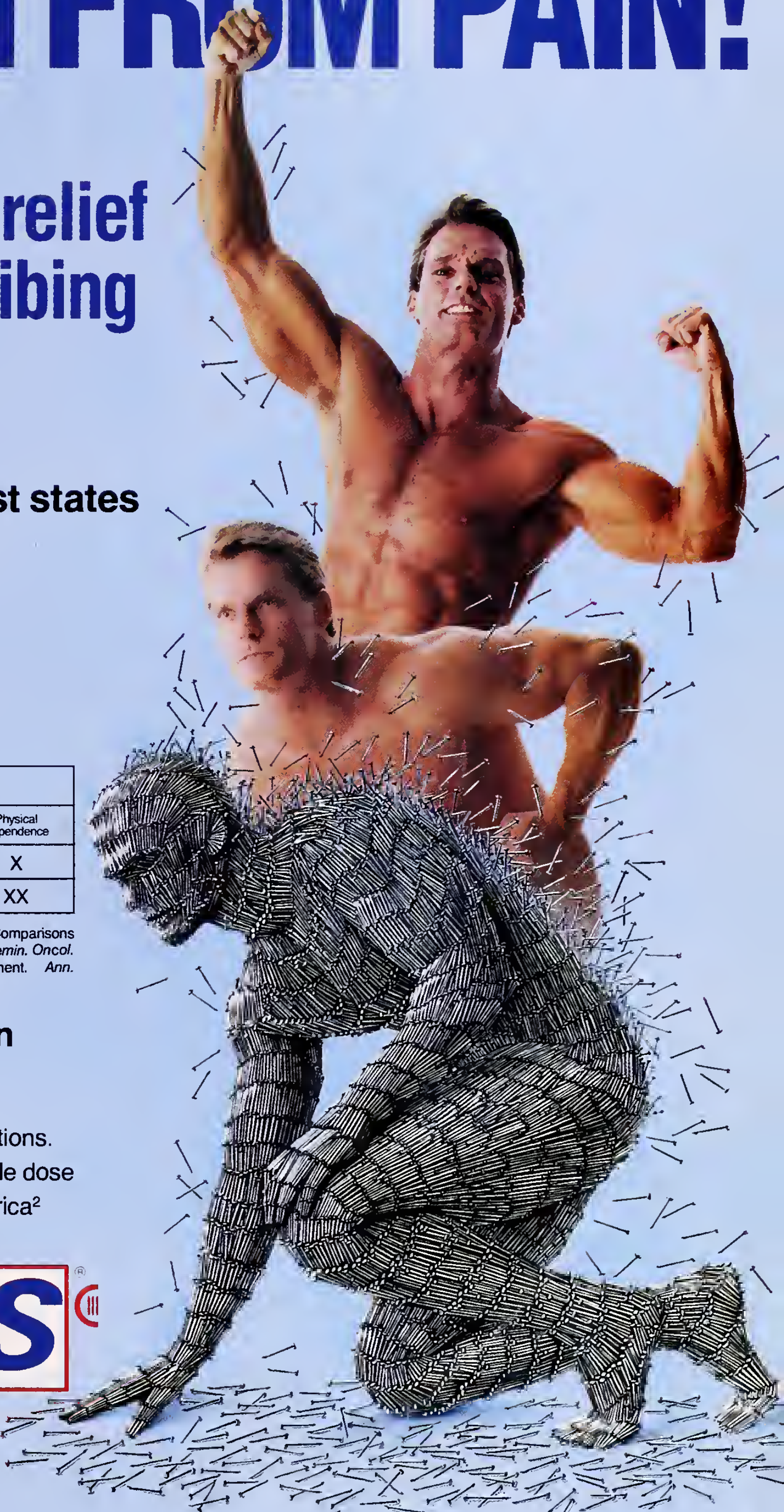

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Furthermore, narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries. **Acute Abdominal Conditions**: The administration of narcotics may obscure the diagnosis or clinical course of patients with acute abdominal conditions. **PRECAUTIONS**: **Special Risk Patients**: VICODIN/VICODIN ES Tablets should be used with caution in elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, prostatic hypertrophy or urethral stricture. **Cough Reflex**: Hydrocodone suppresses the cough reflex; as with all narcotics, caution should be exercised when VICODIN/VICODIN ES Tablets are used postoperatively and in patients with pulmonary disease. **Drug Interactions**: Patients receiving other narcotic analgesics, antipsychotics, antianxiety agents, or other CNS depressants (including alcohol) concomitantly with VICODIN/VICODIN ES Tablets may exhibit an additive CNS depression. The use of MAO inhibitors or tricyclic antidepressants with hydrocodone preparations may increase the effect of either the antidepressant or hydrocodone. The concurrent use of anticholinergics with hydrocodone may produce paralytic ileus. **Usage in Pregnancy**: **Teratogenic Effects**: Pregnancy Category C. Hydrocodone has been shown to be teratogenic in hamsters when given in doses 700 times the human dose. There are no adequate and well-controlled studies in pregnant women. VICODIN/VICODIN ES Tablets should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. **Nonteratogenic effects**: Babies born to mothers who have been taking opioids regularly prior to delivery will be physically dependent. The withdrawal signs include irritability and excessive crying, tremors, hyperactive reflexes, increased respiratory rate, increased stools, sneezing, yawning, vomiting, and fever. **Labor and Delivery**: Administration of VICODIN/VICODIN ES Tablets to the mother shortly before delivery may result in some degree of respiratory depression in the newborn, especially if higher doses are used. **Nursing Mothers**: It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from VICODIN/VICODIN ES Tablets, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. **Pediatric Use**: Safety and effectiveness in children have not been established. **ADVERSE REACTIONS**: The most frequently observed adverse reactions include light-headedness, dizziness, sedation, nausea and vomiting These effects seem to be more prominent in ambulatory than in nonambulatory patients and some of these adverse reactions may be alleviated if the patient lies down. Other adverse reactions include: **Central Nervous System**: Drowsiness, mental clouding, lethargy, impairment of mental and physical performance, anxiety, fear, dysphoria, psychic dependence and mood changes. **Gastrointestinal System**: The antiemetic phenothiazines are useful in suppressing the nausea and vomiting which may occur (see above); however, some phenothiazine derivatives seem to be antianalgesic and to increase the amount of narcotic required to produce pain relief, while other phenothiazines reduce the amount of narcotic required to produce a given level of analgesia Prolonged administration of VICODIN/VICODIN ES Tablets may produce constipation. **Genitourinary System**: Ureteral spasm, spasm of vesical sphincters and urinary retention have been reported **Respiratory Depression**: Hydrocodone bitartrate may produce dose-related respiratory depression by acting directly on the brain stem respiratory center. Hydrocodone also affects the center that controls respiratory rhythm, and may produce irregular and periodic breathing If significant respiratory depression occurs, it may be antagonized by the use of naloxone hydrochloride. Apply other supportive measures when indicated. **DRUG ABUSE AND DEPENDENCE**: VICODIN/VICODIN ES Tablets are subject to the Federal Controlled Substance Act (Schedule III). Psychic dependence, physical dependence, and tolerance may develop upon repeated administration of narcotics; therefore, VICODIN/VICODIN ES Tablets should be prescribed and administered with caution. **OVERDOSAGE**: **Acetaminophen Signs and Symptoms**: In acute acetaminophen overdosage, dose-dependent, potentially fatal hepatic necrosis is the most serious adverse effect. Renal tubular necrosis, hypoglycemic coma, and thrombocytopenia may also occur Early symptoms following a potentially hepatotoxic overdose may include: nausea, vomiting, diaphoresis and general malaise. Clinical and laboratory evidence of hepatic toxicity may not be apparent until 48 to 72 hours post-ingestion. **Hydrocodone Signs and Symptoms**: Serious overdose with hydrocodone is characterized by respiratory depression (a decrease in respiratory rate and/or tidal volume, Cheyne-Stokes respiration, (cyanosis), extreme somnolence progressing to stupor or coma, skeletal muscle flaccidity, cold and clammy skin, and sometimes bradycardia and hypotension. In severe overdosage, apnea, circulatory collapse, cardiac arrest and death may occur

Revised March 1992 5890

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POSTAGE. Second class postage is paid at New Hyde Park, New York, and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, PO Box 5404, Lake Success, New York 11042.

New York State Journal of Medicine

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

JULY 1992 / Volume 92, Number 7

Contents

COMMENTARIES

291 **The challenge of controlling tuberculosis in New York City**
MARGARET A. HAMBURG, MD

293 **Tuberculosis in New York: Rapid rise of a preventable disease**
LLOYD F. NOVICK, MD, MPH

294 **An update on the *New York State Journal of Medicine***
MILTON GORDON, MD

RESEARCH PAPER

297 **Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS**
VIJAY CHECHANI, MD; AZMY A. ALLAM, MD; PETER R. SMITH, MD; CAROLYN A. WEBBER, MD; STEPHAN L. KAMHOLZ, MD

CLINICAL CONFERENCE

301 **Münchausen syndrome**

SPECIAL ARTICLES

306 **Healing waters**
JACQUELINE GALLOWAY

307 **Memories of an internship in 1943**
LEO J. KOVEN, MD

CASE REPORTS

313 **Purpura fulminans, metastatic endophthalmitis, and thrombotic thrombocytopenic purpura in an HIV-infected patient**
URMILA SHIVARAM, MD, MARY CASH, PA

314 **Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient**
DAVID A. SHERRIS, MD; PAUL C. HARRINGTON, MD; JOHN D. NORANTE, MD

(continued on p 6A)

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

AUGUST 1992

AROUND THE STATE

COOPERSTOWN

Aug 19. **Sexually Transmitted Diseases.** 3 Cat I Credits. Bassett Hall Conference Center. *Aug 21–23.* **Fifth Annual Pediatric Refresher Course.** The Otesaga Hotel, Cooperstown. Contact: Charlotte L. Hoag, Medical Education Coordinator, Department of Medical Education, The Mary Imogene Bassett Hospital, One Atwell Road, Cooperstown, NY 13326. Tel: (607) 547-3820.

MANHATTAN

Aug 31–Sept 4. **Radiation, Physics, and Biology.** NYU Medical Center. 34 Cat I credits. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295.

AROUND THE NATION

HAWAII

Aug 9–15. **12th Annual Fine Needle Biopsy Course.** Ritz-Carlton Hotel, Mauna Lani. Contact: Extended Programs in Medical Education, Room LS-105, University of California, San Francisco, CA 94143. Tel: (415) 476-4251.

AROUND THE WORLD

BRAZIL

Aug 11–16. **Second Pan American Congress on Vascular Surgery.** Sheraton Rio Hotel. 21 Cat I credits. Contact: Maureen Hanagan, Course Administrator, Maimonides Medical Center, 4802 Tenth Ave, Brooklyn, NY 11219. Tel: (718) 283-7957.

SPAIN

Aug 17–21. **Barcelona 1992 Post-Games Imaging Conference.** Hotel Arts a Ritz-Carlton Hotel, Barcelona. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773. ☐

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Yohimbine exerts a stimulating action on the mood and may increase anxiety. Such actions have not been adequately studied or related to dosage although they appear to require high doses of the drug. Yohimbine has a mild anti-diuretic action, probably via stimulation of hypothalmic centers and release of posterior pituitary hormone.

Reportedly, Yohimbine exerts no significant influence on cardiac stimulation and other effects mediated by B-adrenergic receptors, its effect on blood pressure, if any, would be to lower it; however no adequate studies are at hand to quantitate this effect in terms of Yohimbine dosage.

Indications: Yocon® is indicated as a sympathicolytic and mydriatric. It may have activity as an aphrodisiac.

Contraindications: Renal diseases, and patient's sensitive to the drug. In view of the limited and inadequate information at hand, no precise tabulation can be offered of additional contraindications.

Warning: Generally, this drug is not proposed for use in females and certainly must not be used during pregnancy. Neither is this drug proposed for use in pediatric, geriatric or cardio-renal patients with gastric or duodenal ulcer history. Nor should it be used in conjunction with mood-modifying drugs such as antidepressants, or in psychiatric patients in general.

Adverse Reactions: Yohimbine readily penetrates the (CNS) and produces a complex pattern of responses in lower doses than required to produce peripheral a-adrenergic blockade. These include, anti-diuresis, a general picture of central excitation including elevation of blood pressure and heart rate, increased motor activity, irritability and tremor. Sweating, nausea and vomiting are common after parenteral administration of the drug.[1,2] Also dizziness, headache, skin flushing reported when used orally.[1,3]

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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New York State
Journal of Medicine

316 **Ten-year survival in multiple myeloma: Report of two cases and review of the literature**
FRED ROSNER, MD; HANS GRÜNWALD, MD; ANDREA C. KALMAN, RPAC

318 **Right ventricular cardiomyopathy**
STUART LANDAU, MD; HOWARD L. SACHER, DO; MICHAEL L. SACHER, DO; KAREN WRIGHT, BS, RN; ALLEN CHIEN, MS; KATHLEEN A. DIETRICH, MA, DO

LETTERS TO THE EDITOR

322 **Trauma-related presentation of non-Hodgkin's lymphoma**
JAY K. PATEL, MD; MINAL PATEL, MD; IBRAHAM HITTI, MD; MARSHA CROFFORD, MD; JAE-MUN NOH, MD; PARTHAS NARASIMHAN, MD

322 **Chemotherapy for hepatic angiosarcoma**
ETTA B. FRANKEL, MD

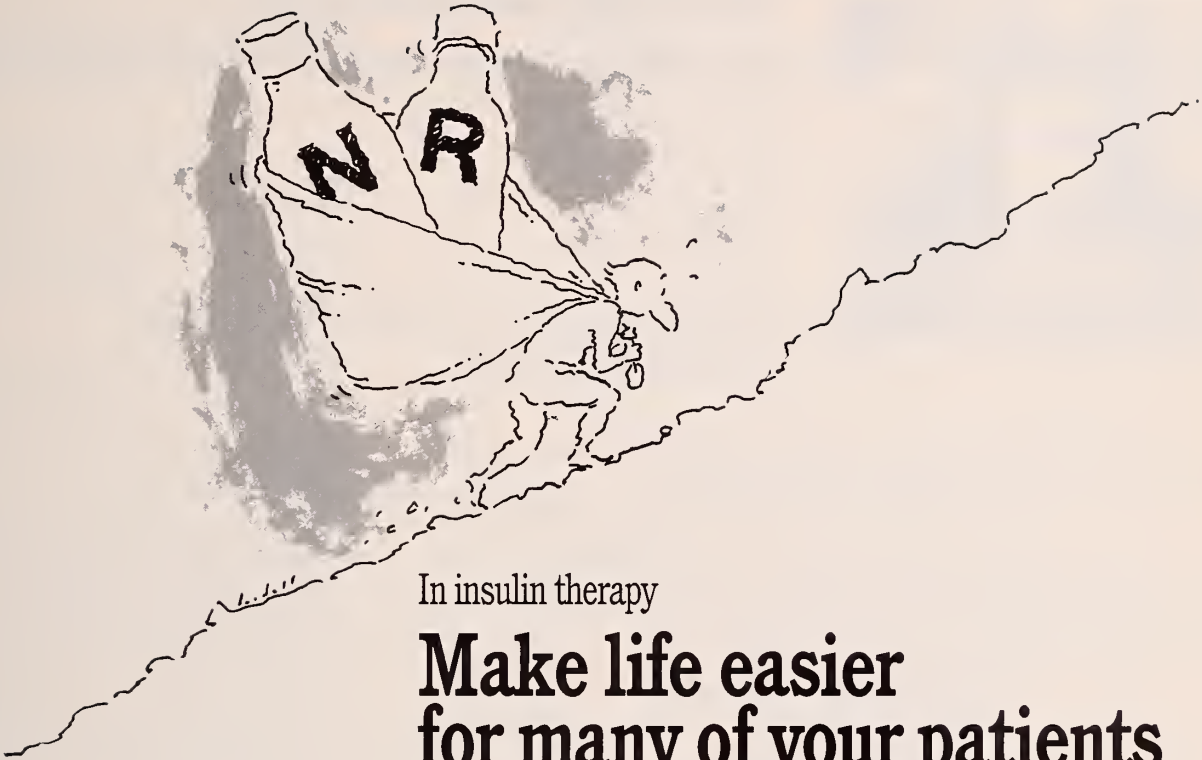
323 **New hospice benefit available for nursing home residents**
JANET E. GARGIULO, MD

323 **The role of third party payors in medical care**
JOHN D. KRAFCHUK

325 BOOK REVIEWS

328 OUR REFEREES

5A MEDICAL MEETINGS AND LECTURES



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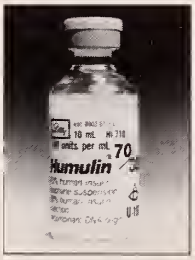

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COMMENTARIES

The challenge of controlling tuberculosis in New York City

Tuberculosis is a curable and preventable disease. For more than 30 years, our medical discipline has had both the knowledge and the tools requisite to those tasks. Had tuberculosis eradication in New York City depended solely on our medical understanding of tuberculosis transmission and treatment, we could have been well on the way to ending the tuberculosis epidemic we now face.

Sadly, though, the spread of tuberculosis in New York City is intimately intertwined with a number of complex social and economic issues. For years, we have witnessed the strain and neglect of our social fabric; a fabric now fraying because of the weight of companion epidemics of poverty, unemployment, family disarray, substandard housing, homelessness, drug abuse, and human immunodeficiency virus (HIV) illness. All of these factors exacerbate the problem of tuberculosis. At the same time, budget pressures and the lack of serious and ongoing commitment to maintaining basic public health services has eroded efforts to control tuberculosis effectively.

In 1984, the Director of the Federal Centers for Disease Control, Dr James O. Mason, challenged the public health community to develop a plan to eliminate tuberculosis in the United States.[1] Ironically just one year later, in 1985, the number of tuberculosis cases began to increase across the nation after decades of steady decline. In New York City, the rise had already begun in 1979, and the City continues to be at the forefront of the nationwide resurgence of the disease. Clearly, the eradication of tuberculosis in New York City—and across the nation—is entirely possible. However, if we are to succeed in our efforts, we must redouble our efforts to define and implement a meaningful strategic plan *and* we must commit the necessary human and fiscal resources.

Later in this commentary, I will outline the major components of the plan necessary for tuberculosis control in New York City. First, though, I want to consider briefly some issues concerning the tuberculosis epidemic in New York City—past and present—to inform effective measures for the future.

Epidemiological Observations

In the past decade, the incidence of tuberculosis in New York City has increased some 132%, and the 1990 case rate, 49.8 cases per 100,000 individuals, was the highest in two decades and more than five times the nation's case rate. The city currently accounts for 15% of the nation's tuberculosis caseload, and although it does not have the highest per capita rate of tuberculosis (it is exceeded by Atlanta and Newark), the largest absolute number of tuberculosis patients in any urban center live in our five boroughs.

In his companion commentary, Dr Lloyd Novick[2] correctly notes that, during the three decades that followed the introduction of effective anti-tuberculosis medication just before 1950, the number of tuberculosis cases in New York State, and indeed across the country, began to wane. So, too, did tuberculosis incidence begin to diminish citywide. However, tuberculosis continued in New York City's poorest (and most medically underserved) neighborhoods. When tuberculosis was at its nadir in New York in 1978, the disease continued to occur in communities like Central Harlem at rates several times the city and national average.

Given the rise in poverty and the decline in social conditions, it is no surprise that disenfranchised neighborhoods continue to contribute disproportionately to the incidence of tuberculosis. In 1990, for example, Central Harlem reported more than 233 cases per 100,000 population, a rate more than four times the citywide average and more than twenty times the national average. In addition, because these areas of concentrated inner-city poverty are overwhelmingly communities of color, the burden of illness on certain racial and ethnic groups is dramatic. New cases are occurring 13 times more frequently among African-Americans than among whites, and among men of color, ages 25–44, (the hardest hit group in the city) at a rate 20 times that of white men of the same age.

In the decade of the 1980s, another factor, HIV/AIDS, began to fuel the city's tuberculosis epidemic. With close to 40,000 AIDS cases reported to date, accounting for some 18% of the nation's caseload, HIV illness has taken a significant toll in New York City. We know that the compromised immune function that characterizes HIV disease allows the tuberculosis organism to flourish. In the setting of increasing prevalence of HIV infection and AIDS, tuberculosis has grown as a collateral, devastating public health problem. Yet, it is clear that the HIV/AIDS epidemic does not wholly account for the rising rates of tuberculosis. Indeed, by the end of the 1970s, tuberculosis was already resurgent in New York City. Although we did not then recognize the nascent HIV infection in our midst, the number of individuals whose immune function was sufficiently compromised to propel rates of tuberculosis disease was probably relatively small.

So what other specific factors contributed to the resurgence of tuberculosis in New York City? At the end of the 1960s, with decreasing case numbers and the availability of oral antituberculosis medications, New York City Mayor John Lindsay justifiably recommended a ten-year phase-out of dedicated tuberculosis hospital beds (approximately 1,000 of them) and the transfer of their fiscal support to expanded community-based tuberculosis outreach and control programs, including drug treatment programs, and for long-term residential care of homeless individuals with tuberculosis. In the mid-1970s, however, we witnessed a now all-too-familiar event—a fiscal crisis in New York City. That crisis resulted in a significant dwindling of promised resources, including those from the State and, during the final years of the decade, from federal sources as well.

Appropriate treatment programs, like case management and supervised therapy (the forerunner of directly observed therapy) were known to work well with high treatment completion rates. However, these were never sufficiently supported to expand beyond pilot-project staffing and become a useful tool in the City's tuberculosis control efforts.

That we, in medicine and public health, stood by and allowed critical resources to disappear from necessary tuberculosis programs is an alarming historical lesson. As Brudney and Dobkin[3] have implied, the mounting absence of patient-sensitive follow-up contributes to the escalation of treatment non-compliance and treatment failure.

In addition, it is specifically treatment non-compliance and failed therapies that have fueled the emergence of multiple-drug resistant tuberculosis. In a recent study documenting a one month sample in New York City, Frieden et al[4] identified nearly half of patients previously treated as resistant to one or more drugs and nearly one-third resistant to isoniazid (INH) and Rifampin. Among those patients with first-time tuberculosis, nearly one-quarter were resistant to one or more drugs and about 7% were resistant to INH and Rifampin. Once INH and Rifampin become lost to the front-line of tuberculosis control, clinicians face fewer options, more complex and expensive treatment regimens and, worse, the specter of untreatable tuberculosis.

TUBERCULOSIS CONTROL IN NEW YORK CITY: AN OUTLINE FOR ACTION

In the face of the current tuberculosis epidemic, we need to develop a cogent citywide plan to bring the disease under control and to foster its elimination. This plan must include the following components:

- We must assure that tuberculosis cases are treated until cured. To achieve that end, we must put in place a sufficient cadre of trained, motivated field workers who will administer directly observed therapy to tuberculosis patients. Moreover, the staff must be individuals likely to reach and be accepted by the patient population, indicating a clear need for effective targeted recruitment and retention, and liaison with community-based organizations, especially in affected communities of color.
- We must upgrade and expand clinical opportunities for treatment of tuberculosis disease.
- We must increase the resources devoted to positive incentive programs (rewards) that will maximize patient participation in treatment.
- We must develop a range of residential facilities for people with tuberculosis who no longer need acute hospitalization, but who do need considerable housing, social service, and/or medical support to complete their treatments.
- We must develop an appropriate legal means of assuring completion of treatment for those patients who are unable to complete treatment or who will not voluntarily (or repeatedly do not) comply with it.
- We must stop the institutional spread of tuberculosis. This will require aggressive (and, unfortunately, expensive) infection control measures in hospitals, shelters, and the correctional system. We must ensure appropriate safeguards to reduce the risk of transmission to workers and to other patients or residents of the facilities.
- We must ensure that laboratory facilities develop state-of-the-art capacities for culturing tuberculosis organisms, determining sensitivities, and reporting these rapidly to the medical community.
- We must improve screening, case finding, and preventive therapy to high-risk populations, especially in HIV-prevention and care programs, in communities where there may be a large number of recent PPD converters, and in non-traditional settings, like drug treatment centers.
- We must enhance public health education, including reducing concerns among the "worried well." We must develop information and materials that are community-sensitive and constituent-relevant, and we must understand the kinds of messages that are most likely to induce behavioral changes when required.
- We must encourage research and development in the tuberculosis arena. For example, government and industry must foster both basic and applied clinical research aimed at the development of antituberculosis drugs (no new ones have been marketed in the past 20 years), at better delivery methods suited to long-term treatment regimens

(sustained release or depot delivery), and at rapid diagnostic approaches.

The beginnings of many programs that are components of this outline are already in place or will begin their development next year as a consequence of the city's infusion of close to $100 million dollars in new needs support. However, clearly, to achieve these goals additional fiscal resources, from both the public and private sectors will be necessary, and the support must be considerable. Sadly, had sufficient finances been devoted to tuberculosis control in past years, the need would not be as great now.

This, then, remains the critical lesson for medicine and public health: We must never again tolerate crippling compromises as we mount an intensive campaign to bring tuberculosis, or any other high-risk disease, under control. The war against tuberculosis can be won, and we will win it. It will be costly, and it will take time.

The challenge although daunting, also offers opportunity. For the control of tuberculosis in New York City is probably the most complex such effort in any urban center in the country. Indeed, our potential successes can serve as a model for others elsewhere. As a slight twist in our anthem conveys: "If we can treat it here, we can treat it anywhere." However, it is up to us, New York.

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1. Centers for Disease Control, A Strategic Plan for the Elimination of Tuberculosis in the United States, *MMWR* 1989; 38:1.
2. Novick LF: Tuberculosis in New York: Rapid rise of a preventable disease. *NY State J Med* 1992; 92:293–294.
3. Brudney K, Dobkin J: Resurgent tuberculosis in New York City: Human immunodeficiency virus, homelessness, and the decline of tuberculosis programs. *Am Rev Respir Dis* 1991; 144:745–749.
4. Frieden T, Sterling T, Pablo-Mendez A, et al: Multiple drug resistant tuberculosis in New York City. *N Engl J Med* (submitted).

Tuberculosis in New York: Rapid rise of a preventable disease

After decades of decline, tuberculosis once again poses an unchecked threat to public health in New York State. During the 1980s the incidence of reported cases more than doubled, increasing 30% from 1989 to 1990 alone. Of the more than 4,000 patients identified statewide in 1990, the vast majority (84%) resided in New York City, where the case rate was 48.9 per 100,000 population—the highest rate in two decades and five times the rate for the nation as a whole.

This epidemic should not have occurred. Highly successful strategies to prevent the spread of tuberculosis have existed for more than 30 years. Between 1950 and 1978, public health measures and the advent of efficacious antituberculosis drugs reduced the number of cases in New York State from 12,493 to 2,060. Then the climb began. Why are we now faced with a major epidemic of this easily preventable disease?

CAUSES OF THE EPIDEMIC

New Populations. The most direct cause of the tuberculosis epidemic has been an increase in populations at high risk for communicable diseases. Their heightened susceptibility is often associated with human immunodeficiency virus (HIV) infection and homelessness. These populations tend to live in crowded communities, along with immigrants from countries where tuberculosis is endemic. One reflection of the shift in patient populations from earlier epidemics is that active disease is now found predominantly among members of minorities aged 35 to 44 years old, rather than among the elderly.

New Priorities. This expansion of susceptible populations coincided with a weakening of the public health activities that had succeeded in controlling tuberculosis: contact investigation, case management, tracking of patients with active disease, and follow-up to ensure full compliance with treatment. Reichman has described this slacking of resources as "the U-shaped curve of concern," in which resources are shifted from a temporarily controlled disease to new public health priorities, resulting in a resurgence of the no-longer-controlled disease.[1]

In the case of tuberculosis, appropriations for Federal Project Grants for Tuberculosis Control were cut from $20 million in 1969 to $5 million in 1983. State and New York City funding for tuberculosis prevention was also reduced during this period.

New Pathogens. The rapid rise in the number of tuberculosis patients noncompliant with therapy has led to the emergence of multiple-drug-resistant (MDR) strains of the bacillus, a situation of the utmost public health concern. Within the past two years at least six major nosocomial outbreaks of MDR tuberculosis (MDR-TB) have occurred in hospitals in New York State, including two outbreaks at upstate facilities. MDR-TB has been responsible for the deaths of several prisoners at an upstate correctional institution. Health care workers at the referral hospital for this prison have experienced PPD conversions, indicating primary infections with tuberculosis.

A review of all positive tuberculosis cultures in New York City in April 1991 revealed that more than one-third of the patients were infected with strains resistant to one or more drugs and that nearly one in five patients were resistant to both isoniazid and rifampin. Because of these findings the New York City Department of Health now recommends that every tuberculosis patient in the city who has not been previously treated for tuberculosis receive at least four antituberculosis drugs.

The re-emergence of tuberculosis at epidemic rates, the pervasive noncompliance with therapy, and the proliferation of MDR organisms raise difficult questions for health care facilities regarding the adequacy of isolation, ultraviolet light, and other protective measures against MDR-TB.

Challenge of Epidemic Control

What public health measures are needed to control this epidemic, which is now complicated by MDR and consequently prolonged periods of infectiousness?

Transmission of tuberculosis can only be prevented by ensuring full compliance with therapy by patients with active disease. A study by Brudney and Dobkin[2] of 224 patients at Harlem Hospital documents the extent of noncompliance in one community at risk. Of 178 patients discharged for tuberculosis treatment, 89% were lost to follow-up or failed to complete therapy. More than one-quarter had to be readmitted to the hospital with active tuberculosis, and most of these were again lost to follow-up.

Because tuberculosis infection and active disease are linked with other complex health and social problems, the causes and control measures for the epidemic are multifactorial. To succeed, the control strategy must be effective in communities at risk, where human services programs are struggling with limited success—communities marked by poverty, substance abuse, homelessness, and inadequate access to health services.

Many tuberculosis patients suffer coexisting diseases, including HIV infection and acquired immunodeficiency syndrome (AIDS) and have other unmet needs, including permanent shelter and treatment for alcohol or drug addiction. Treatment for these patients thus tend to involve multiple interventions and a need for close coordination among providers. Such coordination is often lacking but vital.

Measures to Control the Epidemic

Compliance with the full course of antituberculosis therapy, in which multiple medications are taken at least weekly for several months, is the key strategy to controlling the epidemic. To achieve the goal the New York State Department of Health, in collaboration with other state and New York City agencies and providers, is launching the nation's largest program of directly observed therapy (DOT). Approximately $7 million has been made available for outreach and enrollment of as many as 1,000 patients, who will take their medications under direct supervision in a variety of settings.

Hospitals across New York State must enhance their capability to care for patients with tuberculosis by constructing isolation facilities, adhering to infection control guidelines, and upgrading their laboratory services. The New York State Department of Health is addressing these issues systematically with the hospitals and has recently issued comprehensive guidelines on infection control.[3]

Ambulatory care centers for tuberculosis patients should be created, linked to hospitals that treat substantial numbers of inpatient tuberculosis cases. These Tuberculosis Centers of Excellence should have outreach, diagnostic, laboratory, treatment, and referral capabilities for comprehensive health care, including treatment for substance abuse and HIV infection. Designated AIDS centers, caring for large numbers of HIV-infected individuals susceptible to tuberculosis, should develop the same capabilities.

Along with DOT, ambulatory, and inpatient facilities, the continuum of measures to control this epidemic must include intermediate-care facilities and residential options. Homeless shelters are a major reservoir for tuberculosis in New York City, and screening and health care must be provided there. In addition to the shelter at Bellevue Hospital, now offering tuberculosis care, at least two new shelters, including one for women, are needed.

Jails and prisons have many inmates with active tuberculosis. Screening at the time of admission and assurance of treatment for all inmates with active disease are priorities for controlling the epidemic. Improvements are now being made. Both jails and prisons, for example, are developing isolation facilities for infectious prisoners.

The peril of tuberculosis is not restricted to high-risk populations or to communities at risk. The rising incidence of active disease, particularly of MDR-TB, is likely to result in more general transmission for years to come, unless decisive action is taken now. The immediate measures, focused specifically on tuberculosis control, must be combined with programs directed at such related public-health concerns as susceptibility to HIV infection, substance abuse, and homelessness. The full spectrum of programs will require substantial resources and innovative public-health strategies, but the challenge can be met. Control of the tuberculosis epidemic is feasible, and it must be done.

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1. Reichmann, LB: The U-shaped curve of concern. *Am Rev Respir Dis* 1991; 144:741–742.
2. Brudney K, Dobkin J: Resurgent tuberculosis in New York City: Human immunodeficiency virus, homelessness, and the decline of tuberculosis programs. *Am Rev Respir Dis* 1991; 144:745–749.
3. New York State Department of Health: Control of tuberculosis in hospitals. Department of Health memorandum 92-7: 13 March 1992.

An update on the *New York State Journal of Medicine*

The *New York State Journal of Medicine* is now in its 92nd year. It has a total circulation of 27,595, of which 25,505 copies are distributed in New York State and 1,929 in the rest of the country. Foreign subscriptions total 161. The *Journal* is a highly respected peer-reviewed scientific medical publication. This means that submissions to it are evaluated by outside medical and scientific experts as well as subjected to in-house editorial review.

The peer-review process is the cornerstone of a quality medical journal. It assures that the claims made by authors in their papers are substantiated by scientific evidence. It also assures that the information presented by authors is indeed new, unique, or else so insightful as to merit publication. The expert peer reviewers who examine articles for the *Journal* carefully assess claims of a first report or discovery and whether or not the cited medical literature is being accurately represented and quoted. Manuscripts submitted to the *Journal* are examined by two outside peer reviewers, who provide their services gratis. These reviewers, of which there were 150 in 1991, are drawn from around the country and from major academic medical centers such as the Mayo Clinic, the Massachusetts General Hospital and large medical centers in New York State. Some articles are also peer reviewed by members of the Associate Editorial Board in areas of their expertise. All research papers submitted to the *Journal* are additionally reviewed by an epidemiologist/biostatistician to evaluate the soundness of study design, the appropriateness of the statistical analyses performed, and the validity of the conclusions drawn.

The *Journal* adheres to the provisions of the Uniform Requirements for Manuscripts Submitted to Biomedical Journals developed by the International Committee of Medical Journal Editors. These requirements cover not only matters of style and format, but also such issues as prohibitions against the preliminary release of data to the press and media before publication. There are 400 medical journals in the world which adhere to these Uniform Requirements out of the 5,000 medical journals published. These 400 journals, however, include all the leading medical publications whose articles are frequently cited in the literature by other medical writers.[1]

Of the 5,000 medical journals published, only 2,400 are included in the Medline computer data base and the *Index Medicus* by the National Library of Medicine. Many of the remaining 2,600 journals are considered "throw aways" whose content is viewed as potentially unreliable or biased. In addition to annually indexing the content of 2,400 medical journals, the National Library of Medicine also rapidly indexes 112 journals on a monthly basis. The *Journal* is not only indexed annually, but is also among the select 112 (5% of journals) chosen for monthly indexing.

In 1986, a special study was conducted by the Institute for Scientific Information to determine which journals had the greatest inpact on medical science. This was assessed by the frequency with which a journal's articles were cited by medical researchers writing in other journals. The *Journal* was ranked 29th among the 2,400 journals indexed in the *Index Medicus*, placing it in the top 12% of the world's medical journals[2]. No other state medical society journal was included in the top 40 journals on the list. Another measure of the *Journal's* quality was its selection in 1992 for inclusion in a traveling exhibit of publications of excellence sponsored by the Library of Congress.

These external assessments clearly confirm that the *Journal* is highly respected and widely read. Yet, the Committee on Publications, Library, and Archives has always been keenly aware that the opinions of our members and readers are as important as the evaluations of national standard setting bodies. In late 1987, the committee authorized a comprehensive readership survey for the *Journal*. That survey of a random sample of 1,550 readers was conducted by Health Industries Research, a Connecticut-based company recommended by the society's executive vice-president. The response rate to this readership survey was 47.5% Of those who responded, 78%, a clear majority, rated the *Journal* as good or excellent.[3]

External measures and the results of a high-response rate readership survey indicate that the *Journal* is a high quality medical scientific publication that has gained international respect.

In recent years, the costs of producing the *Journal* have been greatly reduced. In 1982, $721,329 were spent on the *Journal*. This figure fell to $587,616 (1988), $573,964 (1989), $549,532 (1990) and $511,854 (1991). Thus, over a period of nine years, the *Journal's* expenditures fell by $209,475, a time when production costs were rising by some 3–4% per year. The reductions in production costs, that is the monies spent for printing and mailing, have been dramatic in recent years. These costs for 1991 ($284,000) were $12,000 less than those for 1990 ($296,000), $43,000 less than those for 1989 ($327,000), and $60,000 less than those for 1988 ($344,000). These savings were achieved in part by text transfer, that is, a system by which the *Journal* is typeset and made up in-house on computers. At the present time, 100% of the *Journal's* content is produced in this manner compared to 60% of content in 1990. However, many of these savings were also achieved by reducing the size of *Journal* issues. These reductions in size were achieved by limiting the length of articles, by using smaller sizes of type to get more type on each page and by reducing the number of pages in each issue of the *Journal*.

These economies have meant that the *Journal* editor and the Associate Editorial Board have had to reject more articles than was the case in the past since the annual number of submissions has remained the same at several hundred. The annual rejection rate for articles has been between 60% and 70% in recent years.

The Committee supported these economies in view of the society's difficult budgetary position in recent years. We consistently and dramatically cut expenses each year in a responsible manner. However, we believe that the *Journal* is now functioning at a bare minimum of budgetary support with a minimum staff of three full time people and one half-time person.

The *Journal's* budget also covers the operation of the library. In 1991, the library handled 684 bibliographic and reference searches for society members and 1,970 telephone calls from members. A total of 1,718 items were requested on interlibrary loan and 3,392 books and journals were circulated. In addition, the library handled some 20–30 calls a day from the general public over its Information Services line. This service was established to improve the society's communications with the public.

Each year, members of the society receive a dues renewal notice that states: "$30 of your dues are applied to a subscription to the *New York State Journal of Medicine*." This income plus advertising and other income produced a profit for the *Journal* of $175,991 in 1991, a point which several Society members emphasized at the recent House of Delegates meeting.

There has been a growing concern by the Committee and other society members over the consideration being given to either abolishing the *Journal* or else downgrading it to a "throw away." It was partly because of these concerns that two resolutions were introduced into the 1992 House of Delegates meeting requesting that the society provide the necessary administrative and financial support to continue publication of the *Journal* as a quality peer reviewed scientific journal. These resolutions were introduced by Nassau County (Resolution number 92-59) and by the Seventh District Branch (Number 92-85). The Reference Committee on Reports of Officers and Administrative matters recommended adoption of the resolution from the Seventh District Branch as it encompassed the intent of the resolution from Nassau County.

The Committee supported these resolutions and in a statement before the Reference Committee expressed its belief that a matter as important as the future status of the *Journal* should be decided by the House of Delegates. When the Reference Committee made its recommendation to approve adoption, an ammendment was added to the resolution of the Seventh District Branch requiring that any recommendation to discontinue or alter the peer-reviewed status of the *Journal* be submitted to the 1993 House of Delegates meeting for deliberation before any final decision is made.

It was clear from the statements made at the Reference Committee and at the House of Delegates meeting that there was strong and broad support for the *Journal*. For the average member who is not active on society committees, the *Journal* represents a very tangible benefit of membership. The *Journal* not only carries timely clinical and scientific information for readers, but also serves as a strong link to the medical schools and teaching hospitals in the state. The faculty and house staff of the state's thirteen medical schools regularly submit articles to the *Journal* for consideration and use the *Journal's* content for teaching purposes.

As was evident from comments made at the annual House of Delegates meeting, there are some in the society who do not believe that it should continue publishing a peer reviewed scientific and clinical journal. They are proposing that the *Journal* be replaced by a new "eye catching throw away" that focuses on political, regulatory, and governmental issues, topics now covered in other society publications. They are also proposing that this new "throw away" carry occasional scientific abstracts or articles. Although the Committee respects the right of these individuals to hold such an opinion, it strongly disagrees with it. We believe that the society possesses a very unique medical journal that is highly respected, widely read and cited, of great value to the membership, and which produces an annual profit from its advertising and other revenues. Discontinuance of such a *Journal* would be tragic, financially unwise given the large profit it makes, and not in the interests of either the society's members nor medicine at large. Replacing it with a "throw away" carrying non-peer reviewed materials or diluting it with "throw away" material will in the long run ensure that anyone with quality scientific and clinical articles or abstracts will submit them elsewhere. One has only to look at some journals that have gone this route to appreciate where the repeatedly rejected scientific and clinical papers eventually get published. Such articles and abstracts are rarely read, usually go unindexed and uncited, and serve only the interests of authors and publishers in need of filling pages. Both downgrading and dilution have significant negative financial consequences as many pharmaceutical advertisers refuse to place their ads in such journals.

The Committee on Publications, Library, and Archives along with the editorial staff has worked over the past several years to produce a high-quality, peer-reviewed scientific and clinical journal in an efficient and cost-effective manner. While our readership survey results and the letters to the editor which we receive tell us that members and readers find the *Journal* of value to them, we are always anxious to improve the *Journal* any way we can. The Committee would appreciate receiving the suggestions of readers so as to enable us to continue this task.

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1. International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *NY State J Med* 1992; 92:122–125.
2. Garfield E: Which medical journals have the greatest inpact? *Ann Int Med*. 1986; 105:313–320.
3. Imperato PJ: Readership survey of the *New York State Journal of Medicine*. *NY State J Med* 1987; 87:606–612.

RESEARCH PAPER

Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS

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ABSTRACT. Purulent bronchitis was identified in 19 of 422 patients undergoing fiberoptic bronchoscopy during a 32-month period because of suspicion of an opportunistic lung infection complicating acquired immunodeficiency syndrome or human immunodeficiency virus infection. Five patients had *Pneumocystis carinii* pneumonia, but other opportunistic lung infections were excluded in the remaining 14 patients. Characteristics of these 14 patients included fever (>38.3°C), cough, and dyspnea in 14 of 14 patients; purulence of expectorated sputum (11/14); and widened alveolar-arterial oxygen gradient (13/14). Rapid (2 ± 1.4 days) clinical response (defervescence and resolution of pulmonary symptoms) occurred with antibiotic therapy in 10 of 14 patients. In three patients, there was no improvement, and adult respiratory distress syndrome developed.

Bacterial isolates from bronchoalveolar lavage included *Streptococcus viridans* (*n* = 12), *Haemophilus influenzae* (*n* = 7), *Staphylococcus aureus* (*n* = 3). Roentgenographic features of bronchiectasis were present in seven patients. Differential cell counts revealed >50% neutrophils in the bronchial washings of all patients with purulent bronchitis. Neutrophil percentages in bronchoalveolar lavage were as follows: patient with purulent bronchitis without *P carinii* pneumonia (*n* = 14), 54.53% ± 29.18%; patients with purulent bronchitis and concomitant *P carinii* pneumonia (*n* = 5), 62% ± 31.9%. In a control group of 17 patients with *P carinii* pneumonia who did not have purulent bronchitis, the neutrophil percentage was 6.8% ± 6.17% (*p* = <0.00001, *t*-test). Purulent bronchitis appears to be a distinct, treatable entity in patients with HIV infection and may accompany bacterial pneumonia, bronchiectasis, and *P carinii* pneumonia

(NY State J Med 1992; 92:297–300)

Fiberoptic bronchoscopy (FOB) is frequently performed because of acute pulmonary symptomatology (with or without roentgenographic abnormalities) in patients known or suspected to have human immunodeficiency virus (HIV) infection. In August 1988, two such patients suspected of having opportunistic lung infection (OLI) underwent FOB and were found to have purulent inflammation of the airways. OLI including *P carinii* pneumonia (PCP) was excluded by appropriate stains and cultures. Rapid clinical but not radiographic resolution followed antibiotic therapy. This finding prompted a retrospective review of FOB records for the preceding 32-month period to identify other patients in whom bronchoscopic detection of purulent bronchitis complicating HIV infection had occurred. The clinical, roentgenographic, microbiologic, and lavage findings in these patients form the basis of this report.

PATIENTS AND METHODS

A retrospective review of all FOB procedures performed at Kings County Hospital Center between January 1986 and August 1988 was conducted. Patients with purulent bronchitis (mucosal erythema/edema/friability and purulent secretions in the airways) who had undergone FOB because of suspected opportunistic lung infection complicating HIV infection were identified. Medical records and chest roentgenograms for these patients were reviewed, and the following data were tabulated: age; sex; risk factors for HIV infection; symptoms; signs; sputum Gram's stain and culture; bronchoscopy results; cytologic examination of bronchoalveolar lavage (BAL) samples; viral, fungal, mycobacterial, and bacterial cultures of BAL; blood cultures; initial and subsequent chest radiographic findings; therapy; and outcome.

BAL was performed in the following manner: the right middle lobe bronchus and/or lingular segmental bronchus were lavaged with two aliquots of 50 mL of 0.9% sterile saline at room temperature, injected, and aspirated via a wedged fiberoptic bronchoscope. The lavage return was pooled and processed as described here. An aliquot of the BAL was preserved with an equal volume of 50% ethyl alcohol and then centrifuged at 5,000 rpm for 15 minutes; smears made from the sediment were stained with Papanicolaou, methenamine silver, and Ziehl-Nielsen stains and then examined. The cytologist described the predominant inflammatory cell and the presence or absence of protozoal, fungal, and acid-fast organisms. The remainder of the pooled BAL sample was sent for bacterial, fungal, mycobacterial, and viral cultures. BAL samples were not cultured for *Chlamydia*. Direct fluorescent antibody for detection of *Legionella* in the BAL was done routinely until October 1987. Since the diagnosis of *Legionella* pneumonitis was made so infrequently (2/415) in patients with AIDS at this institution,[1] routine testing of BAL with direct fluorescent antibody was abandoned.

Bronchial washings consisted of all bronchial secretions collected in a suction trap before and after the BAL. Bronchial washings were separately processed in the same fashion as the BAL.

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The diagnosis of acquired immunodeficiency syndrome (AIDS) was made according to the criteria of the Centers for Disease Control.[2] In lieu of HIV serologic testing, HIV infection was considered to exist when any of the following were present in a patient with a risk factor for AIDS: persistent fever of unexplained etiology of more than three months duration associated with weight loss and generalized wasting; diffuse cervical, axillary lymphadenopathy of more than three months duration; oral candidiasis in the absence of use of systemic antibiotics or oral/inhaled corticosteroids.

Slides of bronchial washings and BAL samples from the patients with purulent bronchitis identified in this series were retrospectively examined by a cytopathologist. A quantitative cell differential was obtained on 500 cells. Since five patients with purulent bronchitis also had PCP, we also performed similar cell differential analysis of BAL from 17 randomly selected patients in whom the diagnosis of PCP had been established, but whose airways appeared normal at FOB. The difference in percentage neutrophil counts in the following three groups was assessed by Student's *t*-test: patients with bronchoscopic evidence of purulent bronchitis in whom stains and cultures excluded opportunistic lung infection (group 1a), patients with bronchoscopic evidence of purulent bronchitis accompanying PCP (group 1b), and patients with PCP without purulent bronchitis (group 2).

Initial and follow-up chest roentgenograms of all patients were reviewed. Bronchiectasis was diagnosed only when persistent "tram-track" shadows and/or cystic changes were identified.[3]

Results

During a 32-month period, 422 fiberoptic bronchoscopies were performed to evaluate clinically suspected OLI complicating AIDS/HIV infection. Nineteen patients with purulent bronchitis and 276 patients with PCP were identified. One patient with purulent bronchitis presented twice within the study period. In addition to the bronchoscopic finding of purulent bronchitis, five of 19 patients were also found to have PCP. Thus, 14 patients had purulent bronchitis in the absence of an opportunistic lung infection. Of 19 patients, 18 were cigarette smokers (1–3 packs per day). Of 17 patients, 13 with PCP (group 2) were smokers.

Characteristics of patients with purulent bronchitis are shown in Table I. Serologic tests for antibody to HIV were obtained in four patients and were positive in all four. Clinical findings included fever (>38.30°C, 14/14), dyspnea (14/14), cough (14/14), sputum production (13/14), yellow/green sputum (11/14), hemoptysis (3/14), and nonpleuritic chest pain (1/14). Physical examination revealed diffuse peripheral lymphadenopathy (10/14), oral thrush (4/14), rhonchi (4/14), and crackles (9/14).

Ten patients had a rapid clinical response (defervescence and resolution of pulmonary symptoms) with antibiotic ther-

TABLE I. Characteristics of Patients with Purulent Bronchitis

Pt No.	Age (yr)	Sex	HIV Risk	A-a DO_2[*]	Serum lactate dehydrogenase (normal, 80–250 IU)	Chest Radiograph[†]	Antibiotic[††]	Days for Clinical Response
1	36	M	IVDA[§]	35	188	RML & RLL bronchiectasis	T-S	5
2	50	M	IVDA	21	212	RML & RLL bronchiectasis, DII	T-S	1
3	32	F	IVDA	50	311	RLL bronchiectasis, DII, bilateral HA	T-S, AMP	3
4	46	M	IVDA	13	144	RML bronchiectasis and collapse	P	1
5	49	M	IVDA	25	237	RML bronchiectasis, DII, right HA	P	3
6	28	M	IVDA	38	183	RML and LLL bronchiectasis	T-S	1
7	38	M	IVDA	42	NA	RLL bronchiectasis	NA	NA
8	39	M	IVDA	54	199	DII, bilateral HA	T-S	3
9	36	M	IVDA	54	188	DII, left HA and PA	OX, AMP	1
10	33	M	Homosexual	35	NA	Left lower lobe interstitial infiltrate, right HA and PA	T-S	1
11	26	M	IVDA	32	622	DII, cardiomegaly, right HA	T-S, V, AK	ARDS,[‖] death on day 6
12	31	M	IVDA, Bisexual	30	187	Bilateral lower lobe interstitial infiltrates, bilateral HA	P	1
13	37	M	IVDA	38	190	Nodular infiltrates (0.5–2.0 cm) in both upper and middle zones, cavitation in one nodule	Pentam	ARDS, death on day 6
14	39	M	IVDA	54	604	Patchy alveolar infiltrates in both lungs	P, CEF	ARDS, death on day 36
15	35	F	IVDA	68	737	DII	T-S, OX	4
16	45	M	IVDA	83	642	DII	T-S, E	ARDS, death on day 10
17	33	M	IVDA	45	435	DII	T-S	21
18	34	M	IVDA	28	475	DII	T-S	22
19	43	M	IVDA	65	576	DII	T-S, E	ARDS, death on day 7

[*] A-a DO_2 = alveolar-arterial oxygen gradient.

[†] RML = right middle lobe; RLL = right lower lobe; DII = diffuse interstitial infiltrates; HA = hilar adenopathy; PA = paratracheal adenopathy.

[††] T-S = trimethoprim-sulfamethoxazole; AMP = ampicillin; P = penicillin; NA = not available; OX = oxacillin; V = vancomycin; AK = amikacin; Pentam = pentamidine; CEF = cefotaxime; E = erythromycin.

[§] IVDA = intravenous drug abuse.

[‖] ARDS = adult respiratory distress syndrome.

apy (2 ± 1.4 days). Data regarding clinical response could not be retrieved for one patient. Three patients never responded to therapy. The clinical course of these three patients deteriorated rapidly, with progressive bilateral diffuse acinar lung infiltration and worsening oxygenation (PaO_2/FiO_2, ratio <150), leading to intubation and mechanical ventilation. In all three, adult respiratory distress syndrome (ARDS) developed, and two of the patients died of respiratory failure. One of these two patients had received pentamidine as the only therapeutic agent. Another patient had improvement of respiratory status before death on day 36, which was possibly due to meningitis of unknown etiology.

Trimethoprim (20 mg/kg/day intravenously), sulfamethoxazole ($n = 7$), penicillin ($n = 4$), and ampicillin ($n = 3$) were the most frequently used antibiotics. A single antibiotic was administered in seven patients. None of the patients had a positive direct fluorescent antibody test for *Legionella*.

Cytomegalovirus (CMV) was isolated by culture from BAL in three patients, but cytopathogenic effects of CMV were not noted in the cells of BAL or in transbronchial lung biopsies. *Candida albicans* was isolated from the BAL in four patients; all had evidence of oral thrush. Blood cultures in these four patients were negative for fungi, and the clinical picture was not compatible with candidal lung infection. Mycobacteria, fungi other than *C albicans,* and *P carinii* were not identified/isolated from bronchial washings or BAL in any of the 14 patients.

Chest roentgenograms showed changes consistent with bronchiectasis (7/14), bilateral interstitial infiltrates (7/14), patchy alveolar/nodular infiltrates (2/14), and localized interstitial infiltrate (1/14). Mediastinal adenopathy was noted in seven patients. None of the patients with bronchiectasis had earlier or coexistent parasitic or mycobacterial infection, although three complained of recurrent bronchitis treated with oral antibiotics. Resolution of infiltrates was not noted in any patient. Progression of infiltrates to diffuse alveolar consolidation was noted in the three patients who did not respond to therapy.

Transbronchial biopsies were done in 6 of 19 patients, three with radiographic features of bronchiectasis and three with diffuse interstitial infiltrates. Histologic findings consisted of acute/chronic inflammation, focal abscess formation, and fibrosis. Bronchial biopsies were obtained in two patients and revealed acute and chronic bronchial mucosal inflammation in one patient and marked thickening of the basement membrane in the other.

Bacteria were isolated from BAL in 15 patients: *S viridans* ($n = 12$); *H influenzae* ($n = 7$); *S aureus* ($n = 3$); *S epidermidis* ($n = 3$); *Klebsiella pneumoniae, Salmonella* species, *Serratia liquefaciens, Enterobacter cloacae, Campylobacter, Citrobacter, Acinetobacter*, and *Neisseria* species (all one each). Cultures were sterile in three patients, and the results could not be retrieved for one patient. Sputum cultures were obtained in seven patients before the FOB. The sputum culture was sterile in one patient, and normal upper respiratory tract flora were isolated in the other six patients. Blood cultures were sterile in 19 of 19 patients.

Results of BAL are shown in Table II. Percentage neutrophil counts in BAL in patients with purulent bronchitis without PCP (54.53% ± 29.18%) were not significantly different (*t*-test) from those of patients with purulent bronchitis with PCP (62% ± 31.9%). Patients with purulent bronchitis (group 1a and group 1b) had significantly higher percentages of neutrophils in the BAL (56% ± 29%) than the patients with PCP without purulent bronchitis (group 2) (6.8% ± 6.17%) ($p = <0.00001$).

TABLE II. Characteristics of Bronchoalveolar Lavage in Patients with Purulent Bronchitis

Pt No.	Days of Therapy Prior to FOB*	BAL[†] % Neutrophils	BAL Bacterial Culture[††]	Other BAL Pathogens[§]
1	6	36	SV	—
2	0	93	SV, HI, SA, KP, Sm	CA
3	4	51	Sterile	—
4	0	86	HI, SA, SV, SL, Cy, EC	CA, CMV
5	0	46	SV, SA	—
6	1	75	HI, SV	—
7	—	58	HI, SV	—
8	5	80	HI, SV	CA
9	7	30	Ct	CMV
10	3	6	SV, N	CMV
11	4	58	SE	—
12	8	2	HI, SV	—
13	0	93	—	—
14	9	33	SV, SE	CA
15	10	36	Ac	PC, MAI
16	8	89	Sterile	PC
17	2	74	SV, HI	PC, CMV
18	8	21	SV, SE	PC, CA
19	4	91	Sterile	PC

*FOB = Fiberoptic bronchoscopy.

[†] Bronchoalveolar lavage (BAL) washing neutrophil count >50% in all patients.

[††] SV = *Streptococcus viridans*; HI = *Haemophilus influenzae;* SA = *Stapylococcus aureus;* KP = *Klebsiella pneumonia;* Sm = *Salmonella* species; SL = *Serratia liquefaciens;* Cy = *Campylobacter* species; EC = *Enterobacter cloacae;* Ct = *Citrobacter;* N = *Neisseria*; SE = *Staphylococcus epidermidis;* Ac = *Acinetobacter.*

[§] CA = *Candida albicans;* CMV = Cytomegalovirus; PC = *Pneumocystis carinii;* MAI = *Mycobacterium avium intracellulare.*

DISCUSSION

The bronchoscopic observation of purulent bronchitis in patients with AIDS/HIV infection has not previously been emphasized. Indeed, our observations confirm that acute pulmonary symptomatology mimicking opportunistic lung infection in this population can be due to bronchitis. Mucosal inflammation in the 19 patients described here was diffuse, although the severity varied from mucosal erythema to severe mucosal friability. Purulent bronchial secretions associated with bronchial mucosal inflammation suggest an acute bronchitis. High neutrophil counts in the BAL sample suggest that this inflammation often extended to the distal bronchial tree. Two of the 14 patients in group 1a had patchy alveolar infiltrates, probably from a bronchopneumonia. In both of these patients, ARDS developed before death. Radiographic improvement was not noted in any of the patients with interstitial infiltrates, although their fever and symptoms resolved with therapy. It is possible that infiltrates in these patients may have had another etiology, such as nonspecific interstitial pneumonitis.

Cytomegalovirus and *Aspergillus niger* have previously been described as causative agents of necrotizing tracheobronchitis in patients with AIDS.[4,5] Although other pathogens were identified/isolated from the BAL in 11 of 19 patients, we believe that the causative agent of purulent bronchitis in these patients was probably bacterial. Presence of high fever as well as rapid defervescence and resolution of cough, dyspnea, and sputum production with antibiotic therapy, and the high percentages of neutrophils in the BAL suggest a bacterial etiology. Bacteria were isolated from the BAL in 15 patients, and *S viridans* was the most common bacterial isolate. A study of bacterial infections in children with AIDS documented that *S viridans* was the most common isolate ($n = 11$) in 24 episodes of pneumonia.[6] These investigators also noted cases of sinusitis, rhinitis, otitis media, and conjunctivitis caused by *S viridans* and *S epidermidis*.[6] In the current study, these same organisms, which are rarely pathogens, may have represented contaminants from the oral cavity. However, in the context of HIV infection, their pathogenetic role in purulent bronchitis cannot be excluded.

In adults, *S pneumoniae* and *H influenzae* are the most common pathogens associated with bacterial pneumonias complicating AIDS.[7-10] These studies did not note the presence of purulent bronchitis in the patients with pneumonia. *S pneumoniae* was not isolated from any of the patients with purulent bronchitis, although *H influenzae* was isolated from seven patients. The spectrum of bacteria isolated from the BAL in the current study is similar to the isolates from gastric aspirates of another group of patients with AIDS from this institution.[11] The similarity between the bacteria isolated from these hypochlorhydric patients and from the BAL in the current report is striking and raises the possibility that the upper gastrointestinal tract may be the source of these respiratory isolates.

Radiographic changes consistent with bronchiectasis were noted in seven patients. None of these patients had earlier or recent PCP, mycobacterial, or fungal infection. All 19 patients with purulent bronchitis were intravenous drug abusers and thus were more likely to have suffered earlier bacterial lung infections.[12] Three of these patients did relate a history of symptoms consistent with recurrent bronchitis. Symptoms recurred when antibiotics were discontinued. A similar smoking history was noted in patients with and without purulent bronchitis. Although bronchiectasis in patients with AIDS has not previously been recognized as a frequent problem, its presence in seven of these patients with purulent bronchitis suggests an association.

The high percentage of neutrophils in the BAL in the patients with purulent bronchitis most likely reflects extension of inflammation to the distal bronchial tree. The percentage of neutrophils in the BAL of patients with PCP, PCP with ARDS, and tuberculosis have varied from 1.8% to 25% in different studies.[13-15] In the current study, neutrophil counts in patients with PCP and with normal airways at FOB (6% ± 6%) were similar to those described previously, whereas the patients with purulent bronchitis had significantly greater neutrophil counts.

In conclusion, purulent bronchitis in HIV-infected individuals can produce significant pulmonary illness. In our experience, purulent bronchitis occurs most often with coexisting pulmonary disease including bronchiectasis, bacterial pneumonia, and PCP. Antibiotic therapy was usually effective in producing a prompt clinical response.

REFERENCES

1. Allam AA, Kamholz SL: Legionella pneumonia and AIDS. *Chest* 1989; 95:707–708.
2. Centers for Disease Control: Revision of the CDC surveillance case definition for acquired immunodeficiency syndrome. *MMWR* 1987; 36:1s–15s.
3. Luce JM: Bronchiectasis, in Murray JF, Nadel JA (eds): *Textbook of Respiratory Medicine*. Philadelphia, WB Saunders Co, 1988, pp 1107–1125.
4. Imoto EM, Stein RM, Shellito JE, et al: Central airway obstruction due to cytomegalovirus-induced necrotizing tracheobronchitis in a patient with AIDS. *Am Rev Respir Dis* 1990; 142:884–886.
5. Pervez NK, Kleinerman J, Kattan M, et al: Pseudomembranous necrotizing bronchial aspergillosis: A variant of invasive aspergillosis in a patient with hemophilia and acquired immune deficiency syndrome. *Am Rev Respir Dis* 1985; 131:961–963.
6. Krasinski K, Borkowsky W, Bonk S, et al: Bacterial infections in human immunodeficiency virus-infected children. *Pediatr Infect Dis J* 1988; 7:323–328.
7. Simberkoff MS, Sadr WE, Schiffman G, et al: *Streptococcus pneumonia* infection and bacteremia in patients with acquired immunodeficiency syndrome with report of a pneumococcal vaccine failure. *Am Rev Respir Dis* 1984; 130: 1174–1176.
8. Murata GH, Ault MJ, Meyer RD: Community-acquired bacterial pneumonias in homosexual men: Presumptive evidence for a defect in host resistance. *AIDS Res* 1984–85; 1:379–393
9. Polsky B, Gold JWM, Whimbey E, et al: Bacterial pneumonia in patients with the acquired immunodeficiency syndrome. *Ann Intern Med* 1986; 104:38–41.
10. Witt DJ, Craven DE, McCabe WR: Bacterial infections in patients with the acquired immunodeficiency syndrome (AIDS) and AIDS related complex. *Am J Med* 1987; 82:900–906.
11. Lake-Bakaar G, Quadros E, Beidas S, et al: Gastric secretory failure in patients with the acquired immunodeficiency syndrome (AIDS). *Ann Intern Med* 1988; 109:502–504.
12. Stoneburner RL, Des Jarlais DC, Benezra D, et al: A larger spectrum of severe HIV-1-related disease in intravenous drug users in New York City. *Science* 1988; 242:916–919.
13. White DA, Gellene RA, Gupta S, et al: Pulmonary cell population in the immunocompromised patient. *Chest* 1985; 88:352–59.
14. Plaza V, Jiminez P, Xaubet A, et al: Bronchoalveolar lavage cell analysis in patients with human immunodeficiency virus related diseases. *Thorax* 1989; 44:289–291
15. Smith RL, El-Sadr WM, Lewis ML: Correlation of bronchoalveolar lavage cell populations with clinical severity of *Pneumocystis carinii* pneumonia. *Chest* 1988; 93:60–64.

Münchausen syndrome

Case Presentation

Ping Fai Wong, MD, (Medical Intern): A 33-year-old black man was admitted with complaints of hemoptysis, gross hematuria, severe low back pain, and two episodes of syncope.

According to the patient, one year prior to admission, in a setting of recurrent hemoptysis, a diagnosis of Goodpasture's syndrome had been made based on positive serologic findings of antiglomerular basement membrane (anti-GBM) antibodies. A kidney biopsy was attempted, but tissue was not obtained for diagnosis. After a course of corticosteroid therapy, symptoms subsided; plasmapheresis was not performed.

On the day of admission, the patient reported a recurrence of hemoptysis, productive of approximately one cupful of blood, together with severe, low back pain; gross hematuria throughout the entire urinary stream; and two episodes of syncope. After examination by an internist, he was referred to the hospital for admission.

The patient denied fever, chills, night sweats, dyspnea, wheezing, chest pain, weight loss, and aspiration of or exposure to toxic materials. His family history was significant in that his father had had Goodpasture's syndrome.

The patient indicated that he had recently completed service as a Navy F-14 pilot and had returned from Saudi Arabia and service in the Gulf war on the day before admission. He did not smoke, consumed alcohol only occasionally, and was allergic to codeine and cocaine.

Physical examination on admission disclosed a well-developed man in mild distress caused by back pain. His blood pressure was 120/80 mm Hg, the pulse was regular at 96 min, the respiratory rate was 20/min, and temperature was 99.1°F. The skin was clear; no lymph nodes were palpable. Examination of the head, eyes, ears, nose, and throat was negative; the neck was supple without venous distention. Breath sounds were well transmitted in both lungs; no rales or rhonchi were audible. The first and second heart sounds were normal; no murmurs, gallops, or rubs were audible. The abdominal examination was negative, but both costovertebral angle areas of the back were markedly tender to palpation. The rectal examination was negative; the stool was negative on Guaiac testing for occult blood. The extremities showed no cyanosis, clubbing, deformities, or edema; all peripheral pulses were readily palpable. The neurologic examination was negative.

The urine was yellow and cloudy and had a pH of 5.5 and a specific gravity of 1.025; it was positive for glucose, positive 1+ for protein, and positive 3+ for occult blood. Microscopic examination disclosed many red blood cells, 2–5 white blood cells, few epithelial cells, and no casts. The hemoglobin was 13.1 g/dL; the hematocrit was 38.7%; and the white blood cell count was 8,800/μL. The platelet count was 450,000/μL; prothrombin time was 12.4 seconds (control 11.2 sec), and partial thromboplastin time was 27.7 sec (control 24.5 sec). The serum electrolyte levels were as follows: sodium, 143 mmol/L; potassium, 4.0 mmol/L; chloride, 107 mmol/L; and bicarbonate, 23 mmol/L. The blood glucose was 79 mg/dL; blood urea nitrogen (BUN) 11 mg/dL; and serum creatinine, 1.5 mg/L. Arterial blood gas determinations, with the patient breathing room air, were pH 7.43; P_{CO_2} 34 mm Hg, P_{O_2} 99 mm Hg; bicarbonate, 22 mmol/L Hg; base excess −1, and oxygen saturation 98%.

The chest film revealed a normal cardiac size and configuration, with no evidence of pulmonary infiltration. The electrocardiogram showed normal sinus rhythm, no axis deviation, and no acute ST- or T-wave changes.

During the first hospital day, the patient complained of continual low back pain, reported an additional bout of hemoptysis, and continued to have hematuria. Intravenous fluid hydration at 150 mL/hr was administered, and treatment with prednisone, 60 mg orally once daily, was begun.

On the second hospital day, symptoms continued. Repeat laboratory tests showed a slight drop in hemoglobin and hematocrit to 12.1 g/dL and 36.7% respectively; the BUN was 8 mg/dL, and the serum creatinine was 1.1 mg/dL; the urine was orange and cloudy with a pH of 7.5, specific gravity of 1.010, protein of 2+, and occult blood of 3+. The microscopic examination revealed 10–15/white blood cells per high-power field, few epithelial cells, and no casts, and was loaded with red blood cells.

Renal sonography showed no abnormalities; serologic tests for antinuclear antibody and for the third and fourth components of complement (C3, C4) were subsequently reported to be within normal limits.

Copies of earlier hospital records were said by the patient to have been mislaid. However, on the third hospital day, the record of the patient's admission to this hospital three years earlier was brought to the attention of the house staff and revealed a history of hemoptysis and hematuria associated with trauma at that time. An extensive diagnostic evaluation, including computed tomography scans, magnetic resonance imaging, bronchoscopy, cystoscopy, and retrograde pyelography at that time had failed to identify the patient's disease process. A psychiatric consultation had been recommended, although the patient now denied that he had been so advised.

In view of the findings from the previous as well as the present admission, all medications were discontinued and psychiatric consultation was requested. On the fifth hospital day, the patient left the hospital against medical advice.

Harold Coppersmith, MD (Attending Physician): The patient reported a history of hemoptysis one year before this present admission.

Dr Wong: He said that he had been evaluated for hemoptysis one year earlier in Los Angeles. This history

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Based on a medical conference held at the New York Infirmary Beekman Downtown Hospital, New York, NY, on April 26, 1991.

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changed on repeated questioning; at times, he would say that it had occurred in New Orleans.

Dr Coppersmith: Do you know whether he was in military uniform when he came to the hospital this time?

Dr Wong: No, he was not.

Dr Coppersmith: So he gave you a spurious history about being a Naval pilot.

Dr Wong: Yes.

Bruce Logan, MD (Acting Chief of Medicine): I note here that you made a diagnosis of malingering. How do you account for the hematuria?

Dr Wong: The hematuria was the result of his pricking his finger and dropping the blood into his urine specimens.

Arturo Constantiner, MD (Attending Nephrologist): I was under the impression that the tests for antinuclear antibody and complement were cancelled when the true nature of the patient's condition was discovered.

Dr Wong: No, those tests were not cancelled. The results of those tests were recorded in our microbiology laboratory. However, the anti-GBM antibody test was cancelled, since that test and others as well would have been costly and pointless.

Dr Coppersmith: Did he give a history of positive serologic findings of anti-GBM antibody?

Dr Constantiner: He said that the test had been positive on three occasions at the Scripps Clinic. His story was very convincing, with the exception that there is no familial tendency for Goodpasture's syndrome. He said that his father had had Goodpasture's syndrome. That discrepancy should have alerted us and raised an important question about the truth of the diagnosis in his case.

John T. Flynn, MD (Associate Chief of Medicine): And yet that statement about his father may have been the only part of his history that was true.

Dr Wong: It seems that he must have known someone who had Goodpasture's syndrome, perhaps his father, perhaps a friend.

Diane F. Giorgi, MD (Medical Resident): I will be talking about the Munchausen syndrome. Although self-inflicted injuries and diseases were recognized in Biblical times, it was in the Middle Ages that the first specific reference was made to hysterics who mimicked hemoptysis by placing leeches in their mouths and produced skin conditions by self excoriation. In 1843, Gavin attempted to distinguish between two groups of individuals who simulated illness.[1] One group was reported to seek compensation or to improve their personal positions and were labeled malingerers; the other group was described as simulating illness for no obvious reason other than an inexplicable gratification in deceiving physicians and others.

The latter syndrome was eventually classified in 1951 by Archer, who attached to it the name of Baron Karl Friederich Hieronymus von Münchausen, a colorful German aristocrat who entertained his guests and listeners with exaggerated accounts of his sporting and military adventures.[2] The patient with Münchausen syndrome travels from hospital to hospital, often over large stretches of the country and even to foreign countries, inventing tales, like the Baron, but about an alleged illness, and very frequently convincing physicians of the need for diagnostic tests and treatment.

According to the DSM-III-R, the syndrome falls under the category of chronic factitious disorders with physical symptoms, with the following diagnostic criteria: intentional production or feigning of physical (but not psychological) symptoms; a psychological need to assume the sick role, as evidenced by the absence of external incentives for the behavior, such as economic gain, better care, or physical well-being; occurrence not exclusively during the course of another Axis I disorder, such as schizophrenia.[3]

Several characteristic features have been named and are worth noting. *Laparotomaphilia migrans,* for example, the most common type, attracts close medical attention to what appears to be an acute abdominal condition, and results in a multiplicity of scars having generated the term "gridiron abdomen." Some of these patients have been operated on so frequently that adhesions leading to the development of true intestinal obstruction may confuse the clinical picture. In hemorrhagica histrionica, the patient specializes in simulating hemorrhagic phenomena, from the lungs or stomach, or some other form of blood loss. These individuals have been labeled "hematemesis merchants" or "hemoptysis merchants." In neurologica diabolica, complaints of paroxysmal headaches, loss of consciousness, or peculiar seizures attract medical attention. Dermatitis autogenica is characterized by skin lesions artificially produced. Hyperpyrexia figmentatica includes complaints of high fever and related symptoms, seemingly substantiated by falsely elevated thermometer readings.[2,4–6]

The patient with this syndrome is usually admitted to the hospital with an apparently acute illness embellished by an initially plausible and frequently dramatic history. The story is composed largely of falsehoods, repeated on numerous occasions in the past, and capable of misleading large numbers of people. The patient almost always quarrels violently with the nurses and physicians and discharges himself against advice. The correct diagnosis is almost impossible to make at first, and it requires a bold emergency department physician to refuse admission. Following are some of the features that may help to identify a Münchausen patient[1]:

1. *Pseudologica fantastica*: habitual and uncontrollable pathological lying with content intriguing to the listener, characterized by fantastic details, such as being a military commander captured by the enemy, an undersea diver working with Jacques Cousteau, a fighter pilot, a former member of underground resistance forces, a submarine commander tortured by the Gestapo, a wrestling champion, a criminal lawyer, a private investigator, or even a physician.[5] The usually dramatic medical histories, however, are vague in their details, inconsistent, and embellished with ready excuses for discrepancies.
2. Evidence, often visible, of previous medical or surgical care, such as scarring of all superficial veins, venous cutdown scars, gridiron abdomen, and cranial burrholes.[1,6,7] The patient often has actually wandered from hospital to hospital, to different cities, different parts of the country, or even to foreign countries.[4,6]
3. Medical sophistication: These patients can often recount textbook descriptions of the onset and causes of their alleged illnesses.
4. Disruptive hospitalization:[4] Patients flaunt hospital rules, shift complaints from one organ system to another, and require additional consultations.
5. Demands for medication, most frequently for narcotic and non-narcotic analgesics.
6. Absence of visitors: In spite of long hospital stays, these patients rarely have visitors; if they do, more often than not the visitor turns out to be an accomplice supplying necessary paraphernalia to continue the simulation.[1]
7. Truculence and evasiveness in manner.
8. An immediate medical history that portrays acute and harrowing events, but yet one that is not entirely convincing. No real evidence of a pathologic lesion or dysfunction is discernable, in spite of an apparent acute presentation of illness. Examples

include overwhelmingly severe abdominal pain of an uncertain type, cataclysmal blood loss unsupported by corresponding pallor, or dramatic loss of consciousness.

9. A family or medical history that proves to be unreliable.
10. A better than usual grasp of medical terminology.

The medical literature suggests that the course of the disorder begins with hospitalization in childhood or in adolescence, for a physical illness.[8] The image of the hospital is retained by the patient as a nurturing milieu, which the patient seemingly attempts to recreate. The onset of Münchausen syndrome is usually in early adult life; males outnumber females five to one in some reports, although there is no sex differential in others.

Predisposing factors seem to include the following: anger against the medical profession that may have originated in past medical mismanagement of an illness; employment in the medical field, as nurses, technicians, or even medical students; a significant relationship with a physician in the past; parental influence; and underlying dependent, manipulative, or masochistic personality traits.[1]

In the differential diagnosis, several entities need to be considered:[6]

1. It is first important to rule out a genuine physical illness. Many of the diagnostic and attempted therapeutic procedures that Münchausen patients undergo can lead to genuine physical problems and complications, such as intestinal obstruction. In addition, many of the means that these individuals use to produce the appearance of an illness, such as injection of coumadin, injection of fecal material, opening of surgical wounds with unsterile sharp instruments, can cause potentially life-threatening situations. Malingering differs from Münchausen syndrome in that the symptoms are produced in pursuit of a goal that is obvious and recognizable.
2. Although many Münchausen patients request drugs, it does appear that they obtain these drugs neither primarily for the purpose of intoxication nor to support an addiction. Studies show no physical or psychological dependence on drugs or other substances; when drugs are used in an effort to keep patients in a hospital, they are usually rejected.
3. Patients with somatoform disorders do not voluntarily produce symptoms and are not usually as sophisticated in relation to medical terminology and procedures as are Münchausen patients.
4. In hypochondriasis, there is an overinterpretation of minor symptoms or signs but no voluntary production of illness.
5. Unlike psychotic patients, Münchausen patients rarely have hallucinations, delusions, or severe thought disorders.

The majority of patients elude meaningful psychiatric intervention by resorting to abrupt departure from the hospital and failure to keep follow-up appointments.[6] Psychiatric hospitalization is probably an essential first step in treatment, but this is impossible without the patient's consent, which is often not forthcoming. The personal reactions of physicians or of staff members have considerable impact on the opportunities for treatment and the establishment of an effective cooperation with these patients. This type of patient, however, is expert at generating in physicians and nurses feelings of futility, bewilderment, betrayal, hostility, and contempt. Staff members attempt to deal with a situation in which they are denied one of the fundamental elements in their relationship with patients, truthfulness.[6,9,10]

Physicians should try to avoid expressing feelings of resentment when these patients belittle their diagnostic prowess or are verbally abusive or disruptive. They should also avoid any public unmasking of the individual's condition, which may portray the patient as an adversary and a fraud. This may precipitate the patient's flight from the hospital.

Dr Logan: I had the opportunity to see this patient and refer him to Dr Constantiner with the possible diagnosis, according to the patient, of Goodpasture's syndrome. I must say that the patient was very convincing, so much so that he used as a reference a former student of mine. The patient was very knowledgeable about his supposed condition and in fact gave me a lecture on the subject.

Michael Palmer, MD (Chief Resident): When I mentioned to this patient that I had been in the Air Force, he referred to a former friend of mine, a pilot, who was stationed in Sardinia with me in 1976, and said that he knew this friend. So, for the moment, his statement about a period of duty in the Navy seemed genuine.

Dr Constantiner: I feel badly that we missed the diagnosis initially. Very seldom do we have a case like this, and very seldom on this service have we needed to call on a psychiatrist as the definitive consultant.

Goodpasture's syndrome is not a condition that we see very often. In the course of overlooking the true diagnosis of Münchausen syndrome during the first few days of his hospital stay, we were guilty of several oversights. I do not feel as badly as the physicians in two other major medical centers in this metropolitan area must feel, who have already performed at least two renal biopsies on this individual.

There were a few things that did not fit the picture from the beginning. The patient said that he had been receiving the Silver Medal from Mayor Dinkins when he collapsed in the park and was brought here. He said he had been in a hospital with Goodpasture's syndrome for nine months. In a patient with a past history of hematuria and proteinuria, one of the historical items that should be checked is the employment or military record, to determine whether at earlier times the patient had ever been denied work or military service because of proteinuria-hematuria.

It is obvious that a patient with Goodpasture's syndrome would have failed his military physical examination and would not have been sent to fight for nine months in Saudi Arabia. The man, however, was very convincing, as Dr Logan said. He entered my office with a chart from another hospital, but we were so preoccupied with the urgency of arranging for his admission to our hospital here, that I did not at that time review in detail the chart that he had given us.

When I did so later, I found that it contained only the medical information that was obtained by Dr Palmer in Dr Logan's office, together with many reports of radiographic studies, lung scans, and reports of other diagnostic tests. However, it contained no definitive information of any positive value.

When he initially entered my office, he immediately asked me if I had a FAX machine, so that he could have the results of his past anti-GBM antibody tests faxed. After requesting these over the next two or three days, he said that they were at his house and that his girlfriend was going to get them, but she failed to do so.

The other examination that I did not perform personally at the time of my initial consultation or even on his first hospital day, was a microscopic examination on his urine. If I had done so, I would have been very skeptical about the possibility of renal hematuria. The urine was grossly bloody, and the dipstick test was positive for blood, just as it was initially in Dr Logan's office. However, eventually when I did the microscopic examination myself, the urine was loaded with round red blood cells and nucleated red blood cells that probably were not of glomerular origin; there were no other elements in the urine, no general casts or red blood cells casts.

I must credit Dr Muthiah Sukumaran for his suspicion a day or so later that this patient was a fabricator, when he pointed out that if the patient was a victim of Goodpasture's syndrome with bleeding, the chest film should be abnormal, which it was not.

His renal function was normal; the serum creatinine was 1.5 mg/dL, which after additional intravenous fluids, was reduced to 1.1 mg/dL. I was sure that this patient did not have Goodpasture's syndrome. It is probable that his father may have had that disease, and that was the source of his information about the disease, or perhaps he learned about it from another source.

Two other mishaps contributed to the problem. One was inadequate communication among our physicians. I discovered that the patient had been previously admitted to this hospital when, two or three days after his admission, I met Dr David Liu in the parking lot. I mentioned this patient to him. Dr Liu said that he had never seen a case of Goodpasture's syndrome, but he believed that three years earlier a patient with hemoptysis and hematuria had been admitted to this hospital, but proved to have no disease. I told Dr Liu that I did not think that this man was the same patient. However, it turned out that this patient was using the same name as the person on the earlier admission.

I called the Medical Records Department and was told that the chart of the earlier admission had been sent to the inpatient unit two or three days previously, where it was actually available to the house staff, and included the history of hemoptysis and hematuria but had not been reviewed in detail. After reviewing the earlier chart myself, it was obvious that the two patients were one and the same person, so I spoke with the patient's resident, and we cancelled all the remaining tests. I was able to stop the anti-GBM test and several other tests, but apparently not all of them.

My second comment is one that is brought to mind by a consultation request recently forwarded to me concerning another patient, who was reported to have had a serum creatinine level of 29 mg/dL. When I asked the house staff about the urinalysis, they said that it had not yet been obtained. My comment relates to the issue of obtaining basic information about a patient before pursuing additional and often expensive studies and consultations. I include myself as well. I would have arrived at the truth in this case far more quickly if I had personally examined this patient's urine under the microscope initially.

In addition, I learned from Dr Vecchio that as a medical student he had had prior contact with this patient, at which time the patient was masquerading as a wide receiver for the San Francisco 49ers.

Andrew J. Vecchio, MD (House Physician): When I was a medical student, I was working at a hospital in Brooklyn at the time that a friend of mine was on the renal service there. She told me about a patient who had been admitted stating that he had Goodpasture's syndrome and that he had been an all-pro wide receiver for the San Francisco 49ers. The renal attending became suspicious because microscopic examination of the urine did not show any casts, and the red cells were not sufficiently crenated to have passed through the kidneys.

The house staff team taking care of this patient then consulted by telephone with the sports department of a major New York newspaper and obtained a dosier on the genuine all-pro wide receiver of the same name. The house staff then sent in a medical student, a 49ers fan, armed with this dosier, to interview the patient; when his story did not fit with the information from the newspaper, the house staff began to observe the patient more closely. A few days later a nurse observed the patient as he disconnected his intravenous infusion and added blood to his Foley collection bag.

About a month after this, I was completing an elective in fluid and electrolytes at a major public hospital in Brooklyn. At renal rounds, I learned that a patient with Goodpasture's syndrome was to be presented with renal biopsy findings. Halfway through the presentation, I raised my hand, in spite of startled looks around me, and asked for this patient's name. It turned out to be the same as that of the patient I had learned about at the other hospital. I then told the audience the story of that earlier experience.

When Dr Giorgi told me that she was presenting a case of Goodpasture's syndrome that turned out in fact to be an instance of Münchausen's syndrome, I asked the patient's name, and was not surprised to find it was again the same person. I called a prominent nephrologist at my medical school and asked if he had reported this patient in the medical literature. He had not, but he told me of a Kidney Foundation meeting at another major medical center in New York, during the course of which a pathologist excused herself to examine a biopsy of a patient allegedly suffering Goodpasture's syndrome. Again, the patient was the one whose name was now so familiar to me.

It turned out that four of the nephrologists at that meeting were also familiar with this patient, and in addition, the nephrologist at my school told me that he knew of at least 11 hospital admissions and five renal biopsies that this patient had had in the New York City area alone.

So I do not think Dr Constantiner should be too hard on himself or on the house staff team. At least he and the team can take consolation in the fact that they alerted themselves to the true facts before the patient had a number of expensive and unnecessary tests, including still another kidney biopsy.

Kurt Shinbach, MD (Attending Psychiatrist): I agree with the presentations made, and I agree as well that we should not feel so badly, since his hospital stay of four or five days should be well within diagnosis-related groups limits for this elusive condition. However, it occurs to me that we are still in a perilous situation with this case. Right now the patient is out there somewhere, practicing his art in some other facility with another group of doctors. It is only a matter of time until he is readmitted here, more experienced in medical syndromes and even more capable in his presentation of history and symptoms. I say this to emphasize the chronic and probably life-long character of Münchausen syndrome. This is not only a life-long condition, but indeed a malignant one as well. When we think of chronic schizophrenia or chronically suicidal patients in state hospitals, none of these other serious mental disorders are more disruptive than Münchausen syndrome, which is more difficult to treat and, indeed, more intractable.

The difference between this condition and malingering is that the latter is entirely conscious and can even be adaptive. In concentration camps, prisoners frequently were malingerers for very good reasons that all of us would support. However, the Münchausen syndrome has an unconscious aspect. The symptoms are produced deliberately, but what is unconscious is that the person cannot do anything other than to continue simulating medical pathology. This condition involves a compulsion, which is beyond the victim's control and which is unconsciously motivated even though the symptoms and medical findings produced are intentional. As additional evidence of the debilitating nature of this disorder, the personal history often includes the absence of any further family involvement and any permanent employment.[11]

Dr Constantiner: The other patient who shared the room with this Münchausen patient said that he was very strange and that he never talked at all.

Dr Shinbach: That is not always typical. Some of these patients are very garrulous, with *pseudologia fantastica* as Dr Gorgi mentioned. They often talk quite a bit about their competence in sports, their involvement in warfare, and endless other exploits.

Solange G. Abunassar, MD (Attending Pathologist): If you told this patient that he really had Goodpasture's disease, would he accept the diagnosis or would he invent another illness as an excuse?

Dr Shinbach: I must say that this patient is a bit different from the usual Münchausen patient. He repeatedly returns with the same story, whereas most people with this condition simulate one disease in one hospital and another disease in another hospital, or even in the same hospital. Often when the physicians have ruled out the supposed primary disease the next disease comes along rather promptly. These patients are compulsive in their disorder.[12] I would like to add that I have never heard of a case of this condition that has been successfully treated.

Dr Flynn: There is another aspect of this disease, Dr Shinbach, of which I am sure you are aware. Münchausen-by-proxy is a situation in which a parent will bring a small child into a hospital and put the child through the same kind of experience that the Münchausen patient ordinarily will pursue. The parent may go from hospital to hospital inflicting this kind of abusive activity on the child. In one such instance, the mother took a small baby to a hospital, with a complaint that the child was excessively drowsy and had had several episodes of fecal vomiting. However, the nurses were skeptical because the baby had a perfectly soft abdomen and a negative abdominal radiograph. It turned out that the mother was feeding this baby a mixture of a sedative and the child's own feces. You might keep in mind this condition of Münchausen-by-proxy when you are working in the emergency department.

Dr Shinbach: You have made a very important point. However, the mothers in such cases may have different diagnoses than that of factitious disorder; they are not infrequently schizophrenic.[13]

Dr Flynn: The lengths to which the Münchausen patient may go in simulating physical illness can be truly appalling to the physician. A recent report from the NIH[14] recounted the course of a patient who surreptitiously administered epinephrine to herself, baffled her physicians completely in their exhaustive testing for a pheochromocytoma, and was subjected to bilateral adrenalectomy. Eventually, the discovery of needles, syringes, and vials of epinephrine in her bedside table uncovered the truth.

Dr Logan: I would not want this audience to have the impression that we should not listen to our patients. This is one case that is so unusual that we can make it the subject of a conference. It is always important for us to inquire into the outside lives of our patients and to listen to their replies.

REFERENCES

1. Hyler SE, Sussman N: Chronic factitious disorder with physical symptoms (the Munchausen syndrome). *Psychiat Clin North Am* 1981; 4:365–377.
2. Asher R: Munchausen's syndrome. *Lancet* 1951; 1:339–341.
3. Diagnostic criteria for factitious disorder with physical symptoms. *Diagnostic and Statistical Manual of Mental Disorders*, ed 3, revised. American Psychiatric Association, Washington, 1987, pp 290, 315–320.
4. Sale I, Kalucy R: Munchausen's syndrome. *Med J Aust* 1978; 2:523–525.
5. Spiro HR: Chronic factitious illness. Munchausen's syndrome. *Arch Gen Psychiat* 1968; 18:569–579.
6. Sussman N: Factitious disorders, in Kaplan HI, Sadock B (eds): *Comprehensive Textbook of Psychiatry,* ed 5, Baltimore, William and Wilkins, 1989, pp 1136–1140.
7. Ireland P, Sapira JD, Templeton B: Munchausen's syndrome. *Am J Med* 1967; 43:579–592.
8. Sussman N, Borod JC, Cancelmo JA, et al: Munchausen's syndrome. A reconceptualization of the disorder. *J Nerv Ment Dis* 1987; 175:692–695.
9. Luce JM: The legacy of Baron Munchausen. *Pharos* 1978; 41:19–23.
10. Nadelson T: The Munchausen spectrum: Borderline character features. *Gen Hosp Psychiatry* 1979; 1:11–17.
11. Burman D, Stevens D: Munchausen family. *Lancet* 1977; 2:456.
12. Stone MH: Factitious illness: Pathologic findings and treatment recommendations. *Bull Menninger Clin* 1977; 41:239–254.
13. Clayton PT, Counahan R, Chantler C: Munchausen syndrome by proxy. *Lancet* 1978; 1:102–103.
14. Keiser, HR: Surreptitious self-administration of epinephrine resulting in "Pheochromocytoma" *JAMA* 1991; 266:1553–1555.

Healing waters

JACQUELINE GALLOWAY

A unique group of approximately 8–10 senior adult Californians meet three times a week in the Rehabilitation Center pool at the University of California, Los Angeles (UCLA). They are not recovering from injuries or recent surgery, nor are they former Amateur Athletic Union (AAU) swimmers, professors in residence, nurses or doctors, nor are they receiving formal physical therapy. Most do not "swim" nor did they plan this distinctive gathering collectively as say, a tennis group or bridge club might add an activity to enhance their skills or develop new friendships.

This unusual group of people are crippled or handicapped from birth defects, chronic disease, or acquired permanent disabilities and each have independently and enthusiastically searched for an activity beyond their regimen of prescribed medications, braces, ice packs, heating pads, transcutaneous electrical nerve stimulator (TENS unit), or support groups. This is a gathering of courageous individuals who instead of giving-in or giving-up, care enough about taking responsibility for their pain to add this "pool meeting" to their medical protocols.

Acute pain is well understood and managed in trauma centers and emergency rooms and treatments are remarkably effective. Once maximum healing has taken place and if permanent damage results, the pain that may accompany the chronic condition can take almost total control of one's life. Although mastering chronic pain is not always possible, coping is a realistic goal.

Doctors have a responsibility to diagnose and treat underlying pathology and to coordinate methods with their patients to relieve pain. However, the patient has the obligation to make decisions about the choice(s) of rehabilitation (ie, surgery, braces, physical therapy, medication). The ideal relationship between the doctor and patient is co-partnership and not parent-child dependency.

Traditionally, physicians focus on diagnosing and treating disease and symptoms with less attention to long-term pain management or side effects from even minimal pain medication. There should be legitimate concern by both the treating physician and the patient about fostering addiction when prescribing pain medication. In my own experience, I discovered the need for a specialist in pain management to achieve effective levels of pain relief when my pain went from acute to chronic. Today, with the shift in modern medicine to specialization, I find primary physicians prudently apprehensive about initiating an aggressive course of pharmaceutical dependence for their patients with chronic pain. The dichotomy becomes a struggle by the patient to cope and by the doctor to prescribe safely and effectively.

The innovative Pool Program at UCLA is an adjunct to pain relief and was established by the physical therapists and developed to provide former physical therapy patients (especially Medicare recipients who are seriously handicapped) the opportunity to exercise independently and take part in an informal and more social pool activity. The program is not covered by medical insurance since it is not a formally prescribed therapy. An affordable fee was created to make the program available to handicapped patients who may have fixed incomes.

When I recently joined the group, I discovered an instant rapport with my fellow enthusiasts and our lively conversations were expanded because we are so individual in our medical histories and experiences. I found it refreshing to meet with patients who did not share my symptoms, such as one meets in specialized support groups. There is a mysterious sensitivity that occurs when we compare our personal physical limitations with others whose problems are dissimilar and as we gain new insights we also find strength within from the experience.

Represented in this elite group (but not limited to) are patients with Parkinson's Disease, Guillain-Barré Syndrome, poliomyelitis, peripheral neuropathy, systemic lupus erythematosus, cerebral palsy, multiple sclerosis and osteo and rheumatoid arthritis.

Uniting us in our newly found radical freedom of being in the buoyant warm water (which is maintained at 90°F), we achieve maximum movement within the confines of our physical restrictions. We agree that our pool time affords a special kind of emancipation. In fact, once we are all simply face-to-face in the pool and no longer see twisted limbs, paralyzed legs, limping or dystrophied muscles, we focus on our mirrored smiling faces as if we have been transformed by a seemingly mystical power in the water. We may not be transformed per se, but we have, however temporarily, transcended our constellation of problems.

The only time this scene is belied is when we enter or leave the pool via the special ramp or the Hoyer lift and have wheel chairs, canes, walkers, braces, or crutches awaiting us as we move toward the dressing room area.

The assigned aide, usually a pre-medical student who oversees our safety during the time we are in the water, provides light classical background music on a radio and has a ready supply of cold water for us to drink. The aide also helps us use an array of unique equipment. Along the deeper end of the 20′ × 30′, above-the-ground, stainless steel pool at approximately 5′, is a walkway with hand rails for those who can enjoy the "sense" of walking. A large steel table is in the water for us to either lie on or use for push-ups or leg-lifting calisthenics. There is a bench simply to rest, or to use for knee raising exercise, and we can add weights to our hands, elbows, ankles. Kick boards are

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available to just rest and float and finally for the child within, we can even reach for a yellow rubber duck that sits on the shelf at the edge of the pool.

Once in the water, our relationship to each inexplicably changes from tolerance to compassion. Reaching out to each other in compassion is a challenge, for tolerance is a live and let-live attitude. Our compassion motivates us to understand and experience each other's pain deeply enough to become involved with it and share a vulnerability. I base this on the fact that since we arrive at various times, it is quite usual for us to note a fellow friend entering the water and pull their favorite water tool toward them. If one is struggling to the edge of the pool for a glass of water, the person closest to the ledge will just pass it. In addition, since our handicaps are as diverse as the colors of a rainbow, we readily share any news of our progress or regression, but we are not reduced to total submissiveness even in frailty. We draw from each other a measure of inexhaustible hope. However, perhaps our signature attitude is that we are people with dignity who help ourselves and who have no illusions about miracles.

The image often perceived by people not disabled is that the handicapped live a sedentary life. However, our dedicated activity in the pool erases any notions of malaise existing in our lives. The age group represented ranges approximately from 60 to 83 years. The extended effort for each of us simply to get to the university for this exercise immediately eliminates any possible idea that we have retreated into defeat. Our "activity," in fact, is a bold contrast to the rest of our day and affords us a temporary return to an independence not available out of the water.

Although most are totally and permanently disabled, our group members represent a formidable array of talents, some current, others past. For instance, there is a physicist, an author and journalist, an elementary school teacher, a biologist, a former professional ice skater and coach, a real-estate investor, and a computer programmer. Conversations can be quite lively and eclectic.

The weightless environment of water allows us to do what is otherwise impossible. This special time builds self-respect and confidence and allows our reality of physical pain and limitation to be temporarily dispersed. This laudable, lofty endeavor becomes a transformation of our otherwise confined life. The water seems to be the catalyst that allows for new parameters and sets apart our daily struggle. It is one way to achieve self-motion, erasing the poverty of our earthly confinement.

The pool is not a panacea for pain and for some, it does not even reduce pain, but we collectively agree that the water reinforces a sense of independence and dignity. It is one alternative technique and the perfect solution to retaining a certain amount of control once ordinary movement becomes impossible.

This is a tribute to the resourceful physical therapy department at UCLA for establishing this uplifting activity and a commendation to the participants who have chosen the arduous path to self-help.

The treasure we in the Pool Program have is a light we bring from the buoyancy of our water sessions as we continue our day and return to our world outside of limited movement. Like children working toward the three R's, our three R's include working toward being refreshed, relaxed, and renewed.

Memories of an internship in 1943

LEO J. KOVEN, MD

During World War II our military establishment was in urgent need of medical officers to provide health services for the armed forces which inducted 17,000,000 American men and women. Accordingly, to expand the number of available physicians and to do so in the shortest possible time, two plans were put into effect through agreements made with the medical schools as well as hospitals with approved internships.

The schools agreed to shorten the time to graduation by three months by substantially reducing vacations for third and fourth year students. For example, my class on entrance was scheduled to graduate in June of 1943. Now our studies were to be concluded and degrees granted in March of that year. At the same time, the hospitals began our House Staff service on April 1 and reduced the customary one-year rotating internship to nine months, concluding on December 31, 1943. The combination of these condensed programs made us available for induction six months sooner than scheduled.

At that time, house staff selection was different from current practice in that there was no matching program. Candidates were interviewed in depth by a committee of the hospital's attending physicians. We, the applicants, approached this ordeal with trepidation. Now, many years later, I still recall my own interview in great detail. It was a harrowing experience and quite bullying. The committee felt it had much to offer those who were seeking this highly prized appointment and seemed to question our fitness for such a signal honor.

It was with great relief that shortly after my interview I received notification of my appointment to the Jewish Hospital of Brooklyn. The interns chosen along with me were outstanding candidates. Some remain my close personal friends to this day.

There are today many basic differences in the practice and art of medicine compared to the underlying processes and purposes we followed in 1943. At that time, evaluating the patient required a most intensive and searching performance in history taking and physical examination. Laboratory studies played a much lesser role in arriving at a diagnosis and selecting an appropriate therapeutic regime. With the subsequent development of so many sophisticated tests as well as the application of computer science, the history and physical have taken on a less dominating role.

Remember also that in 1943 much of the laboratory work was an arduous manual and time-consuming exercise, much of it assigned to the intern staff. This was our "scut work." Furthermore, the clinical work was accomplished by a very much smaller house staff than we have today.

Perhaps the greatest need of the patients was for eye contact, hand-holding, and other means of bonding. More specific remedies still lay in the future. There were no steroids, beta blockers, chemotherapeutic agents, and only the first generation of antibiotics, just to mention a few of our limitations. In view of the limited nature of our therapeutic armamentarium, we were called on to render care and concern at a more involved and personal level. We were always aware of our shortcomings, particularly because the patients so frequently literally clung to us for hope and reassurance. In the end, a relatively pain-free terminal bronchopneumonia was often the best friend of the elderly sick. Within those restrictions, I choose to think we left behind a legacy of an unusually high degree of excellence and commitment.

The first member of the hospital family I met when I began my internship and moved into staff quarters, and the last to note my final departure was Charlie. He was the watchdog of the kingdom; a veritable Cerebus at the gate. He operated the elevator that brought me to my room on the ninth floor on the Louria Building where I was housed. He was short and stocky with a round, owlish, ashen, dour mien. He invariably wore a dirty, sleeveless, knit cardigan vest. It was his uniform and badge of office. The stub of a cigar was firmly stuck in one corner of his mouth. He chomped on it incessantly, and the elevator cab reeked of his cheap stogies. A good five-cent cigar was still one of our country's unfilled needs. He was the consummate misanthrope, yet tolerated because without him it was a tough climb up nine steep flights.

I was assigned to a spartan room and shared it with Fred Tunick, since then a lifelong friend. The rooms, with their peeling paint known as "hospital" green and scarred concrete floors, were furnished with a pair of narrow surplus Army (circa World War I) cots whose exposed springs were covered by a thin pad called, by someone with an errant sense of humor, a mattress. To complete the decor there were a few wall hooks; a narrow table; a rickety chair; and a single, bare, overhead bulb of low, economizing wattage.

This building had been erected about 15 years previously and named in tribute of the late Dr Leon Louria, one of the earliest, and perhaps most eminent of the hospital's Chief Attending Physicians in the Department of Internal Medicine. Outside, facing St Mark's Place, it is still memorialized in an inscription carved in stone as the gift of Charles A. Levine. One may not recognize the name, yet it is a footnote in history. Levine was the first passenger to fly across the Atlantic Ocean, thereby qualifying him for inclusion in the Guinness Book of Records or at least a query in Trivial Pursuit. He had hired an aviator named Chamberlain to fly him on this journey. I rather think the cabin of the small, two-passenger biplane that made this historic flight had greater comfort and offered more amenities than our staff quarters.

The day we first reported to the hospital, we were given an indoctrination talk by the superintendent, Dr Morris Hinenberg. His title was the one then in vogue for a hospital Chief Executive Officer. I suppose that much later, seeing it shared by apartment house janitors, it fell out of fashion and was replaced by President. In those days, the superintendent and his principal assistants were usually physicians, but this never outwardly seemed to make them sympathetic to the problems of the house staff. It was wartime, and we were limited to 18 interns, and a similar number of residents, to staff a hospital of 616 beds. Today, the house staff at that facility numbers over 160, and they probably feel as overworked and imposed upon as we felt then.

Dr Hinenberg was an excellent administrator. He was so well regarded that after the war he left the institution to supervise all hospital affairs of the Federation of Jewish Philanthropies. While heading the hospital, he did not delegate his duties down through a chain of command. He was everything and everywhere and did not rely on any administrative infrastructure. He ruled by fiat and remained remote from the house staff.

I learned a valuable lesson in the art of negotiation from him. It involved the application of semantics. One day the interns met and decided they wanted a raise in their $10-a-month salary. At that time, this was more or less the standard wage in all the better voluntary hospitals. We petitioned Dr Hinenberg. As might be expected, he stonewalled our request. We then concocted a strategy, which seems quite juvenile in retrospect but which oddly enough was effective. The following day at noon, all 18 of us in succession called in to the paging operator and told her we would be out of the building for lunch for an hour. We signed out all our calls to Dr Hinenberg whose expected reaction quickly followed. We met outside his office, and I was elected with one vote of dissent (mine) to go in alone to represent the group. Timorously, I entered his sanctum sanctorum and faced him. He dispensed with all formalities and kept me standing, a most telling technique. He said, "How much do you radicals want?" Quite timidly, I proposed a $5-a-month raise. He responded, "When you go out never say I am giving a $5 raise. Instead, tell them they are getting a 50% salary increase." There was a twinkle in his eye, and I knew that he understood that we would get his point. He was actually human after all.

An institution is a depersonalized entity until you meet and know its many unforgettable characters. No one better typified our hospital as a living, dynamic, symbolic Mr Jewish Hospital than Max Abelman.

For some years, he had been an aide to a former chairman of the Board of Trustees in the latter's business pursuits. When the chairman retired, he arranged a sinecure for his faithful aide, Abelman. Either he was never given an official title or if he had one it is lost to memory; nor were his duties specifically laid down. He served à la Grover Whalen of that era as our official hospital greeter, public relations man, an ombudsman of sorts, and general factotum and facilitator. If you knew nowhere else to go, you went to Max. If you had a problem with no apparent solution, he found one and oddly enough, more often than not, it worked. He always lent a sympathetic ear, and if you were in need of one, a shoulder to cry on.

Although at times, his busy intrusions may have been a hindrance, on balance he was a greatly treasured asset. If it happened to be Sunday and you found yourself off duty, he would reach into his vest pocket and produce two free tickets for seats over the Dodger dugout; or for a rare night off, how about two, front center, house seats to the best show in town? If you were complaining of the heat and humidity in a summer without air-conditioning, you might find yourself with a guest membership at a nearby private swimming pool.

Max had an unabashed worship of titles and kudos. His small cubicle office next to the door of the front elevator was decorated with an abundant clutter of autographed pictures inscribed to him by various celebrities, as well as letters of appreciation, certificates of commendation, and the like. His greatest weakness was for titles of nobility. He might never become Lord Max, but he had aspirations at least of hob-nobbing with members of rank and privilege.

During my internship, he pulled off his greatest coup. Viscount Halifax had lost a son in combat during the war. Max seized the opportunity and invited His Lordship to visit us and unveil a generously sized, bronze plaque dedicated to the memory of his fallen heir. The East Pediatric ward was named in his honor. All of us were well rehearsed, freshly scrubbed and laundered, and placed on our very best behavior for the dedication ceremony. The Viscount arrived accompanied by the popping flash guns of the press, and the strains of "Pomp and Circumstance" which Max had selected thinking it most appropriate for the occasion. Halifax addressed our man as Mr Abelman, to which he responded with deep humility due such an august presence, "Call me Max." The noble Lord obliged, and Max ever after referred to him as "my very good friend, His Lordship; we're on first name terms you know."

The nursing staff were almost without exception our close pals and some even lovers, platonic and otherwise. Many strong bonds were forged, quite a few leading to matrimony.

Many of these angels (and I use the term selectively) were students and then graduates of the Nursing School. Nursing was considered an excellent avenue to a lifelong career for women. In those days unless a young woman came from a wealthy home, she was indoctrinated in her formative years to the necessity of earning an independent livelihood. Marriage was not a certainty, and even then her future family needs might require that she return at some time to gainful employment. The highest vocations for women were considered to be teaching and nursing. Although graduate nurses had a lower pay scale than teachers, longer hours ($90 a month for a 48-hour work week was standard), and fewer and shorter vacation periods, nursing unlike teaching did offer excitement, diversity of input, and an opportunity to meet a broad variety of people including many eligible bachelors.

Student nurses were completely involved in the day-to-day, hands-on delivery of patient care. This care, properly applied, was perhaps the most effective treatment modality in an era when so few specific remedies were available. Interns and nurses and students as well as graduates enjoyed working together while supporting each other's bruised egos and sharing their successes and failures.

The nursing students at the hospital were a particularly well-protected group. They even had their own Social Director and a chaperone system was in operation when we visited them in their lounge. They were so well guarded that we interns were not allowed to use the large swimming pool situated in the basement beneath their quarters. We, the predominantly male house staff, were told that the coliform count of the waters went up precipitously when we used that facility. We never quite understood the differences in perianal hygiene between the sexes and why chlorination failed in the presence of bacteria originating from the male body.

I spent a regrettably short time on the Internal Medicine rotation. There were four chiefs of service, each with his own staff. They rotated in charge of the wards and clinical instruction every three months. I felt very fortunate that my assignment came when Dr Meyer Rabinowitz was on service. The others were excellent directors and teachers, but he had the aura of being a diagnostician with a capital D. This title has now fallen into disuse, but in those days it was the highest tribute that could be paid a physician. An internist was a local luminary specializing in Internal Medicine, but a diagnostician was looked up to as a minor God. He was the court of last resort. I recall the story of the grieving wife who opened the door to receive a house visit from Dr Leon Louria. She cried that he had arrived too late because her husband had just expired. He stared at her haughtily through his awe-inspiring pince-nez and informed her that "No one is dead until *I* say so." The story may be apocryphal, but it conveys the image diagnosticians arrogated to themselves.

The ultimate manifestation of their right to the title was to come up with an obscure esoteric diagnosis, contradicting everything told the patient and family by the prior treating internist or family practitioner. If there were no demise and autopsy, the final arbiter, the diagnostician, was incontrovertibly correct. If a post-mortem examination took place and supported one by a previous physician, the diagnostician would shrug it off and declare the pathologist was undoubtedly mistaken, or at worst, he might lamely explain that he had simply been trying to teach his staff what to look for in a similar situation should it arrive in the future.

Dr Rabinowitz, when closely queried, would resort to quoting from obscure foreign medical journals as his supporting authority. Not only was their language usually unfamiliar to us, but also their very existence was thought to be open to question. He was a prodigious and wide-ranging reader. In his early years in medicine, reading was the only training readily available if one wished to be a specialist. You read the literature and went to hospital clinics seeking to apply it. I think our doubts concerning the source and accuracy of Dr Rabinowitz's quotations reflected on our own inadequacies rather than inventions on his part. He was a provocative presence who taught us the beauty and reward of study and hard work.

Undoubtedly the most fascinating patient I dealt with, among many of great interest, was a certain young lady. In those days we were inculcated in thinking of our charges as "patients" and not "cases."

She was admitted by wheelchair one evening and assigned to my ward. She was too feeble to walk independently. She was in her early twenties. On admission she was only 72 pounds even with massive edema and effusions. After being administered diuretics, her weight fell to 59 pounds. Before undergoing a clinical workup, she was transfused with plasma and glucose solutions and injected with massive doses of vitamins. She was a living museum piece exhibiting virtually every known intake deficiency. Her teeth were loose, and she was covered with subcutaneous petechia. Her tongue was black, and she had the dementia and diarrhea characteristic of pellagra. Her hair had a reddish tinge, and she had the abdominal tumescence of an advanced protein deficiency. These were only some of the clinical manifestations of her nutritional deprivation illness.

She was markedly resistant to our efforts to improve her condition although we had barely, yet dramatically, snatched her from an almost certain grave. She had to be restrained from pulling out all her life-supporting, intravenous lines. Her throat tissues were so eroded and fragile that we could not safely pass a gastric feeding tube. After she improved a little, she promised to eat if we would remove her restraints.

We did so, and put her on a soft, high-calorie diet. Two days later, a blind diabetic in the next bed, who was on a measured restricted intake, went completely out of control. One of the other patients on the ward told me that my patient would switch food trays with her unsuspecting sightless neighbor. We then assigned a student nurse to stand at her bedside and supervise feedings. I passed by one day shortly thereafter and asked her why she was coughing so much. She said she had great pain on swallowing because of her throat lesions. She had a box of tissues next to her, would cough into one with almost every bite, then toss the tissue under her blankets. I pulled them aside and found each tissue contained a bolus of unswallowed food. After this we removed the tissues, but then her trips to the bathroom became quite frequent. One of the nurses followed her in once and caught her with her fingers down her throat inducing retching.

Finally, she promised to be good and cooperative, but only if she could talk to me at length. She confided that her mother was an incessant shrew who constantly reminded her of her physical unattractiveness. Her features were rather unbecoming, and she had been moderately obese as a young adult. She was taken reluctantly by her mother to a marriage broker, who arranged a match made palatable by a substantial dowry. She came to the connubial bed a virgin and with no conception of the sexual act. She had received no instruction in this regard from her mother and lacked the curiosity to inquire of her few friends.

She was married off to a coarse truck driver who would throw himself on her passive supine form, rapidly satisfy himself, and then roll off to fall into a deep sleep. He was quite a satyr who would repeat this several times a day. He rarely spoke to her but conveyed his wishes at home with liberally applied kicks and punches. She retreated into severe anorexia. This finally succeeded in making her so utterly repulsive that her bestial husband presumably found other outlets for his insatiable demands.

I spoke to her, and her mother at some length. The mother finally seemed to understand the problem. We were able to discharge the patient to her mother's care after she brightened up and gained 15 pounds through a well-received dietary regime during a prolonged hospitalization. I hoped for the best, although I had reservations. It was a personal triumph to have talked a patient and her family into a possible cure.

Two months later, a fellow intern told me she had been admitted from the Emergency Room the previous evening again in an advanced state of nutritional deficiency as had been noted on the first occasion. Unfortunately, she died early that morning.

Her mother was waiting in the hallway to see me. When I arrived she said, "Why didn't I listen to you? I made her go back to live with that man."

The mid-1930s witnessed the discovery of the first broad spectrum group of effective antibiotics, the sulfonamides. However it became apparent that their range was limited, and they were soon found generally useless since most pathogenic organisms quickly developed resistance to them. Later, in the same decade, Fleming's serendipitous earlier finding regarding penicillin was published. The drug had not been synthesized and had to be extracted from the common, blue-green, fluffy bread mold. To manufacture it, the mold was cultured over large, flat surfaces containing an appropriate liquid media, and the top growth produced penicillium which was then skimmed off and extracted.

There was a company located in Brooklyn, a small chemical and drug manufacturing firm, whose principal product up to then had been citric acid extracted from sawdust for use in agriculture. The company's nearby neighbor was a brewery. At that time, during the war, activities were suspended at the brewery. The pharmaceutical concern's president had the inspiration for a use for the idle vats. He instituted the first large-scale production of penicillin employing these leased copper basins on which to grow the mold.

The federal government had decided that production of this new, quite potent, antibiotic should be logistically aligned with the war effort. Dr Chester Kieffer of Boston was appointed to control its distribution. It was determined that the most logical priority would be to use it to treat venereal disease in the Armed Forces. Venereal disease was epidemic and this new treatment would return large numbers of men back to active duty quite rapidly. Only very small doses were needed in those early days when resistance had not yet appeared.

Dr Leo Loewe was a prominent and creative member of our Department of Internal Medicine. The pharmaceutical company's executive secretary developed subacute bacterial endocarditis (SBE) as the result of a *Streptococcus viridans* infection of a damaged heart valve. She went to Dr Loewe who informed her and her employer that up until then the condition had been invariably fatal but perhaps she could be cured if given the new wonder drug. No one could possibly know for sure, for it had never been tried for this condition. If some of this closely sequestered product could somehow be diverted, we could treat her condition; in effect it was a testing of uncharted waters. We received a supply of penicillin and administered it to her. Her rapid recovery was seen as virtually a miracle.

An eight-bed room was soon set up on Ward 2 Center. I was assigned to a two-month, full-time rotation to work exclusively with her and other patients with similar illnesses under Dr Loewe's supervision. I did the house staff work while a technician named Russell did the lab work.

In the short period of perhaps two weeks or so, we were treating eight patients with verified SBE and seven were recovering. This was achieved with the minuscule doses of penicillin then available. I recall that 5,000 U/day were administered by continuous intravenous drip. I remember this quite well because the vials were kept in a separate, locked refrigerator on the floor. I was the keeper of the key, and each morning gave the medication nurse the required amounts.

The results were so dramatic in their newly discovered, life-saving potential that Dr Loewe immediately had them published. He received a warning letter from Dr Kieffer for disregarding national guidelines regarding the distribution and use of this new and scarce drug. He demanded that Dr Loewe immediately cease using it. Under pressure, Dr Loewe agreed to a public debate to be held in Louria Auditorium. It was a meeting moderated by the highly respected, venerable Dr Bernard Oppenheim of New York

City. The audience was so large it overflowed the main floor and the balcony, as well as the aisles, and spread out into St Mark's Place. To accommodate those who couldn't get inside, a loud speaker was hastily set up.

I prepared the graphs and slides, but they were dispensed with because Dr Kieffer immediately admitted the results. He emphasized that the issue did not concern the efficacy of penicillin, but rather the flouting of national guidelines. There was much flag-waving and, in its use against venereal disease, penicillin was declared to be the equivalent of adding an entire infantry division to the war effort. Dr Kieffer carried the day not because of the popularity of this argument, but simply by exerting his executively granted authority.

Dr Loewe was undaunted, however. He made an arrangement with the United State Public Health Hospital on Staten Island where there was a separate ward for Merchant Seamen who were being treated with penicillin for venereal disease. He arranged that all their urine be collected in large carboys set out in the center aisle of the room. When filled, the containers were rolled out on pallets and trucked over to our hospital to fill the large Lyster bag suspended from the ceiling of Ward 2 Center. Eight tubes, by gravity drip, carried this suddenly precious excreta to the bed sides and were hooked to gastric tubes passed into each patient. Clamps were set to deliver predetermined amounts of the urine into the eight unsuspecting stomachs. We had no way of assaying the obviously diminished capacity of the recycled drug via this most uncertain route. Yet surprisingly Loewe was able to record a few further cures. He had circumvented the guidelines, yet had not broken any Government regulations. Working with Dr Loewe on this penicillin project was the most exciting aspect of my all too brief internship.

Each month one intern was assigned to the private service of Dr William Linder. He had been Director of Surgery almost since the hospital's founding in 1901. He was past the mandatory retirement age and was now a consultant to the department. He had been offered an extension of the directorship but turned it down since he no longer wished to be involved in administrative duties. He continued with his very large private practice at the St Mark's Place office shared with his brothers, John and Sam. To reward him for his past services, he was assigned the exclusive services of a monthly rotated intern. It was a most sought after assignment, and I felt privileged to receive it.

Every morning, at 7:30 AM, I met him at the front entrance of the hospital. I helped him out of his coat and hung it up in the staff room. I then followed behind carrying his little black bag, a badge of office without which no physician ever made hospital rounds. His brother Sam usually trailed along and did the dressing changes, cleaned wounds, and removed sutures. Dr William, the name to which he had become referred, would discuss the progress of each patient, and a trail of attending surgeons and house staff members hung on every pearl of wisdom he uttered.

After this, we would proceed to the operating room. After scrubbing and prepping the patients, he would operate, and the surgical resident would tie and clamp while I pulled on the retractors. It was marvelous to watch him apply his skill while noting how often his preoperative diagnosis was confirmed at surgery.

He was an autodidact who had reached the highest rung of his chosen ladder. Those days produced a breed of famed clinical greats and in my minor and peripheral role, I was privileged to have seen some at work.

The foremost neurosurgeon in the country was acknowledged to be Dr Harvey Cushing of Yale. His first assistant for many years had been Dr Leo Davidoff. For personal reasons, Davidoff reached the point where he wished to leave New Haven. When I began my internship, he had recently accepted the surgical directorship at the Jewish Hospital even though he was not certified as general surgeon. Dr Davidoff was a distant and unapproachable man, quite regal in manner. He never indulged in social chitchat or made a nonessential remark. He was viewed with great awe by all of us. Yet, he stimulated us to work up to our fullest potential and perform at an extremely high quality level.

Only once did I see him falter. One day the hospital barber, Morris Sukoff, actually bested him. Sukoff and I were waiting for the elevator on the ground floor. When the door opened, Dr Davidoff, as usual, lost in deep thought, walked up from behind, thrust himself between us, absent-mindedly brushing us aside, and entered first. Sukoff, with his highly waxed and pointed bristling mustache, turned lobster red and appeared to be on the verge of an apoplectic fit. Dr Davidoff and I were going to the operating room on the tenth floor. Sukoff waited until the elevator let him off at his shop on the eighth. He strode to the door of the cab, drew himself up to his full height, all 5′3″ and as he was about to leave, turned toward Dr Davidoff and said, "Some people may think you are a great neurosurgeon but when they need a head doctor, they call on me, Morris Sukoff. Good day! " The ride from the eighth to the tenth floor was interminable. For once, Dr Davidoff had lost his composure.

Our greatest general surgeon at that time was undoubtedly Dr Rudolph Nissen. He had been first assistant and heir apparent to perhaps the most renowned surgeon of the first half of this century, Dr Sauerbruch of the Charite Hospital in Berlin. To everyone's surprise, Sauerbruch, in the late 1930s, had accepted an appointment as the Nazi SS Chief Medical Officer. Yet, he must have had some guilt feelings about Nissen who, under the Nuremberg Laws, qualified as a future victim of the Holocaust. After the Krystalnacht riots in 1938, Sauerbruch had him spirited away to Turkey where he was immediately given a cabinet post as Minister of Health. Two years later, in the approaching shadow of World War II, he found asylum in the United States.

He operated primarily at our hospital. He suffered indignities from other staff members who perhaps viewed him as an economic threat. He was constantly sought out by patients with very serious surgical conditions bearing the most ominous prognoses. There was no branch of surgery or its subspecialties he could not undertake with great expertise. I was very impressed by the superb technique I witnessed each time I was called on to hold retractors for him.

His practice was international and his fame widespread. His practice included such luminaries as Queen Juliana of the Netherlands and Albert Einstein, on whom he operated for an abdominal aneurysm. When he returned to Europe to head surgery at the University of Basle, they built a free-standing, 400 bed hospital for him and his department.

In spite of his genius, however, he had a fatal flaw, at least for us as interns. He never developed a comfortable command of English. He would only make rounds or operate with a fellow Berliner, Dr Albert Schiffman, who was a surgical resident at the time. They spoke exclusively in German. We followed behind or held retractors, never understanding their conversations. This was unfortunate because he had such an incredible wealth of skill and knowledge, which was not being transmitted to us. We were left with a blurred memory of deftly moving fingers and never a wasted motion.

My last month of clinical rotation at the hospital was on the obstetrical service. Two interns and three residents were assigned there. The hospital was then averaging 600 deliveries a month. The demands on us were so intense we hardly ever had time to return to our rooms in Louria. Instead, we ate and slept in the On Call Room in the obstetrical pavilion. Many deliveries, perhaps most, involved our non-paying ward (now called service) patients. As an intern, I was to deliver the uncomplicated multiparous patients. In addition to assisting at about 200 deliveries, I personally brought 70 newborns into the world.

I think the word "characters" was created for our obstetrical attending staff members. I can recall sitting and waiting with Nathan Adler and hearing one of his patients in a labor room screaming wildly with each contraction. I informed him that she must be ready, judging by the decibel level of her shrieks. He said no, not until she yelled "MAMA!" He was right. Soon came the scream for her mother, followed a few minutes later by a fine new baby girl.

Overall it was great fun. I have never worked harder yet the environment was most stimulating. The highest reward would come when a mother would ask me to choose a name for her newborn. It made it all seem very worthwhile to a neophyte with a newly bestowed license.

Two events marked our final days at the hospital. With our nine months of servitude concluded, we would be receiving our orders to proceed to military posts.

The first of these events was a banquet at which the lay board and administration of the institution expressed their gratitude for our devotion to the hospital and promised us staff appointments when we returned to enter practice. Since most of the older attendings escaped military service, they remained behind and secured their turf by adopting a bed allotment quota system that was favorable to them. At first, when we returned from military service, we found ourselves in pools of 20 physicians each, with one bed at a time available for the entire group. Under greater protest, this was modified to a half-a-bed quota for each one of us, or about 180 patient bed occupancy days per annum for each newly returned physician.

As a reward for our services, we were each given a 24-karat, gold-plated pen and pencil set at a final banquet. They made much of telling us it was heavily gold-plated, (although the underlying base metal was soon exposed through wear). The instruments were engraved, not with our initials, but with the hospital's, which was JHB. Most of us referred to it colloquially as the BJH, but over the stone lintel of the front entrance at 555 Prospect Place one can still see the engraved Jewish Hospital of Brooklyn Non Sectarian, a great oxymoron.

The final affair was a New Year's Eve Party held in the Louria Auditorium. For weeks, our house staff committee had been dunning the attending staff for financial contributions. Some were generous, and some dug deep down to come up with a munificent 25¢. Our caterer was Ben and Sol of local delicatessen fame. Somehow, in spite of wartime rationing, they provided a memorable and somewhat pungent feast. The party came off without a hitch. We departed the next morning in a haze of champagne and fumes of garlicked pastrami, and the sure feeling of a job well done.

Purpura fulminans, metastatic endophthalmitis, and thrombotic thrombocytopenic purpura in an HIV-infected patient

URMILA SHIVARAM, MD, MARY CASH, PA

Purpura fulminans (PF) is characterized by ecchymotic cutaneous lesions and is preceded by infection.[1] It has been described as a complication of pneumococcal sepsis in patients who have undergone splenectomy.[2] In their review, Spicer and Rau[3] suggested that the three primary clinical features of PF include fever, hypotension, and large purpuric ecchymotic areas of skin. The extracutaneous manifestations included involvement of kidneys, lungs, adrenals, rhabdomyolysis, and mycotic cerebral aneurysm.[4] It is increasingly recognized that thrombocytopenic purpura (TTP) can occur in patients with human immunodeficiency virus (HIV-I) infection.[5,6] Therapy with plasmapheresis results in a favorable outcome.

In this report we describe a case of purpura fulminans that developed subsequent to an episode of *Streptococcus pneumoniae* septicemia in a splenectomized HIV-infected patient. His stormy hospital course included bilateral metastatic endophthalmitis, bilateral maxillary sinusitis, herpetic lesions in the airways, and TTP, all of which fully resolved. To our knowledge, these complications have not previously been described, to be associated with *S pneumoniae* septicemia resulting in PF.

CASE REPORT

A 34-year-old man was admitted to the hospital with complaints of fever, not feeling well, and purplish spots on the body for one day prior to admission. In 1981, he had been diagnosed as having idiopathic thrombocytopenic purpura, which was treated with splenectomy; subsequently, pneumococcal meningitis developed. He had a history of intravenous drug abuse.

On admission, the patient exhibited symptoms of toxicity, had a temperature of 104°F, and was confused. The chest and abdominal examinations were unremarkable. There was a positive Kernig's sign. He had bluish red patches over both lower extremities. The tip of the nose and upper lip were bluish in color. All the peripheral pulses were palpable. A chest roentgenogram was normal, and remained normal throughout his hospital stay.

The peripheal white blood cell count was 27,300/μL, with 93% neutrophils, 3% band cells, and 4% lymphocytes. The hematocrit was 49%, and the platelet count was 45,000/μL. The serum electrolyte and hepatic enzyme levels were within normal limits. The creatine phosphokinase count was 1,796 U/L; prothrombin time was 16.9 sec (control, 11.6 sec); activated partial thromboplastin time was 94 sec (normal, 24.6 sec); serum fibrinogen level was 311 mg/dL; and fibrin splint products (FSP) were greater than 40 mg/dL. The patient was thought to have fulminant pneumococcal or meningococcal meningitis, sepsis, disseminated intravascular coagulation (DIC), and purpura fulminans. A lumbar puncture was thought to be too risky a procedure to perform in this patient because of the coagulopathy. Intravenous penicillin and heparin were started.

The following day, gangrene developed in the tip of the nose, lip, right ear, and both feet and there was pus in the anterior chamber of both eyes. The patient remained alert and maintained normal blood pressure and good urine output. The blood cultures were positive for *S pneumoniae*. The test for D-dimer, a breakdown product of heparin, was positive.

On the third hospital day, visual acuity in both eyes was limited to light perception with bilateral conjunctival injection, cloudy corneas, and hypopyon (Fig 1). The fundi could not be visualized because of fibrinous exudation. Local steroids (prednisolone sodium phosphate, two drops in each eye) were administered hourly for the next two weeks. An echocardiogram to test for endocarditis was normal. A computed axial tomography (CAT) scan of the head revealed bilateral maxillary sinusitis. After four days of therapy with penicillin and heparin, there was marked clinical improvement, and the coagulation profile returned to normal.

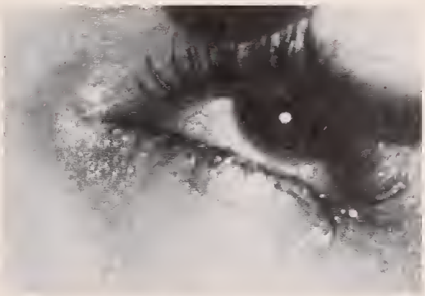

FIGURE 1. Gangrene of the tip of the nose and hypopyon of the left eye.

On the seventh hospital day, herpetic lesions developed over the patient's neck, around the lips, and in the larynx, trachea, and bronchi. A biopsy of the lesions and bronchoalveolar lavage grew herpes simplex virus (HSV-I); serology for HSV-I was positive (1:32). The patient was treated with intravenous acyclovir, 350 mg every eight hours for ten days, with good response. On the ninth hospital day, the patient had surgical drainage of both maxillary sinuses. There were gangrenous areas of the skin and soft tissue over both lower extremities that later became well demarcated.

On the 16th hospital day, fever and leukocytosis developed, and the platelet count decreased to 20,000/μL. The source of infection was thought to be the gangrenous legs. He underwent bilateral above-the-knee amputation. On the 17th hospital day, thrombocytopenia worsened (platelet count, 10,000/μL) the hematocrit dropped to 22.5%, PT, PTT, fibrinogen, and FSP were within normal limits; blood urea nitrogen (BUN) was mildly elevated (55 mg/dL); and creatinine was 1.6 mg/dL. A peripheral smear revealed evidence of microangiopathic hemolytic anemia. A diagnosis of thrombotic thrombocytopenic purpura was made. He was initially treated with fresh frozen plasma with no response. He later received plasmapheresis for the next two weeks with good response, and his platelet count was 265,000/μL at the end of this period. The vision in his eyes improved remarkably within two months of admission to near normal vision (visual acuity of 20/50 in the right eye and 20/25 in the left eye). There was evidence of a Roth spot in the left eye and no evidence of cytomegalovirus (CMV) retinitis. His serum was positive for HIV.

After ten weeks of intensive management of his various complications, the patient was transferred to a rehabilitation medicine ward. He remained in the hospital for six months. Since discharge, he has been admitted to the hospital twice, once for a viral syndrome and once for gram-negative septic shock. He is currently being followed in the Infectious

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Disease clinic, and he takes prophylactic amoxicillin, 250 mg twice daily.

Discussion

PF is an uncommon complication of septicemia. Most of the clinical manifestations of PF are secondary to DIC. Reports of PF in splenectomized hosts after pneumococcal septicemia[2] raise the possibility of the role of the spleen in the prevention of DIC and PF. In the patient described here, unusual manifestations of PF developed, including bilateral metastatic endophthalmitis and bilateral maxillary sinusitis. The maxillary sinusitis might have been the source of the *Streptococcal pneumoniae* septicemia, since his chest roentgenogram and arterial blood gases were entirely normal.

In a review of 38 patients with acute states of PF reported in the literature, all patients were noted to be in shock and to have hypotension. This is described as one of the cardinal clinical features of sepsis with PF.[4] However, the patient presented here, in spite of being very ill, maintained good blood pressure and urine output throughout his acute illness.

In the same review of 38 patients, the most frequent extracutaneous manifestations in PF were pulmonary (23%), renal (21%), gastrointestinal bleeding (13%), adrenal hemorrhage (5%), and mycotic cerebral aneurysm (2%). In some other patients, rhabdomyolysis has also been reported as a complication of sepsis resulting in PF.[2] In our review of the literature, metastatic endophthalmitis and sinusitis have never been reported to be associated with PF, although endophthalmitis has been seen in association with pneumococcal sepsis.[7]

The mechanism of endophthalmitis is not clear in this patient, and it may be related to hematogenous dissemination of infection or direct spread from contiguous sites, such as the sinuses.

Although most patients fail to regain vision after endophthalmitis with medical therapy alone,[8] this patient regained useful vision with intensive medical management; this has been reported previously in one patient after meningococcal septicemia.[9] We could not aspirate the pus in the anterior chamber or institute any intraocular antibiotics because of severe coagulopathy at the time of presentation. *S pneumoniae* infection has been previously associated with acquired immunodeficiency syndrome. It has been recommended that treatment courses should be prolonged in these patients.[10]

Another interesting aspect of this case was the development of herpetic laryngotracheitis which responded to prompt therapy with acyclovir. Although herpetic laryngotracheitis may develop with severe underlying infections, this complication has not been associated with PF and may be related to the decreased immune status of the patient. Various pathogenic factors like endotoxins, immune complexes, or mediators of endothelial cell damage result in vascular injury, such as occurs in patient with TTP.

A secondary form of TTP has been reported in association with herpes simplex virus infection.[11] This may be the result of an infection-triggered immune response. Penicillin, a commonly used antibiotic, has also been reported to result in microangiopathic hemolysis and thrombocytopenia.[12] A variety of factors like HIV infection, HSV infection, or penicillin adminstration could have been resulted in TTP in this patient.

The association of TTP with HIV-I infection has been previously recognized.[5,6] Therapy with steroids and plasmapheresis has been found to be successful in patients with HIV infection. The presence of fever, alteration in mental status, and thrombocytopenia should alert the physician to the possibility of TTP in patients infected with HIV.

We report a case of *S pneumoniae*, resulting in PF, sinusitis, metastatic endophalmitis complicated by herpetic laryngotracheobronchitis in a patient with positive HIV serology. This represents an unusual clinical presentation of *S pneumoniae* septicemia. We suggest that a decreased immune response on the part of the host due to HIV infection could have resulted in these unusual complications. Aggressive management resulted in the patient's remarkable recovery.

References

1. Dugegeon DL, Kellogg DR, Gilchrist GS, et al: Purpura fulminans, *Arch Surg* 1971; 103:351–358.
2. Hautekeete ML, Berneman ZN, Bieger R, et al: Purpura fulminans in pneumococcal sepsis. *Arch Intern Med* 1986; 146:497–499.
3. Spicer TE, Rau JM: Purpura fulminans. *Am J Med* 1976; 61:566–571.
4. Chu DJ, Blaisdell FW: Purpura fulminans. *Am J Surg* 1982; 143:356–362.
5. Leaf AN, Laubenstein LJ, Raphael B, et al: Thrombotic thromobocytopenic purpura associated with human immunodeficiency virus type I (HIV-I) infection. *Ann Intern Med* 1988; 109:194–197.
6. Nair JMG, Bellevue R, Bertoni M, et al: Thrombotic thrombocytopenic purpura in patients with the acquired immunodeficiency syndrome (AIDS) related complex. *Ann Intern Med* 1988; 109: 209–212.
7. Jarrett WH, Wells JA, Hyman BN: Metastatic endophthalmitis: A report of 3 cases in proven septicemia. *South Med J* 1971; 64:194–198.
8. Rowsey JJ, Newsom DL, Sexton DJ, et al: Endophthalmitis: Current approaches. *Ophthalmology* 1982; 89:1055–1066.
9. Jay WM, Schanzlin DJ, Fritz KJ: Medical therapy of metastatic meningococcal endophthalmitis. *Am J Ophthalmol* 1979; 87:567–568.
10. Armstrong D, Gold JWM, Dryjanski J, et al: Treatment of infections in patients with the acquired immunodeficiency syndrome. *Ann Intern Med* 1985; 103:738–743.
11. Myers TJ, Wakem CJ, Ball ED, et al: Thrombotic thrombocytopenic purpura: Combined treatment with plasmapheresis and antiplatelet agents. *Ann Intern Med* 1980; 92:149–155.
12. Parker JC, Barnett DA: Microangiopathic hemolysis and thrombocytopenia related to penicillin drugs. *Arch Intern Med* 1971; 127:474–477.

Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient

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Benign lymphoepithelial lesions (BLL) of the parotid gland were until recently a rare pathologic entity representing one manifestation of Sjögren's syndrome.[1] In the past five years, several investigators have reported cases of parotid masses that are histologically identical to BLL in patients with human immunodeficiency virus (HIV) infection.[2–8] In some cases, these parotid masses have been the presenting symptom of HIV infection.[3,4,6,8] We present two cases of BLL associated with HIV infection and discuss the diagnosis and treatment of this entity. Because of the common association of BLL with HIV infection, primary care physicians and specialists alike must presume that any patient presenting with a parotid mass typical of this entity is infected with HIV until proven otherwise.

Case Reports

Case 1. A 39-year-old, homosexual man with acquired immunodeficiency syndrome (AIDS) presented to the Division of Otolaryngology in January 1990 complaining of a right pre-auricular mass. The mass was painless and had been slowly enlarging for 6–7 months. The patient denied fever, chills, weight loss, cough, dyspnea, vomiting, or diarrhea. His

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medical history was significant for right-sided Bell's palsy five years before this presentation. The right facial nerve weakness persisted.

Physical examination revealed a 3 × 2 cm mass in the right tail of the parotid gland which was rubbery, moveable, and nontender. The patient also had weakness of the entire right seventh cranial nerve, which he said was unchanged in the past five years. The remaining physical examination was noncontributory.

A fine-needle aspirate of 25 ml of straw-colored fluid from the mass was negative for malignant cells or microbes. The magnetic resonance imaging (MRI) scan after aspiration demonstrated a slightly lobulated soft tissue mass of the right parotid gland (Fig 1). The appearance was nonspecific, and the differential diagnosis included lymphoma as well as inflammatory disease. Superficial parotidectomy was recommended.

On admission the complete blood cell and platelet counts, blood chemistries, electrocardiogram, and chest film were all within normal limits. Immunologic cell marker studies demonstrated a helper-suppressor T-cell ratio of 0.36, and a reduced absolute number of helper T-cells. The patient underwent an uncomplicated right superficial parotidectomy. Pathologic studies demonstrated a benign lymphoepithelial lesion of the parotid gland (Figs 2, 3).

This patient underwent diagnostic superficial parotidectomy to rule out malignancy because of the uncharacteristic appearance of the lesion on MRI scan. The abnormal appearance may well have been due to the fine-needle aspiration of the cyst just before the scan. The cystic fluid had not yet reaccumulated, and the appearance of the lesion was uncharacteristically solid.

Case 2. A 64-year-old, white man presented with a two-week history of a nontender, slowly enlarging, left pre-auricular mass. The patient denied pain, fever, chills, facial paralysis, or any HIV risk factors.

Physical examination revealed a 5 × 5 cm nontender, doughy, mobile mass in the left tail of the parotid gland. The rest of the physical examination was unremarkable. There was no palpable cervical adenopathy or facial nerve weakness.

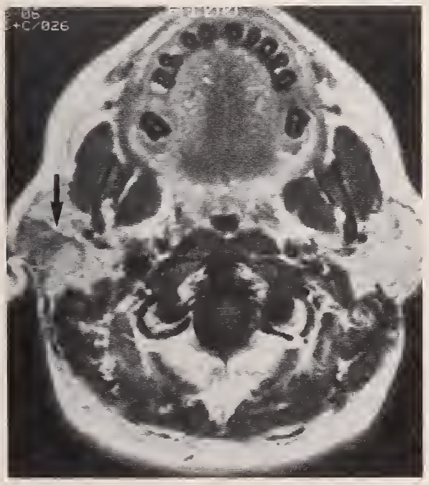

FIGURE 1. The mass does not enhance on T^2-weighted image (arrow). This MRI image does not represent the typical appearance of a cystic BLL associated with HIV infection even though the final pathology was consistent with this diagnosis.

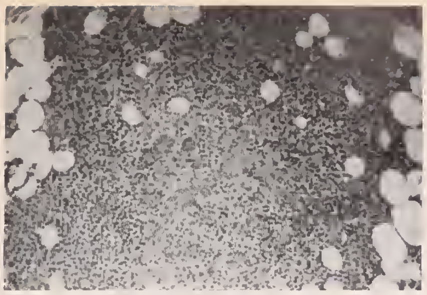

FIGURE 2. Infiltration of parotid parenchyma with small, heterogeneous lymphocytes, and focal germinal center formation commonly found in cystic BLL. (hematoxylin and eosin stain; original magnification × 100).

Fine-needle aspiration of the cyst yielded 10 ml of straw-colored fluid. The cytology was consistent with a benign lymphoepithelial lesion. The fluid reaccumulated in one week. An MRI scan demonstrated a bilobed mass in the left parotid gland with surrounding inflammatory change characterized by ill-defined T^2 hypersensitivity around the mass (Fig 4). There was no significant cervical adenopathy and no right parotid lesions. On further questioning, the patient admitted to being homosexual. He had been found to be HIV negative three months earlier. He subsequently refused serologic testing at the time of presentation to us but was suspected to be HIV positive on the basis of his homosexual behavior. Because of the characteristic findings on fine-needle aspiration cytology, radiology, and physical examination, no surgical intervention has been undertaken, and the patient is being followed as an outpatient.

DISCUSSION

Cystic benign lymphoepithelial lesions of the parotid gland were rare until the early 1980s. This entity became much more common since that time in association with HIV infection.[2–8] BLL was the presenting entity for HIV infection in about 50% of cases in one series.[8] The parotid swelling is uniformly cystic, nontender, and bilateral in 15% to 80% of cases reported. The overlying skin, salivary flow, and Stensen's ducts all remain normal. This entity is associated with persistent generalized lymphadenopathy syndrome in 15% to 50% of patients. Constitutional symptoms of weight loss, anorexia, fever, chills, and diarrhea are uncommon in association with BLL, and should alert the practitioner to a possible underlying malignancy.

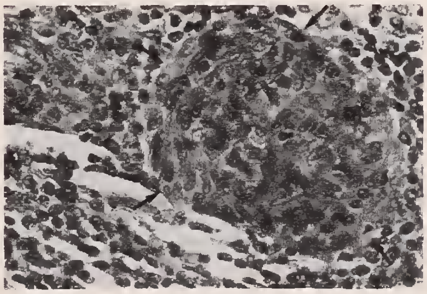

FIGURE 3. Epimyoepithelial island typical, but not pathopneumonic of BLL (arrows). (hematoxylin and eosin stain; original magnification × 400).

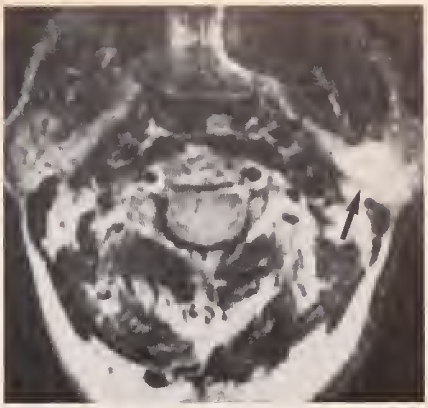

FIGURE 4. T^2-weighted axial MRI image demonstrates hyperintensity of the mass and surrounding parotid gland parenchyma, indicating inflammatory changes (arrow). This MRI image represents the typical appearance of a cystic BLL associated with HIV infection.

The HIV serology is positive for infection in 70% to 100% of patients tested.[4,6–8] The T^4/T^8 (helper-suppressor T-cell ratio) is uniformly inverted.[7,8] Sperling et al[8] suggest that BLLs are an early symptom of HIV infection as evidenced by the following findings in their series of 15 patients: BLLs represented the presenting entity of about half of the patients, and the absolute helper T-cell count was greater than 200 cells/mL in all of the patients who had T-cell subsets counted.

Computed tomography (CT) and MRI studies of these lesions reveal multiple parotid gland cysts.[4] These cysts appear as thin-walled, low attenuation masses on CT scan. MRI scan reveals low-to-intermediate attenuation on T^1-weighted images and high attenuation on T^2-weighted images (Fig 2). There may be ill-defined T^2 hyperintensity in the surrounding parotid gland, signifying some inflammatory changes. BLL may also be associated with cervical or intraparotid adenopathy on CT or MRI scan.

Fine-needle aspiration can be both diagnostic and therapeutic. On gross examination the fluid tends to be clear to straw-colored and, less commonly, turbid. Finfer et al[9] found BLL most consistently associated with findings of a heterogeneous lymphoid population, scattered foamy macrophages, and squamous cells. Occasionally, squamous cells were absent in their series of samples, and salivary gland tissue was always scarce or absent. The fluid in the cysts usually reaccumulates rapidly; if it does not, the fine-needle aspiration can result in a smaller and more esthetically appealing mass.

The benign lymphoepithelial lesions associated with HIV infection appear tan to pink and cystic. The cysts usually contain watery, yellow fluid. On microscopic examination, the lesions demonstrate partial-to-near total replacement of parotid parenchyma with small lymphocytic infiltration and focal germinal center formation (Fig 3). The lesions contain few or no acini, and ducts are in varying stages of degeneration. Epimyoepithelial islands are commonly found throughout the lesions (Fig 4).

The pathogenesis of BLL has been theorized by numerous investigators.[1–3,5–7,11,12] Most speculate that the BLL associated with HIV infection originates in the intraparotid lymph nodes that are either incompletely encapsulated, or contain epithelial inclusions, or both.[2,5–7,11] The areas of affected parotid gland parenchyma then represent areas where the lymphocytes extend through defects in the lymph node capsule, causing proliferation of ductal epithelium and pressure necrosis of the acinar structures. Others speculate that the changes seen in the parotid gland are attributable to an autoimmune process, as evidenced by the presence of myoepithelial islands (a hallmark of autoimmune damage to salivary glands) in the affected glands.[3,5,12] In this scenario, the damage incurred by the disease process closely resembles the effects of Sjögren's disease on the salivary glands.

Several investigators have addressed the problem of nomenclature with regard to these lesions. Bernier and Bhaskar[1] deliniate the difference between benign lymphoephithelial cysts (BLC) and BLL by stating that the former represent cysts within the lymph nodes, whereas the latter represent a process that originates in the lymph nodes but extends into the glandular tissue, causing the pathologic changes previously outlined. The cases reported in the literature demonstrate a spectrum from lesions entirely encapsulated in lymph nodes to near total replacement of the parotid gland with disease. Case 1 reported here demonstrates pathologic findings characteristic of BLL. We believe that the literature supports that BLC and BLL in association with HIV infection represent different points along the spectrum of the same disease process (originating in the intraglandular lymph nodes), and we therefore propose that both lesions be named BLL associated with HIV infection.

The treatment of BLL associated with HIV infection is not well defined. If the patient is in the early stage of HIV infection, has characteristic radiologic and clinical findings, and demonstrates fine-needle aspiration cytology consistent with BLL, observation is a reasonable option. Because of the low but real risk of Kaposi's sarcoma[9,10] and lymphoma[2,5,7] presenting in the salivary glands of HIV-infected patients, one must follow these patients closely. Most patients reported with these associated malignancies have demonstrated signs of malignancy not characteristic of BLL, such as a painful mass, rapid growth of the mass, or constitutional symptoms of weight loss, anorexia, and weakness. Additionally, two of three patients found to have lymphoma in one series had a T^4/T^8 ratio of 0.2 or less, indicating a later stage of HIV infection. If any signs of malignancy develop or if characteristic diagnostic findings are not present we suggest biopsy of the mass. Superficial parotidectomy or open parotid biopsy are reasonable procedures in these patients. Recurrence of these masses after superficial parotidectomy has not been reported.

References

1. Bernier JL, Bhaskar SN: Lymphoepithelial lesions of salivary glands: Histogenesis and classification based on 186 cases. *Cancer* 1958; 11:1156–1179.
2. Ryan JR, Ioachim HL, Marmer J, et al: Acquired immune deficiency syndrome-related lymphadenopathies presenting in salivary gland lymph nodes. *Arch Otolaryngol Head Neck Surg* 1985; 111:554–6.
3. Ulirsch RC, Jaffe ES: Sjögren's syndrome-like illness associated with the aquired immunodeficiency syndrome-related complex. *Hum Pathol* 1987; 18:1063–1068.
4. Shugar JM, Som PM, Jacobson AL, et al: Multicentric parotid cysts and cervical adenopathy in AIDS patients. A newly recognized entity: CT and MR manifestations. *Laryngoscope* 1988; 98:772–775.
5. Smith FB, Rajdeo H, Panesar N, et al: Benign lymphoepithelial lesion of the parotid gland in intravenous drug users. *Arch Path Lab Med* 1988; 112:742–745.
6. Finfer MD, Schinella RA, Rothstein SG, et al: Cystic parotid lesions in patients at risk for the acquired immunodeficiency syndrome. *Arch Otolaryngol Head Neck Surg* 1988; 114:1290–1294.
7. Ioachim HL, Ryan JR, Blaugrund SM: Salivary gland lymph nodes: The site of lymphadenopathies and lymphomas associated with human immunodeficiency virus infection. *Arch Pathol Lab Med* 1988; 112:1224–1228.
8. Sperling NM, Lin PT, Lucente FE: Cystic parotid masses in HIV infection. *Head Neck* 1990; 12:337–341.
9. Finfer MD, Gallo L, Perchick A, et al: Fine needle aspiration biopsy of cystic benign lymphoepithelial lesion of the parotid gland in patients at risk for the aquired immune deficiency syndrome. *Acta Cytologica* 1990; 34:821–826.
10. Reath DB, Noone B, Columbus M, et al: Primary Kaposi's sarcoma of an intraparotid lymph node with AIDS. *Plast Recon Surg* 1987; 80:615–618.
11. Ioachim HL, Ryan JR: Salivary gland lymphadenopathies associated with AIDS. *Hum Pathol* 1988; 19:616.
12. Jaffe ES, Ulirsch RC: Salivary gland lymphadenopathies associated with AIDS. *Hum Pathol* 1988; 19:1120.

Ten-year survival in multiple myeloma: Report of two cases and review of the literature

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Improved treatment of patients with multiple myeloma over the past three decades has resulted in an increased median survival rate. Although five-year survival rates have been frequently reported,[1–5] ten or more years of survival in multiple myeloma is still uncommon. We cared for two patients, one who survived ten years, the other who is still alive with active disease 12 years after diagnosis. In this article, we describe these two patients, as well as 48 other patients reported in the literature, who survived ten or more years with multiple myeloma.

CASE REPORTS

Case 1. A 50-year-old man presented in 1979 complaining of low back pain, hand swelling, and impotency. A diagnosis of immunoglobin G (IgG) κ-multiple myeloma was made on the basis of anemia, bone marrow plasmacytosis of 85%, urinary excretion of 1.37 g of free κ-chains/24 hours, and monoclonal serum IgG level of 4.2 g/dL. The patient was treated for two years with monthly courses of melphalan, cyclophosphamide, carmustine, and prednisone. This led to disappearance of the monoclonal spike from the serum and urine, an increase in hemoglobin, and a decrease of bone marrow plasma cells to 10%.

The patient suffered a relapse in 1988 with worsening anemia and massive κ-light-chain proteinuria (16 g/24 h) but no reappearance of the serum spike. Therapy with melphalan and prednisone was restarted and continued monthly for two years with improvement clinically and on laboratory studies. His urine protein level dropped to less than 1 g/24 h, and his serum IgG was 572 mg/dL. He underwent hemicolectomy in 1989 for stage 1 (Duke's level A) colon cancer. In late 1991, the patient is free of pain and has a normal

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hemoglobin level, trace κ-light-chain proteinuria, and serum polyclonal IgG of 1.7 g/dL. He is receiving no therapy for his myeloma.

Case 2. A 60-year-old man presented in 1981 with progressive weakness. Blood studies revealed that he was anemic (hemoglobin 8.4 g/dL) and had 9.8 g/dL of a monoclonal IgG κ-paraprotein. Bone marrow plasmacytosis of 60% confirmed the diagnosis of multiple myeloma. *Strongyloides stercoralis* and *Giardia lamblia* were found in his stools, and the patient was treated for these parasites. He was given intermittent melphalan for multiple myeloma for three years, and his condition markedly improved. The serum IgG level decreased to 5.4 g/dL (but the paraprotein on electrophoresis persisted), and the hemoglobin count increased to 14.5 g/dL. Chemotherapy was discontinued in 1984 with the patient asymptomatic. In 1986, worsening anemia and increasing serum IgG levels (to 10 g/dL) prompted the reinstitution of therapy with monthly melphalan and prednisone. Late in 1990, the hemoglobin decreased to 7.9 g/dL, serum IgG level increased to 11 g/dL, and the patient experienced progressive weakness and bone pain. A single course of vincristine, doxorubicin, and dexamethasone (VAD) was administered, but pseudomonal sepsis developed, and the patient died in March 1991. Post-mortem examination was refused by the family.

DISCUSSION

In 1972, Pankovich and Griem[6] described a patient with plasma cell myeloma whom they followed for 30 years. The patient presented initially with a lytic lesion (plasmacytoma) of the ischium, followed several years later by multiple additional lytic lesions and paraproteinemia. The patient responded to radiotherapy, and diffuse plasma-cell myeloma did not develop. In 1978, Gomez[7] reported a patient who, following laminectomy for spinal cord compression, was found to have diffuse multiple myeloma. The patient was alive and well ten years later but still had 45% plasma cells in his bone marrow and a serum IgG level of 2.4 g/dL. Berrebi and Estrov[8] followed a patient for 20 years and considered him to have smoldering myeloma. Another ten year survivor was reported by Boccadoro et al[9] in 1971. In 1982, Franklin et al[10] reported a patient with multiple myeloma at an unusually young age of onset, characterized by IgG paraproteinemia, epibulbar involvement, and a 14-year survival.

In 1983, Kyle[11] reviewed the records of 870 Mayo Clinic patients diagnosed as having myeloma over an 11-year period. Nineteen patients (2.2%) survived for ten years or longer. All had symptomatic disease requiring chemotherapy at the time of diagnosis. The major difference between the 19 long-term survivors and the larger group of 851 was the response to therapy. Of the long-term survivors, 18% (17/19) had an objective response (>50% reduction in monoclonal serum spike or urine protein) to the chemotherapeutic alkylating agent within one year. The response to therapy in the overall group of 870 patients was not given, since most of them were not followed by the authors.[1] Lee and Myers[12] cite at least four patients alive with multiple myeloma more than ten years after diagnosis. In 1984, Dutcher and Wiernik[13] reported a patient with stage III multiple myeloma with documented disease-free survival of more than 16 years. The investigators suggest that the patient is cured since he is alive and well without evidence of disease. Another patient with non-secretory multiple myeloma developed Ph^1-negative chronic myelogenous leukemia with plasma cell antigens 15 years later.[14]

In 1985, Alexanian[15] reported that 13 of 305 (4%) consecutive patients with multiple myeloma survived ten years or more. Virtually all were less than 65 years old, presented with low- or intermediate-stage disease and responded well to chemotherapy. Acute leukemia caused the death of two of six long-term survivors. Three more ten year survivors were described in 1986 by Bladé et al.[16] A patient who survived for 21 years with IgD multiple myeloma died of lung cancer with no evidence of multiple myeloma at autopsy and is labeled as cured.[17] This patient was included in a previously reported series of long-term survivors.[11]

In 1989, a 17-year survivor in continuous complete remission for 13 years since a

TABLE I. Reported Cases of Ten and More Years of Survival With Multiple Myeloma

Authors	Year	Ref.	No. Cases	Sex*	Age at Diagnosis (yrs)	Immunoglobulin Type	Duration of Survival (yrs)	Outcome	Remarks
Pankovich and Griem	1972	6	1	M	48	IgG	31+	—	Presented with plasmacytoma. Multiple lytic lesions and paraprotein two years later.
Gomez	1978	7	1	M	41	IgG	11+	Alive	
Berrebi and Estrov	1981	8	1	M	56	IgG-L	20+	Alive	
Boccadoro et al	1981	9	1	M	44	IgG-K	10+	Alive	
Franklin et al	1982	10	1	M	37	IgA	14	Died	Also had multiple plasmacytomas
Kyle	1983	11	19	11 M, 8 F	NS	12 IgG, 4 IgA, 1 IgD 2 K	10+	14 died 5 alive	3 died of acute leukemia; 1 cure?
Lee and Myers	1983	12	4	NS	NS	NS	10+	4 alive	All 4 patients untreated
Dutcher and Wiernik	1984	13	1	M	36	IgG	16+	Alive	Cure?
Paietta et al	1985	14	1	M	64	nonsecretor	15	Died	Transformed to CML†
Alexanian	1985	15	13	3 M, 10 F	NS	9 IgG, 4 IgA	10+	6 died 7 alive	2 died of ANNL‡
Blade et al	1986	16	3	1 M, 2 F	64	IgG-L	11	1 died	
					51	IgA-K	14+	2 alive	
					52	IgA-L	15+		
Kyle	1988	17	1	M	52	IgD-L	21	Died	Presented with plasmacytoma. Multiple myeloma six months later. Cure? Died of metastatic cancer. No evidence of myeloma at autopsy.
Suyehira and Lawrence	1989	18	1	M	49	IgG-K	17	Alive	
van Hoeven et al	1990	19	1	F	65	IgG	14	Died	Cure? Died of metastatic cancer. No evidence of myeloma at autopsy.
Present series	1991		2	2 M	50	IgG-K	10	Died	
					60	IgG-K	12	Alive	Also had colon cancer

*M = male, F = female, NS = not stated.
†CML = chronic myelocytic leukemia.
‡ANNL = acute non-lymphocytic leukemia.

relapse four years after initial treatment was reported.[18] In spite of the prolonged remission, multiple lytic bone lesions had not healed. In contrast, another autopsy-documented cure of multiple myeloma 14 years after aggressive chemotherapy was described in 1990.[19] The patient died of metastatic lung cancer.

DISCUSSION

Five-year survivals in multiple myeloma are no longer rare,[1–5] but survival for ten or more years is still unusual. We have found 48 reported cases and add two of our own (Table I). Twenty-six of these patients are men, 19 are women, and the sex is not stated in five cases. The median age at diagnosis of myeloma was 51 years. Thirty-one patients had IgG myeloma, ten had IgA, one had IgD, two had κ-light chains, one was a non-secretor, and the immunoglobulin type was not stated in four cases. Twenty-six patients are still alive, most of whom still have evidence of myeloma. At least six patients died of leukemia, and two more died of metastatic cancer. At least three patients are thought to have been cured, two of whom had no evidence of multiple myeloma at autopsy. At least three patients presented with one or more plasmacytomas.

Are there clinical or laboratory features that suggest long-term survival? Kyle found that the response to chemotherapy is the most important factor in recognizing long-term survivors.[11] The absence of any lytic bone lesions and a low myeloma protein concentration at diagnosis may identify patients likely to live for ten years or more.[20] High serum interleukin-2 levels may also predict prolonged survival.[21] Initial hemoglobin and blood urea levels as well as performance status is also related to long survival.[2,3] Most patients with ten-year survivals have prolonged unmaintained remissions, slow tumor growth, and successful treatment of relapsing disease.[15] Some of these patients can be characterized as having indolent[22] or smoldering[23] multiple myeloma.

Improved survival in myeloma in the past 20 years can be attributed to a combination of earlier diagnosis, better control of complications, and more effective chemotherapy at diagnosis and relapse. Nevertheless, the prospects for survival at ten years from diagnosis is still disappointingly small, since no plateau is observed in patients who survive ten or more years.[16] Only three of the 50 reported ten-year survivors are thought to have been cured.[13,17,19] Even long-term survivors remain at risk of death from either the disease or its complications. The most devastating occurrence is a rapidly fatal second neoplasm, particularly acute non-lymphocytic leukemia, a complication pointed out as early as 1970.[24,25] Second malignancies developed in 18% of 38 five-year survivors of multiple myeloma in one series of patients.[2]

REFERENCES

1. Kyle RA: Multiple myeloma: Review of 869 cases. *Mayo Clin Proc* 1975; 50:29–40.
2. Buckman R, Cuzick J, Galton DG: Long-term survival in myelomatosis: A report to the MRC working party on leukemia in adults. *Br J Haematol* 1982; 52:589–599.
3. Franco M, Fuzibet JG, Cassuto JP, et al: Myélome multiple avec 5 ans de survie: Etude des facteurs prognostiques initiaux. Place de la bêta-2-microglobuline. *Rev Rhum Mal Ostesartic* 1985; 52: 619–623.
4. Fuzibet JG, Bataille R, Bagarry D, et al: Survie prolongée au cours du myélome multiple: Caracteristiques de présentation et de réponse au traitment de 73 malades ayant vécu 5 ans ou plus. *Presse Med* 1986; 15:1913–1916.
5. Finish Leukaemia Group: Improved long-term survival in multiple myeloma. *Eur J Haematol* 1989; 43:385–388.
6. Pankovich AM, Griem ML: Plasma cell myeloma: A thirty year follow up. *Radiology* 1972; 104: 521–522.
7. Gomez AR: Long-term survival in multiple myeloma. *W V Med J* 1978; 74:181–183.
8. Berrebi A, Estrov Z: Twenty years follow-up in a patient with multiple myeloma. *Acta Haematol* 1981; 66:269–270.
9. Boccadoro M, Van Acker A, Carmagnola A, et al: Monitoring of a long survival myeloma patient. *Tumori* 1981; 67:571–573.
10. Franklin RM, Kenyon KR, Green R, et al: Epibulbar IgA plasmacytoma occurring in multiple myeloma. *Arch Ophthalmol* 1982; 100:451–456.
11. Kyle RA: Long-term survival in multiple myeloma. *N Engl J Med* 1983; 308:314–316.
12. Lee BJ, Myers J: Long-term survival in multiple myeloma. *N Engl J Med* 1983;308:314–316.
13. Dutcher JP, Wiernik PH: Long-term survival of a patient with multiple myeloma—A cure? A case report. *Cancer* 1984; 53:2069–2072.
14. Paietta E, Goldstein M, Papenhausen P, et al: Transformation of myeloma into Ph^1 negative CML with plasma cell antigens. *Am J Hematol* 1985; 19:289–292.
15. Alexanian R: Ten-year survival in multiple myeloma. *Arch Intern Med* 1985; 145:2073–2074.
16. Bladé J, Rozman C, Montserrat E: Long-term survival in multiple myeloma. *Arch Intern Med* 1986; 146:1637–1638.
17. Kyle RA: IgD multiple myeloma: A cure at 21 years. *Am J Hematal* 1988; 29:41–43.
18. Suyehira LA, Laurence HJ: Seventeen year survival in multiple myeloma. *Am J Hematol* 1989; 30:192–193.
19. Van Hoeven KH, Reed LJ, Factor SM: Autopsy-documented cure of multiple myeloma 14 years after M2 chemotherapy. *Cancer* 1990; 66: 1472–1474.
20. Alexanian R, Barlogie B, Dixon D: Prognosis of asymptomatic multiple myeloma. *Arch Intern Med* 1988; 148:1963–1965.
21. Cimino G, Arvisati G, Amadori S, et al: High serum IL-2 levels are predictive of prolonged survival in multiple myeloma. *Br J Haematol* 1990; 75:373–377.
22. Alexanian R: Localized and indolent myeloma. *Blood* 180; 56:521–525.
23. Kyle RA, Greiffe PR: Smoldering multiple myeloma. *N Engl J Med* 1980; 302:1347–1349.
24. Kyle RA, Pierre RF, Bayrd ED: Multiple myeloma and acute myelomonocytic leukemia: Report of four cases possibly related to melphalan. *N Engl J Med* 1970; 283:1121–1125.
25. Rosner F, Grünwald HW: Multiple myeloma terminating in acute leukemia: Report of 12 cases and review of the literature. *Am J Med* 1974; 57:927–939.

Right ventricular cardiomyopathy

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Idiopathic dilated cardiomyopathy is a disease of obscure etiology, which rarely has been reported to involve primarily the right ventricle, producing characteristic structural and electrophysiologic abnormalities. A continuum of diseases, the right ventricular cardiomyopathies include arrhythmogenic and non-arrhythmogenic right ventricular dysplasia (RVD), Uhl's anomaly, and idiopathic right ventricular dilated cardiomyopathy (IRVDC).

Until recently, the role of the right ventricle in clinical disease has been overlooked and underestimated, awaiting resolution of the technical difficulties inherent in measurement of this geometrically complex chamber.[1–3] With the verification of the near equality of the ejection fractions in the normal heart,[4] emerged the importance of the right ventricular cardiomyopathies as a cause of sudden death and cardiovascular morbidity. Diagnosing a suspected right ventricular cardiomyopathy may be difficult in cases with atypical presentations. The individual at greatest risk is an athlete with "silent" arrhythmogenic disease. It is not uncommon for a right ventricular cardiomyopathy to be overlooked in a young woman in whom symptoms are presumed secondary to an anxiety state or to a coexisting mitral valve prolapse. A high index of suspicion in the appropriate clinical setting may avert unnecessary invasive procedures, and will alert the clinician to the potentially fatal but often treatable complications of this disorder.

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TABLE I. Radionuclide Evaluation of the Right Ventricle

	Right Ventricle	Norm ± SD
Resting EF* (%)	29	51 ± 4
Stress EF (%)	32	59 ± 6
End-diastolic volume (mL)	210	79 ± 10
End-systolic volume (mL)	149	32 ± 6
Cardiac output (L/min)	2.9	
Cardiac index (L/min/m^2)	1.7	
RV/LV† end-diastolic volume	1.6	≤1.5
RV/LV end-systolic volume	3.2	≤1.8

*EF = ejection fraction.
†RV/LV = right ventricular/left ventricular.

CASE REPORT

A 40-year-old, female physician presented for evaluation of dyspnea on exertion and easy fatigability. Her medical history was significant for biopsy-proven myocarditis in 1971 associated with a five fold titer elevation in coxsackie B-2; idiopathic pericarditis in 1976 and 1989; and enterococcal endocarditis from 1989 to 1990.

The viral myocarditis had been complicated by ventricular tachyarrythmias, but at recovery in 1971, a Holter monitor disclosed no significant ventricular ectopia and the electrocardiogram was normal. During the first year after her recovery, the patient noticed mild dyspnea on exertion characterized by a persistent inability to duplicate earlier collegiate athletic performances. Medical attention was not sought for symptoms of exercise intolerance until 1983 when she was evaluated by a cardiologist and a pulmonologist. The differential diagnosis at that time included primary pulmonary hypertension and chronic pulmonary embolization. The clinicians concluded that the low probability of the aforementioned did warrant invasive testing, and the patient was discharged on no medications, with a diagnosis of mitral valve prolapse syndrome.

Subsequently, the patient noticed progressive dyspnea on exertion, and she sought treatment. On presentation, she reported no chest pain, hemoptysis, orthopnea, palpitations, presyncopy or syncopy, and she denied tobacco, alcohol, or illicit drug use. She was taking no cardiac medications. She is allergic to penicillin, which produces urticaria. On physical examination, the patient did not appear tachypneic or cyanotic. Her vital signs were within normal limits, and there was no clinical evidence of right heart failure. The point of maximal impulse was non-displaced, and a right ventricular heave was not palpable. The first heart sound (S1) was normal; there was wide respiratory splitting of the second heart sound (S2). The pulmonic second heart sound (S2) component was not accentuated, and there was no S3. An S4 gallop was heard along the left sternal border. There was a I/VI early systolic murmur heard at the apex accompanied by an intermittent systolic click. The remainder of the physical examination was unremarkable.

The electrocardiogram was unchanged from the earlier tracing in 1983, and demonstrated a sinus tachycardia, indeterminate axis, right bundle branch block pattern, and inferior peaked P waves, with a poor R wave progression from V1 to V5 (Fig 1). The chest film was normal, revealing no cardiomegaly, pulmonary artery prominence, or pruning. Arterial blood gas studies were normal, including a normal A-a gradient, and pulmonary function testing was within normal limits. A ventilation-perfusion scan was interpreted as "low probability" for pulmonary embolism. The possibility of an intracardiac shunt was excluded by a radionuclide cineangiogram shunt study, and an echocardiogram using the saline microbubble technique. A 24-hour Holter monitor revealed frequent premature ventricular contractions of a left bundle branch block morphology consistent with Lown classification II. A radionuclide cineangiogram utilizing the first pass method revealed a slightly dilated but diffusely hypokinetic right ventricle without evidence of segmental abnormality (Fig 2). Left ventricular wall motion and hemodynamic index were entirely normal. A summary of the right ventricular hemodynamic data is presented in Table I.

A two-dimensional and M-mode echocardiogram with color-flow Doppler (Interspec CFM 725) was performed and confirmed prolapse of the mitral valve with mild mitral regurgitation. No vegetations were identified, and the qualitative left ventricular ejection fraction was 68%. All left ventricular wall motion and hemodynamic indices were normal, and no pericardial effusion was visualized. Attention was directed specifically at the right ventricule (Table II), which revealed an unremarkable pulmonic valve without evidence of stenosis or regurgitation. The "a" wave was present, and there was no evidence of mild systolic closure. The tricuspid valve was in normal position and moderate regurgitation was visualized, permitting calculation of right heart pressures via the modified Bernoulli equation as follows: right ventricular (pulmonary artery) systolic pressure, 26 mm Hg (normal range, 15–30 mm Hg); mean pulmonary artery pressure, 15 mm Hg (normal range, 8–18 mm Hg); and total pulmonary vascular resistance, 225 dyne-sec cm^{-5} (normal, 210 ± 51 dyne-sec cm^{-5}. The septal diameter was normal, and paradoxical motion was not observed. Right ventricular wall motion appeared slightly hypokinetic without evidence of akinesia, dyskinesia, sacculations, diverticuli, aneurysms, or moderator band hypertrophy.

A diagnosis of right ventricular cardiomyopathy was made on the basis of noninvasive testing. No cardiac medications were prescribed as the patient was not a candidate for antiarrhythmic therapy, and vasodilator therapy to enhance functional capacity is unproven. Repeat examinations find the patient to be clinically stable 26 months after diagnosis.

DISCUSSION

In the proper setting, isolated right ventricular enlargement and/or ejection fraction depression associated with a wall motion abnormality and with no other identifiable cardiac or pulmonary malfunction is diagnostic of a right ventricular cardiomyopathy. In select cases, the diagnosis may be made using noninvasive methods such as echocardiography, a highly accurate indicator of right ventricular structural abnormalities. The morphologic stigmata of right ventricular dysplasia (RVD) are well defined,[6–7] and the validity of the noninvasive determination of right heart pressure is well established.[8–9] A shortened pulmonic valve acceleration time is the most consistent echocardiographic finding in pulmonary hypertension.[6] This determination, which is highly accurate and relatively operator-independent, here substantiates a normal right ventricular systolic pressure.

First pass radionuclide cineangiography is acknowledged as the superior method to evaluate the right ventricle and identify intracardiac shunts.[10] An underused diagnostic modality, first pass angiography has proven reproducibly accurate in the diagnosis of right ventricular disease.[11] It has been proposed[6] that normal echocardiographic and nuclear scintigraphic studies effectively exclude a right ventricular cardiomyopathy. Each technique has its relative merits and limitations, and some discrepancies are not unusual, since each method displays the anatomical data differently. In this case, correlation between the two modalities is high, leading to the diagnosis of a

TABLE II. Echocardiographic Assessment of the Right Ventricle

	Right Ventricle	Norm ± SD
RVEDD* (cm)	2.1	1.7 ± 0.4
RVESD* (cm)	1.9	1.4 ± 0.3
RV/LV† EDD	0.5	0.3 ± 0.1
RV/LV† ESD	0.7	0.5 ± 0.1
Qualitative RVEF‡ (%)	30–35	≥45
Tricuspid jet velocity (m/sec)	2.0	≤2.5
Pulmonic valve time to peak (msec)	130	137 ± 24

*RVEDD = right ventricular end diastolic diameter; RVESD = right ventricular end systolic diameter.
†RV/LV = right ventricular/left ventricular.
‡RVEF = right ventricular ejection fraction.

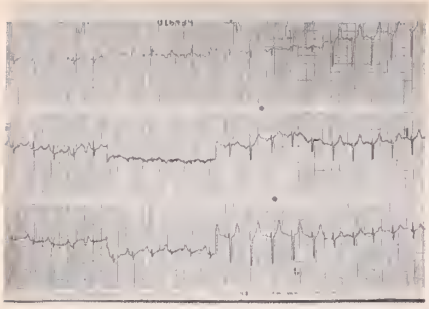

FIGURE 1. Twelve-lead electrocardiogram demonstrating sinus tachycardia, indeterminate axis, peaked P waves inferiorly, right bundle branch block pattern with a poor R wave progression V1–V5.

right ventricular cardiomyopathy, representing either IRVDC or diffuse nonarrhythmogenic RVD.

The prevalence of the right ventricular cardiomyopathies is unknown primarily because there exists no diagnostic "gold standard" sensitive enough to identify the subtle, atypical, and presumably more frequent forms. The typical patient is young (80% between ages 7–40 years)[12] and presents with complaints of dyspnea on exertion, easy fatigability, palpitations, or syncope. In some cases, the diagnosis may be established readily from the history and from noninvasive testing. Most importantly, the practitioner must maintain a high index of suspicion, since RVD or IRVDC may present at any age with a constellation of symptoms and a paucity of physical findings. A family history of sudden death is elicited in only a minority of cases. Typically, the chest radiograph and the physical examination are normal; and, the most important physical clue, if present, is a widely split S2.[13] The electrocardiogram, in the majority of cases, demonstrates T-wave inversions in the anterior precordial leads; bundle branch blocks and evidence of right atrial enlargement are variable findings. Exercise electrocardiography may precipitate ventricular tachycardia, often hemodynamically well-tolerated. If the patient is engaging in strenuous physical activity, it is important that the Holter monitor be worn during such times.

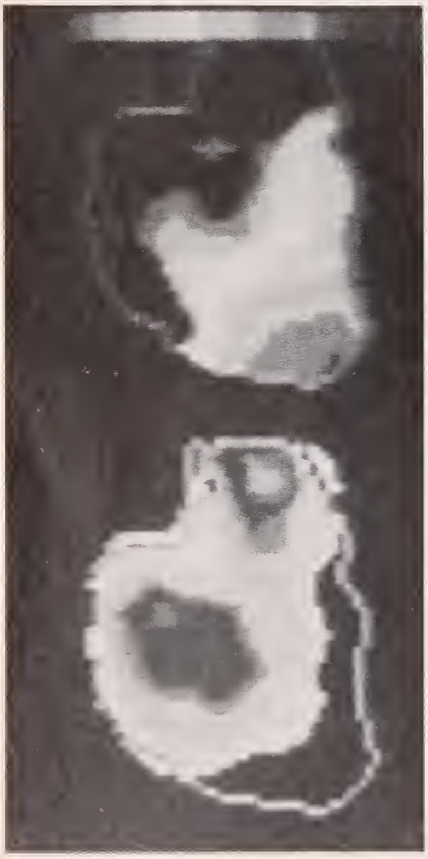

FIGURE 2. Resting first pass cineangiogram revealing a mildly dilated and diffusely hypokinetic right ventricle.

The primary clinical objective in the diagnosis of right ventricular disease is the prevention of life-threatening arrhythmias. In particular, RVD has been recognized as an important cause of sudden death in young adults,[14,15] and tragically, sudden death is often the first manifestation of disease. In the absence of specific guidelines, it would appear that the clinical evaluation is dependent on the symptomatology, arrhythmia status, probability of active myocarditis or coronary artery disease, and the lifestyle of the patient. At greatest risk of sudden death is the athlete with "silent" arrhythmygenic disease. Frequently, such individuals have normal ventricular function, permitting a deceptively high level of athletic performance in spite of underlying arrhythmic symptoms. In such cases, mandatory cardiac catheterization and electrophysiological studies have been recommended.[15]

Although it has been asserted that IRVDC and RVD are histologically distinguishable,[16] endomyocardial biopsy has many recognized limitations, especially in segmental disease. A more precise diagnosis in this case is academic in view of the similarity of therapies and clinical outcomes. Studies suggest that the right ventricular cardiomyopathies are slowly progressive unless interrupted by malignant arrhythmia.[14,17] The fact remains that 30%–40% of patients experience sudden death within a decade of diagnosis irrespective of the diagnostic label,[14,17] with the majority of survivors remaining stable on antiarrhythmic therapy or following catheter ablation of their reentry pathways. It is highly probable that right ventricular disease can arise from a variety of causes producing a kaleidoscope of fibrolipomatous myocardial lesions. Not only are the diagnostic criteria not standardized, but it also remains unclear whether "diffuse" RVD is IRVDC. Disease susceptibility, expression, and rate of progression probably relate to genetic factors, and an autosomal dominant inheritance with incomplete penetrance and variable expression has been postulated. There is evidence that viral myocarditis and dilated cardiomyopathy are intimately related in approximately 40% of cases;[18] some experimental cases of myocarditis have produced pathologic changes limited to the right ventricle.[19] In the murine model,[19] infection with coxsackie B-2 and B-3 serotypes has produced major myofiber scarring and necrosis, with survivors developing dilated cardiomyopathies.[20]

Unlike the ventricular tachycardia (left bundle branch block morphology), which may be seen in the structurally normal heart,[21] arrhythmias associated with the right ventricular cardiomyopathies are generally very resistant to calcium- or β-blockade, requiring instead class I and occasionally class III antiarrhythmics. A significant number of cases prove refractory to medical therapy, and in such cases, surgical ablation has been proven to be lifesaving.[22] Patients are not routinely placed on antiarrhythmic therapy if they are Lown classification III or less, if their ectopy is not aggravated by exercise, and if they demonstrate a normal or mildly dilated right ventricle on two-dimensional echocardiography.[12] The medical therapy for congestive symptoms has had questionable efficacy and traditionally has involved digitalis and diuretics. Theoretically, any intervention that reduces right ventricular filling pressures is potentially deleterious; and it remains to be proven that digitalis normalizes the functional inequality. As in cases of primary pulmonary hypertension, hydralazine, prozosin, and nifedipine have been administered without definitive success.

Acknowledgments. The authors acknowledge the assistance of Frances Dooley, RN, MSN, Joan Shanfeld, RN, and Linda Theisinger, RN.

REFERENCES

1. Ferlinz J: Right ventricular function in adult cardiovascular disease. *Prog Cardiovasc Dis* 1982; 25:225–254.
2. Goldman ME: Emerging importance of the right ventricule. *J Am Coll Cardiol* 1985; 5:925–927.
3. Ferlinz J: Right ventricular performance assays: Slowly coming of age. *J Am Coll Cardiol* 1989; 13:360–362.
4. Marving J, Hoilung-Carlson PF, Chraemmer-Jorgensen B, et al: Are the right and left ejection fractions equal? Ejection fractions in normal subjects and in patients with first acute myocardial infarction. *Circulation* 1985; 72:502–514.
5. Sacher HL, Miller WC, Landau SW, et al: Relapsing native-valve enterococcal endocarditis: A unique cure with oral ciprofloxacin combination drug therapy. *J Clin Pharmacol* 1991; 31:719–721.
6. Manyari DE, Duff HJ, Kostuk WJ, et al: Usefulness of noninvasive studies for diagnosis of right ventricular dysplasia. *Am J Cardiol* 1986; 57: 1147–1153.
7. Kislo J: Two-dimensional echocardiographic arrhythmic right ventricular dysplasia. *Eur Heart J* 1989; 10(suppl D):22–26.
8. Kitabatake A, Ito H, Inoue M, et al: Evaluation of pulmonary hypertension by doppler echocardiography. *Pract Cardiol* 1986; 12:136–149.
9. Berger M, Haimowitz A, Van Tosh A, et al: Quanitative assessment of pulmonary hypertension in patients with tricuspid regurgitation using contin-

uous wave doppler ultrasound. *J Am Coll Cardiol* 1985; 6:359–365.

10. O'Rourke RA, Chatterjee K, Dodge HT, et al: Guidelines for clinical use of cardiac radionuclide imaging. *J Am Coll Cardiol* 1986; 8:1471–1483.

11. Bruzzone F, Borziani S, Gaita F, et al: Right ventricular dysplasia: Radioisotopic angiography. *Eur Heart J* 1989; 10(suppl D):37–41.

12. Volta SD: Arrhythmogenic cardiomyopathy of the right ventricle: Thoughts on aetiology. *Eur Heart J* 1989; 10(suppl D):2–6.

13. Rossi PA: Arrhythmogenic right ventricular dysplasia—Clinical features. *Eur Heart J* 1989; 10(suppl D):7–9.

14. Matsui S, Murakami E, Takekoshi N, et al: Idiopathic dilated right ventricular cardiomyopathy: A report of eight cases. *J Cardiogr* 1985; 15:1027–1038.

15. Furlanello F, Bettini R, Bertoldi A, et al: Arrhythmia patterns in athletes with arrhythmogenic right ventricular dysplasia. *Eur Heart J* 1989; 10(suppl D):16–19.

16. Frustaci A: Right ventricular dysplasia vs. idiopathic dilated cardiomyopathy. *Eur Heart J* 1989; 10(suppl D):92–94.

17. Marcus Fl, Fontaine GH, Gallagher JJ, et al: Long-term follow-up in patients with arrhythmogenic right ventricular dysplasia. *Eur Heart J* 1989; 10(suppl D):68–73.

18. Olsen EJ: The value of endomyocardial biopsies in myocarditis and dilated cardiomyopathy. *Eur Heart J* 1991; 12(suppl D):10–12.

19. Thiene G, Corrado D, Nava A, et al: Right ventricular cardiomyopathy: Is there evidence of an inflammatory etiology? *Eur Heart J* 1991; 12(suppl D):22–25.

20. Lerner AM: Viral myocarditis as an incidental discovery. *Hosp Pract* 1990; 6:73–81.

21. Ashan AJ, Cunningham D, Rowland E: Low energy catheter ablation of right ventricular outflow tract tachycardias. *Br Heart J* 1991; 65:231–233.

22. Leclercq JF, Chouty F, Cauchemez B, et al: Results of electrical fulguration in arrhythmogenic right ventricular disease. *Am J Cardiol* 1988; 62:220–224.

FROM THE LIBRARY

TUBERCULOSIS CONTROL

The attitude of practicing physicians to tuberculosis was the subject of serious discussion by the Committee on Public Health and Medical Education of the Medical Society of the State of New York, at its meeting on January 18th in the Hotel Pennsylvania.

The County Medical Society is the natural organization of physicians to assume the leadership and to advise the other groups engaged in tuberculosis work. The first activity of the Medical Society of the State of New York will be to influence the county medical society of each county to engage in tuberculosis work, and to assist the doctors to develop lines of work suited to that county.

EDITORIALS
(*NY State J Med* 1930; 30:149–150)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Trauma-related presentation of non-Hodgkin's lymphoma

TO THE EDITOR: The role of trauma in the causation of cancer is unproven and controversial.[1] The tumors that are remotely associated with trauma are osteogenic sarcoma, meningioma,[2] and testicular cancer.[3] Less frequently, trauma may indirectly lead to a diagnosis of malignant neoplasm by becoming the site of metastatic growth. We report here such a case where a traumatic hematoma of the scalp brought to clinical attention a malignant lymphoma.

Case Report In January 1990, a 48-year-old white man presented because of a scalp mass. The patient had sustained head injury during an automobile accident in August of 1989, and a hematoma developed at the site of the trauma. A computed tomography (CT) scan the head demonstrated a subgaleal hematoma and an underlying small subdural hematoma was observed (Fig 1). Microscopic examination of the aspirated material showed only red blood cells and inflammatory cells. During the weeks that followed, serial CT scans of the head showed no progression of the subdural hematoma; however, the subgaleal hematoma persisted in spite of repeated aspirations. By December 1989, the site of trauma became a large scalp mass that was biopsied. The biopsy revealed a large cell non-Hodgkin's lymphoma (Fig 2).

The patient had no significant medical or surgical history prior to his head trauma. A review of systems revealed no further evidence of fever, chills, weight loss, visual or neurological deficits.

On physical examination, the scalp showed a large, hard mass in the left parieto-occipital region that measured 14 cm in diameter. There was also evidence of cervical and left axillary lymphadenopathy. The remainder of the examination was remarkable. His laboratory workup was non-contributory, and the CT scan of the chest was normal; however, the abdominal CT scan revealed a left renal mass that measured 7 cm in diameter. Fine-needle aspiration of the mass yielded rare malignant cells. A left nephrectomy was performed because of the suspicion of renal cell carcinoma. Pathological examination of the surgical specimen showed diffuse, large, large-cell malignant lymphoma encasing the renal hilum and invading the renal parenchyma. The patient responded initially to chemotherapy and local radiotherapy to the scalp; however, his scalp mass and his lymphadenopathy recurred after the chemotherapy, and he was referred for an autologous bone marrow transplant, after intensive chemotherapy.

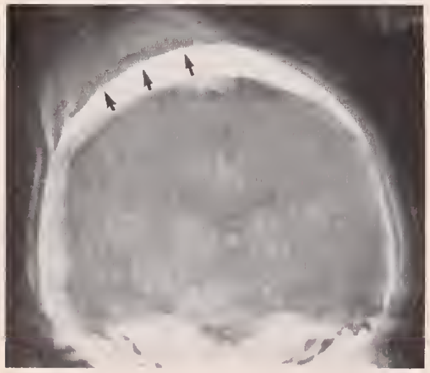

FIGURE 1. CT scan showing large subgaleal hematoma.

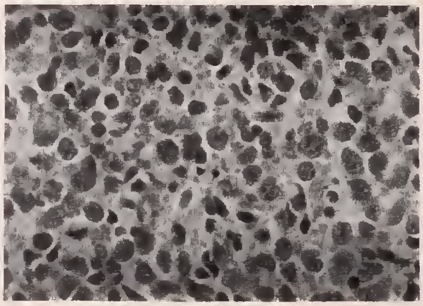

FIGURE 2. Photomicrograph showing pleomorphic diffuse large-cell lymphoma.

Discussion It is not uncommon for trauma to bring to medical attention the primary neoplasms of organs such as the testicles, the skeleton, and so forth. Rarely, however, the site of trauma may become the focus of a metastatic growth of a primary neoplasm of a distant organ. The neoplasm may have been previously unknown or may have been treated in the past. This usually represents an anecdotal case report or a personal communication and includes neoplasms, such as renal cell carcinoma and squamous cell carcinoma of the uterine cervix.

Our case documents such an instance where the trauma-related subgaleal hematoma was colonized with lymphoma cells. The potential of a traumatic site to act as a favorable medium for metastatic growth should be kept in mind whenever such a site follows an atypical clinical course, so not to delay appropriate investigation including microscopic examination.

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1. Holland JF, Frei E: Etiology of Cancer: Trauma and Inflammation, in Holland JF, Frei E (eds) *Cancer Medicine*, ed 2, Lea & Febiger, 1982, pp 120–123.
2. Walshe F: Head injuries as a factor in the etiology of intracranial meningioma. *Lancet* 1961; 2: 993–995.
3. Swerdlow AJ, Huttly SR, Smith PG: Is the incidence of testicular cancer related to trauma or temperature. *Br J Urol* 1988; 61:518–521.

Chemotherapy for hepatic angiosarcoma

TO THE EDITOR: Limited clinical experience is available regarding specific chemotherapy for hepatic angiosarcoma.[1–4] We report here our experience with one patient who was effectively palliated with mesna, doxorubicin, ifosfamide, and dacarbazine (MAID regimen).

Case Report A 42-year-old white male presented in August 1987 with two weeks of progressive right upper quadrant pain. He had experienced a 20-lb weight loss over several weeks and temperatures of 99–101°F. He had no previous liver disease and no known exposure to vinyl chloride, thorotrast, or other toxins. He had two to six mixed drinks a day and smoked one to one-and-a-half packs of cigarettes a day for 21 years. He denied risk factors for the human immunodeficiency virus.

The physical examination revealed a temperature of 101.2°F, a pulse of 88/min, respirations of 20/min, a blood pressure of 110/70 mm Hg, and weight of 136 pounds. Head, ears, nose, and throat were normal. There was no adenopathy. The heart rhythm was regular, without a murmur. The lungs were clear bilaterally. The abdomen was soft, with bowel sounds present; the right upper quadrant was mildly tender, and the liver extended 12 cm below the right costal margin.

The complete blood count showed a Hgb of 11.4 gm/dL, Hct of 34.5%, Wbc of 13,200/cc, with 82% polys, 15% lymphs, and 5% monos. The platelet count was 241,000/cc. Chemistry studies showed a total bilirubin of 0.2 mg/dL, SGOT of 73 units/1, SGPT of 70 units/L, alkaline phosphatase of 306 units/L, GGTP of 161 units/L, LDH of 449 units/L, total protein of 6.2 gm/dL, albumin of 3.1 gm/dL, BUN of 14 mg/dL, and creatinine of 0.7 mg/L. The CEA was 2.4 U/L and the α-fetoprotein was undetectable. The prothrombin time was 13.1 sec, and the partial prothrombin time was 34.5 sec. The chest x-ray, EKG, and urinalysis were all normal. An abdominal sonogram showed a large liver containing multiple soft tissue masses. An abdominal CAT scan demonstrated a large liver containing multiple masses; the largest mass was in the left lobe, measuring 13 × 11.5 cm. The biliary system was normal; the pancreas and stomach were somewhat compressed by the liver; the kidneys, spleen, and adrenals were normal; there was no adenopathy (Fig 1). A mesenteric arteriogram showed multiple hypervascular masses in the liver, with evidence of tumor neovascularization. An exploratory laparotomy with wedge liver biopsy revealed tumor in the liver, with no other intra-abdominal disease apparent. Pathologic examination of the biopsy showed hepatic angiosarcoma of intermediate grade.

The patient was treated with the MAID regimen, as follows: mesna 2.5 gm/m^2/day, days 1–4; doxorubicin 15 mg/m^2/day, days 1–4; ifosfamide 2.5 gm/m^2/day, days 1–3; dacarbazine 250 mg/m^2/day, days 1–4. Doxorubicin and dacarbazine (DTIC) were given in the same bottle by continuous infusion through a central line. Ifosfamide and mesna were given in the same bottle by continuous infusion through a peripheral line. Cycles were given every 21-days, at the highest tolerable doses. Appropriate dose modifications were made for hematologic, neurologic, and other toxicities.[5]

Treatment was well tolerated, with no life-threatening toxicity. Occasional transfusions of packed red blood cells and platelets were required. Antiemetics were given to control nausea, and acetaminophen was given to avoid the flu-like syndrome associated with DTIC.

The patient responded well to treatment, with a gradually increasing level of well being, normalization of temperature, and weight gain back to baseline of 155 lbs. After seven cycles of therapy, the liver function tests were normal: total bilirubin, 0.3; alkaline phosphatase, 131; SGOT, 26; SGPT, 18; GGTP, 76; and LDH, 177. The largest tumor measured 8 × 7 cm, a decrease of greater than 50% in two dimensions, thus making this a partial response (Fig 2).

The patient had two additional cycles of chemotherapy. Follow-up CAT scans, however, showed a minimal increase in the size of the largest tumor to 9 × 7.5 cm and an increase in a second smaller tumor from 4 × 3 cm to 6.5 × 5.2 cm. Because of apparent developing resistance to chemotherapy, the patient elected to undergo liver transplantation. Pre-operative evaluation, including CAT scans of the brain, chest, abdomen, and pelvis; bone scan; and bilateral bone marrow biopsies showed the tumor was still confined to the liver. He underwent autologous liver transplantation June 9, 1988. Unfortunately, the tumor recurred, with multiple small lesions noted in the liver and lung in September 1988. Fine-needle aspiration of a lung lesion was positive for angiosarcoma. The tumor progressed rapidly, was unresponsive to methotrexate or VP16, and the patient expired with widespread metastatic disease November 19, 1988.

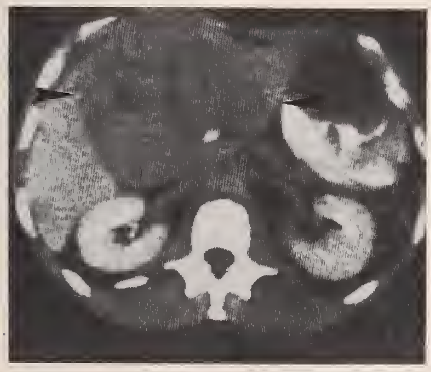

FIGURE 1. Abdominal CAT scan showing multiple liver masses. Largest mass (T1) measures 13 × 11.5 cm.

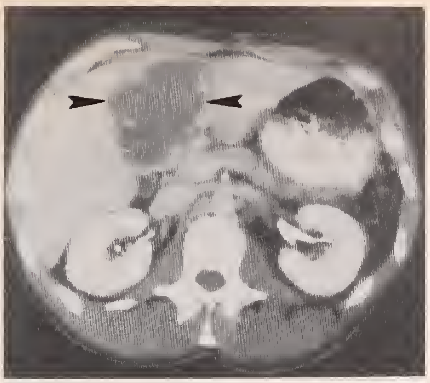

FIGURE 2. Abdominal CAT scan after seven cycles of chemotherapy. T1 measures 8 × 7 cm.

Discussion This case suggests that the MAID regimen has significant activity in the treatment of hepatic angiosarcoma. The patient had a partial response, with excellent clinical palliation. Additional cases will further define the effectiveness of MAID in the treatment of this rare tumor.

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1. Dannaker CL, Tamburro, CH, Yam LT: Chemotherapy of vinyl chloride associated hepatic angiosarcoma. *Cancer* 1981; 47:466–469.
2. Louagie YA, Gianello P, Kestens PJ, et al: Vinyl chloride induced hepatic angiosarcoma. *British J Surg* 1984; 71:322–323.
3. Noronha R, Crussi-Gonzalez F: Hepatic angiosarcoma in childhood. *Am J Surg Pathology* 1984; 8:863–871.
4. Weinman MD, Chopra S: Tumors of the liver, other than primary heatocellular carcinoma. *Gastroenterol Clin North Amer* 1987; 16:627–631.
5. Elias A, Ryan L, Sulkes A, et al: Response to mesna, doxorubicin, ifosfamide and dacarbazine in 108 patients with metastatic or unresectable sarcoma and no prior chemotherapy. *J Clinical Oncology* 1989; 7:1208–1216.

New hospice benefit available for nursing home residents

TO THE EDITOR: Recent Federal and State legislation has been enacted that allows certified hospice programs to provide "home care" services to nursing home residents. This was based on the interpretation of the nursing home as "home" of the resident. If a nursing home resident becomes terminally ill, he or she may elect to receive services from a certified hospice program that has a contract with the nursing home. There residents would be able to receive the combined benefit services from both the nursing home and the hospice. The New York State Department of Health must approve each contract, which must indicate exactly how the program would operate in compliance with federal and state regulations.

The goal of hospice care in a nursing home is to assist the staff in palliation of symptoms and to help patients, families, and staff make appropriate treatment decisions in anticipation of the patient's final days. The patient's primary physician can continue to direct care and prescribe appropriate medication in collaboration with the hospice and nursing home team.

For a nursing home, the incentive to use hospice services is the possibilty of enhanced patient care in a terminal illness through supplemental staffing and supportive services. In addition, patients can be managed without resorting to hospital admission in a medical crisis.

Specific services provided by the hospice can include the following:

1. Nursing visits to effect symptom control.
2. Around-the-clock private duty nursing care during periods of crisis.
3. Short-term inpatient admission to a hospice unit or an area under hospice care when hospitalization is required.
4. Medical consultation (if needed).
5. Pharmaceuticals and biologicals.
6. Emotional and spiritual counseling for the resident and family as well as the nursing home staff and other residents.
7. Other appropriate therapies.
8. Volunteer supportive visits.
9. 24-hour access by phone to hospice staff.
10. Bereavement counseling for the resident's family, the nursing home staff, and other residents.

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The role of third party payors in medical care

TO THE EDITOR: The setting of medical fees by third party payors such as Medicare, Medicaid, and HMOs is distasteful. However, I find it intolerable when these fiscal intermediaries also determine diagnostic and treatment procedures.

I have experienced the following sequence of events with several patients. A patient presents with a dark-pigmented lesion that has been increasing in size over time and which may have bled on occasion. Obviously, one must consider the possibility of malignant melanoma in the differential diagnosis. A biopsy is taken, usually with the removal of the entire lesion. I and the patient are relieved when the pathology report returns with a diagnosis of seborrheic keratoses. However, when the insurance forms were submitted one third party payor refused to cover the procedure claiming that seborrheic keratoses are benign lesions and removal is unnecessary.

I was informed by a physician representing one third party payor that it was their policy not to pay for the removal of seborrheic keratoses. I pointed out that if everyone adhered to this policy, one or more instances of malignant melanoma would be missed. I asked him if he and his organization would be co-defendants in the inevitable malpractice suits that would follow. His only reply was that I was being "impertinent." My association with this company, terminated shortly afterwards, by mutual consent.

It strikes me that we have a serious situation in which a third party payor dictates therapy in the absence of seeing or examining the patient. If we as physicians give up the responsibility and the right for diagnosis and treatment, we will become little more than clerks and technicians in a large bureaucracy.

While the former Communist countries of Europe are discarding centralized bureaucracies, we seem to be in the process of building one.

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RECONSTRUCTION OF THE CHILD'S HAND

Edited by Peter R. Carter, MD. 321 pp, illustrated. Malvern, Pa, Lea & Febiger, 1990. $75.00 (hardcover)

This book grew out of a series of papers on problems of reconstruction of the child's hand presented at a three-day meeting in September 1986 at the Texas Scottish Rite Hospital for Crippled Children in Dallas. The meeting was attended by Dr J. William Littler, honored guest, sixteen surgeons, a pediatric anaesthesiologist, and a geneticist. Dr Peter Carter, the meeting chairman, has edited this volume composed of formal chapters contributed by each of the meeting's participants.

The goal of the book, as described by Dr Carter in the preface, is not to be an "exhaustive or complete presentation of all treatments," but rather "a more informal discussion and focus on new developments" in the field of pediatric hand surgery. Although the book seems to succeed in achieving neither the former nor the latter, it is nevertheless, a well-written, understandable, appropriately illustrated, and professionally presented compendium broadly covering the subject.

The book primarily covers various aspects of congenital malformations, as well as anaesthesia, trauma, rheumatoid arthritis, and new techniques. The section on malformations is comprised of ten chapters each dealing with its subject in a different style: exhaustive, formal, informal, to name a few. This can be distracting and disconcerting to the reader. The chapters on growth and development and on camptodactyl and clinodactyly are particularly well-written, while the chapter on genetic counseling is short and unrewarding. The section on trauma, although covered somewhat briefly, shows the expertise and maturity of the contributors, something of particular value to the reader. The chapters on brachial plexus palsy and the burned hand were especially worthwhile, although much has been written on these subjects elsewhere.

What is best about this book is also the most frustrating. The panel of contributors are among the most knowledgeable and respected in the field. It is clear from the sense of their writings that their collective experience is vast. The reader catches glimpses of this throughout the book. However, the reader does not get a complete look.

The editor states in the preface that the small size of the meeting group encouraged frank discussion. Surely what was said must have revealed a unique and especially rich insight into the problems of reconstruction of the child's hand. While formally presenting one subject, a participant may make the most salient and sagacious remarks about another. Those comments are forever lost to the reader, given the framework of the book. What the book does not say is its greatest weakness. Possibly a short editor's note at the end of each chapter could have satisfied the reader's curiosity.

All in all, however, the book is a good one. It is quite readable and full of valuable insights into treating a range of difficult and challenging problems. It certainly could be of interest to an accomplished hand surgeon and would provide a hand surgery fellow or senior resident with the hard-to-find perspective of a group of experienced and well-respected hand surgeons. I enjoyed the book; it will not gather dust on my bookshelf.

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A HISTORY OF CONTRACEPTION: FROM ANTIQUITY TO THE PRESENT DAY

By Donald W. Helbig, MD. Angus McLaren, 275 pp. Cambridge, Mass. Basil Blackwell, Inc, 1991. $36.95 (hardcover)

Most people, including many physicians, if they think of the history of contraception at all, tend to assume that attempts to prevent conception are a modern phenomenon. Nothing could be further from the truth. The probable reason for this misunderstanding is that contraception is seen by most people as encompassing devices, pharmaceutical products, and coitally related behaviors designed to prevent conception. In fact, our species has probably always attempted to control fertility in one way or another, including trying to avoid conception at the time of sexual intercourse, by terminating pregnancies, by infanticide (overt or masked), or by conscious and not so conscious (cultural) behaviors intended to limit exposure to the risk of possible pregnancy.

A History of Contraception by Angus McLaren goes a long way toward correcting our modern day narrow perspective. The author is currently Professor of History at the University of Victoria in British Columbia. Because of his education in the social sciences, he takes a much broader view of contraception than would most medically trained people. Perhaps the title would have been less misleading if it had been *A History of Fertility Control*. Be that as it may, *A History of Contraception* is a fascinating book because of this broader approach. Certainly, the physician reader will find his or her perceptions expanded. In the author's words, he has "... drawn on demographic, medical, literary, religious, family, and women's history to show the complex ways in which reproductive decision making was entangled (throughout history) in a web of social, cultural, and gender relationships" (p4).

The book is organized into seven chapters plus an Introduction and a Conclusion. Chapter 1, "The Patterning of Fertility in Ancient Greece," and Chapter 2, "Fertility Control in Rome," describe in depth and contrast the family structures, marriage perceptions, beliefs, and behaviors of these two classical civilizations as they pertained to fertility decisions and outcomes. Chapter 3, "Abortion and Contraception in the Christian West," focuses heavily on the influence of Augustine and the early church, pointing out among other observations that the evolution of attitudes regarding sex and reproduction evolved from Stoic philosophy and that ". . . Christians did not so much create this new mortality as act as its conduit" (p76). Chapter 4, "Procreation in the Middle Ages" attempts to correct the conclusions of earlier historians of contraception such as Norman Himes that ". . . in the Dark Ages the limitation of pregnancies would not have been practiced" (p102). The author points out that in spite of strong pronatalist policies by church and state, many women did not, in fact, have large families and that the strong sanctions against abortion and contraception of the times almost certainly meant that these fertility limiting practices were in use. Chapter 5, "Fertility Control in Early Modern Europe," traces the transition toward modern times and observes that "The state began to replace priests in policing motherhood by criminalizing infanticide and abortion, male obstetricians commenced their campaign to replace midwives in the delivery of babies, and writers of tracts on onanism and producers of condoms in effect launched the "commercialization of contraception" (p14). Chapter 6, "Neo-Malthusianism and the Fertility Transition," and Chapter 7, "The Triumph of Family Planning," complete the transition to the modern world and dwell in fascinating detail on such subjects as the eugenic focus of early pioneers in the family planning movement.

It is difficult for this reviewer, a physician and not a historian, to judge the accuracy and completeness of this book's contents. It should be clear, however, that the reader who hopes to find detailed descriptions of the origin, introduction, and history of specific contraceptive methods will be disappointed. Furthermore, the focus is entirely on the history of what we now call the Western world. Nevertheless, given the limitations, for the reader interested in learning about the complex evolution of social attitudes and behaviors regarding fertility control in the Western world, this book is a gold mine of fascinating, mind-expanding insights. For such readers, I recommend it highly.

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INFECTIOUS RISKS IN SURGERY

Edited by Richard J. Howard, MD. 294 pp, illustrated. East Norwalk, CT, Appleton & Lange, 1991. $49.95 (hardcover)

This volume by Richard J. Howard, MD, "... attempts to answer questions surgeons may have about HIV and hepatitis infection." Dr Howard is a professor of surgery at the University of Florida College of Medicine in Gainesville, Florida. Chapters are written by other contributors who have diverse medical, public health, and legal backgrounds.

This book should provide valuable information regarding the risks faced by surgeons and other health care workers in the care of the surgical patient who may be infected with HIV and the various agents of viral hepatitis. In addition, it will serve as a resource in surgical literature for the discussion of issues related to patient and health-care-worker screening for HIV and hepatitis.

It consists of 294 pages and is divided into 11 chapters and five appendices. Topics include infections transmitted by transfusion, a general overview of the HIV epidemic, and a discussion of selected clinical aspects of AIDS. Later chapters address infectious risks faced by surgeons and other health care workers when they care for patients with HIV and hepatitis infection. Finally, legal and ethical issues, including HIV screening of patients and health care workers, are reviewed.

The appendices consist of reprints from *Morbidity and Mortality Weekly Reports*, dealing with definitions of HIV-related diseases, and Centers for Disease Control (CDC) recommendations, regarding transmission of HIV and hepatitis immunization practices (for hepatitis and occupational exposures to HIV).

Overall, *Infectious Risks in Surgery* is clearly written and adheres to the author's stated objectives. The flow of topics is not entirely logical, but the content is accurate and authoritative. Adequate numbers of tables, figures, and photographs are included with the text. Chapter bibliographies are thorough and current.

The material in the appendices is of lesser value. Appendix A provides reprints from 1986 that are outdated by their references to "HTLV-III/LAV" infection. Appendices D and E are more timely, with 1990 recommendations from the CDC regarding hepatitis immunization practices and occupational exposure to HIV. The issue of postexposure prophylaxis with zidovudine is also covered.

In summary, *Infectious Risks in Surgery* is a somewhat limited but valuable addition to the surgical literature regarding viral hepatitis and HIV infection. It will offer guidance to surgeons and other health care professionals in the difficult area of providing care for patients infected with these transmissible agents.

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DEPRESSION IN THE MEDICALLY ILL: AN INTEGRATED APPROACH

By Gary Rodin, MD, John Craven, MD, and Christine Littlefield, PhD 368 pp. New York, Brunner/Mazel Publishers, 1991. $43.50 (hardcover)

The three authors of this volume have written a definitive study as of 1991 on the difficult problem of depression in the medically ill. The book presents in three sections a thorough review of assessment and diagnosis of depression in medically ill patients, a review of current knowledge of the etiology and pathogenesis of depression, and a summary of current treatments of depression when it is identified in the medically ill. The authors have done an exhaustive review of the literature and have evaluated a large number of studies on "Assessment and Diagnosis." They point out the difficulty of making a clear-cut diagnosis and illuminate the methodological problems research in this area presents. Their assessment of large numbers of studies includes both the useful and helpful aspects of each study and the questions and problems the studies present to the practitioner.

In reviewing this book as a psychiatrist, I found it to be extraordinarily useful both as a resource and as a practical compendium of how to identify and deal with depressive illness in medical patients. Although aimed at internists, psychiatrists will benefit by reviewing the various aspects of depressive illness in medical patients, a condition on which they will often be asked to consult. The chapters are well delineated, the style accessible and clear, and the judgments balanced. The authors manage to cover an amazingly large area of complex data and to distinguish between various aspects of depression for an excellent overview of the problem. In the second section of the book, called "Etiology and Pathogenesis," thorough discussions are presented of the nature of "affect;" of the psychological, the social, and the biological factors involved in depression; and of our current knowledge of these factors and their relevance in producing depression in medical patients. I know of no current work that covers as much ground and presents the material with such clarity as this volume. I recommend it to readers in all medical specialties, including psychiatry, as a basic resource that will continue to be useful for years to come.

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FUNDAMENTALS OF PSYCHOANALYTIC TECHNIQUE

By R. Horacio Etchegoyen, PhD. 863 pp. New York, Brunner/Mazel Publishers, 1991. $100.00 (hardcover)

The author of this book is a recognized psychoanalyst and psychoanalytic scholar. He has taken on the monumental task of providing a comprehensive review of the literature. He often quotes or summarizes works originally published in other languages more comprehensively than the authorized translations. He then distills his own views of psychoanalytic technique. This book is intended for specialists in the field. It is meant not only to be read, but also to serve as a reference work in such areas as transference, countertransference, interpretation, acting out, and the negative therapeutic process. There is a brief section on the psychoanalytic interview. McKinnon and Michels have written a more comprehensive work on the psychiatric interview including the understanding of psychodynamics.

American analysts might have some difficulty with the diagnostic categories, essentially including the "classic neurosis and psychosis." We have begun to think more in terms of the DSM III R Categories of personality disorders, anxiety disorders, depression, and a variety of psychoses. What is also missing is any discussion of the role of psychotropic medication in concert with psychoanalysis and its ramifications in the transference and countertransference. Etchegoyen a self-proclaimed Kleinian analyst has ignored Erikson, the ego psychologist; Piaget, the learning theorist; and Stein, the observer of infant behavior. The work of these scholars contributes to the reality of mother/child relationships, contrasting with Kleinian psychology focusing on the inner fantasies of the child (ID psychology), and places the capacity for abstractions, thought formation, and verbal comprehension earlier in development.

It is also worth noting that there are a growing number of psychoanalysts who would not agree that the ascendancy of heterosexuality is a determinant of psychoanalytic termination. They do not consider homosexuality a perversion, to be analyzed away.

These criticisms are minor quibbles. Analysts must be knowledgeable of the wide array of literature and clinical studies. This book is a treasure for the clarity and breadth of discussion put between its two covers. It is a very technical landmark contribution to psychoanalysis.

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A CASEBOOK IN PSYCHIATRIC ETHICS

Formulated by the Committee on Medical Education. By Group for the Advancement of Psychiatry. 128 pp. New York, Brunner/Mazel Publishers, 1990. $14.95 (softcover)

This compact softcover book was put together by the Committee on Medical Education of the Group for the Advancement of Psychiatry (GAP). This group, formed in 1946, is made up of many prominent psychiatrists. One of the goals of GAP is ". . . to collect and appraise significant data in the fields of psychiatry, mental health, and human relations." In dealing with issues involving psychiatric ethics, the authors have put together an interesting and understandable text.

The book is divided into seven chapters. Subjects include confidentiality, paternalism, collegial relationships, sexual attraction toward patients, and valid consent and refusal. The chapters in turn are divided into case presentations and discussions and are followed by a series of questions that are used to clarify further ethical issues. All of the cases reflect areas of concern to psychiatry and the need for a general understanding of patient care. The cases are presented in a clear and understandable manner.

A book of this nature is vitally necessary for a better understanding of issues critical to psychiatry, and although it is short in length, it is potent in the discussion of material that it presents.

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RESOURCES FOR PEOPLE WITH DISABILITIES AND CHRONIC CONDITIONS

Resources for Rehabilitation. 215 pp, Lexington, Mass, 1991. $44.95 plus $5.00 for shipping and handling (softcover)

This catalog of publications and organizations providing information to assist persons with a variety of disabling conditions is one of several similar publications produced by Resources for Rehabilitation, Inc. Resources for Rehabilitation, Inc. is a non-profit organization whose self-stated purpose is ". . . to provide training and information to professionals and the public about the needs of individuals with disabilities, and the resources available to meet those needs." The book is intended to be used by health care professionals, individuals with disabilities and their caretakers and families. It can be used as a source of general information about the disease processes and disabilities, or to locate national and community resources to assist the disabled individual to attain and maintain the highest level of independence possible.

The book is organized into 11 sections. The initial four sections of the book deal with general topics regarding disability, various aspects of rehabilitation services, and government policies and legislation regarding the rights of the disabled. The remaining sections pertain to specific disabilities: communications disorders, diabetes, epilepsy, low back pain, multiple sclerosis, spinal cord injury, and visual impairment and blindness. Each section consists of a brief, referenced, discussion of the topic or impairment, followed by an annotated listing of organizations, publications, and tapes pertaining to the topic.

The discussions of impairments are written to be understood by the average adult reader. In an attempt to simplify, however, the sections on diabetes, epilepsy, and low back pain may be misleading. The section on diabetes may lead a nonmedical reader to believe that type II diabetics never require insulin or that all diabetics who use insulin are classified as type I. The section on epilepsy outlines a progression of seizure management in which a person experiencing a new onset of seizures would not have a diagnostic work-up unless initial attempts at pharmacologic control were unsuccessful. It is possible this approach would delay diagnosis of progressive or potentially treatable etiology of new onset seizures. Finally, in the section devoted to low back pain, information regarding the ability of infrared light to penetrate the skin as compared with application of hot packs is somewhat misleading. More important, though, the statement, "Although exercise is frequently prescribed, there is little scientific evidence to validate its effectiveness . . ." may discourage an already difficult patient population of back pain sufferers from exercising. In general, in the sections discussing physician specialists, the role of the physiatrist is underemphasized.

In spite of the above concerns, this book is a useful compilation of resources for a wide range of disabilities. It could be quite useful to both medical personnel, who manage patients with physical disabilities, and the disabled themselves.

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AN APPRECIATION

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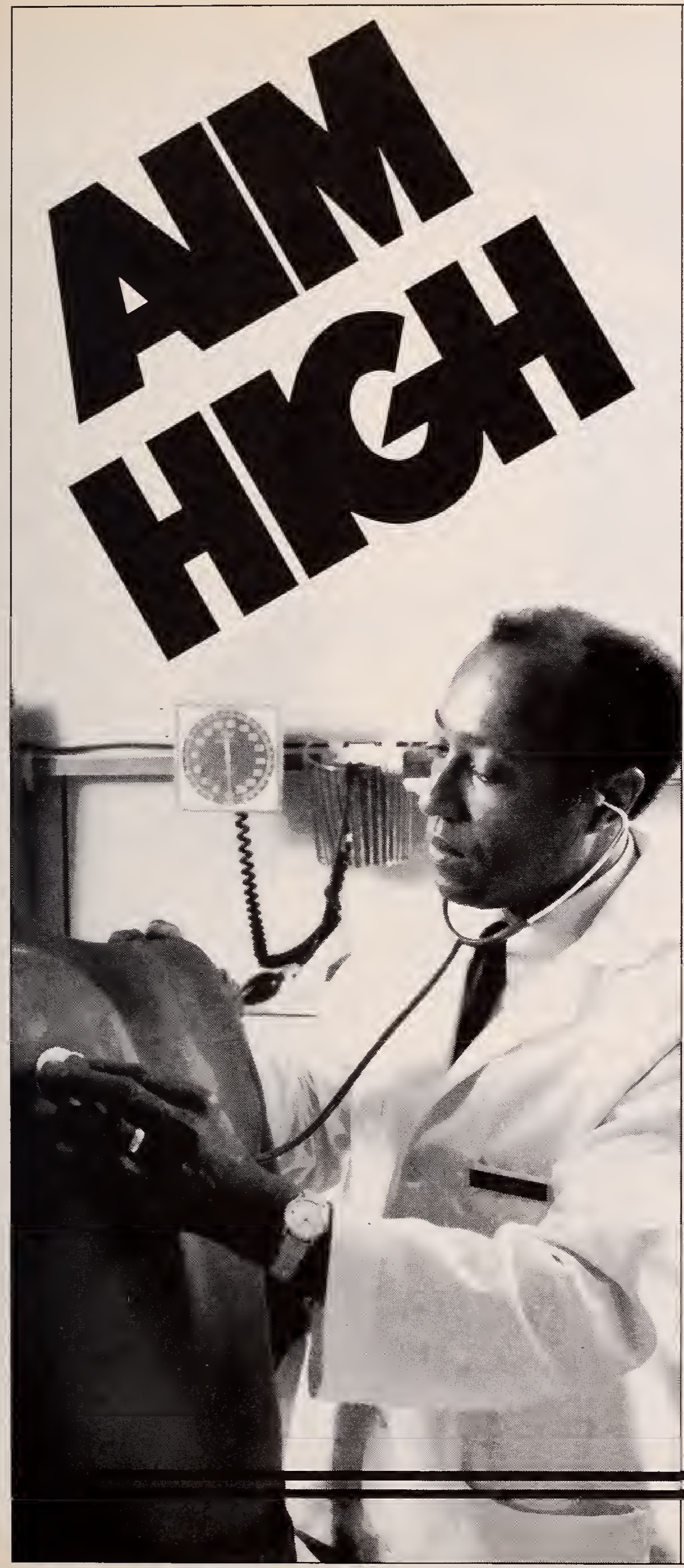

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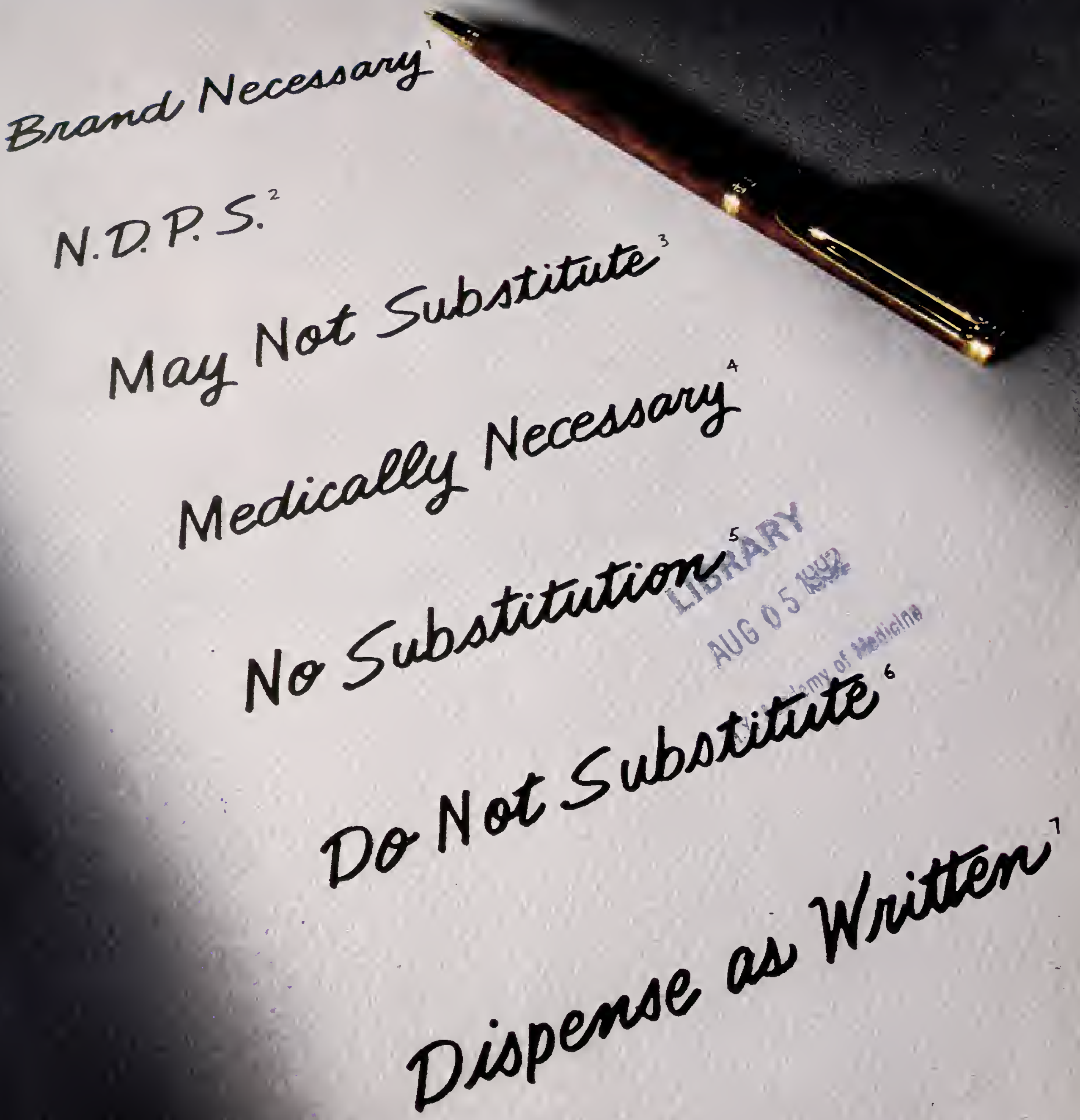

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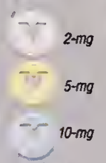

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VOL 92
NO 8
AUGUST
1992

A PUBLIC HEALTH CAMPAIGN FOR NYS

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HEALTH CARE FOR SEAMEN IN THE PORT OF NY: PART I

REGULATED MEDICAL WASTE

ALCOHOLISM-INDUCED BONE NECROSIS

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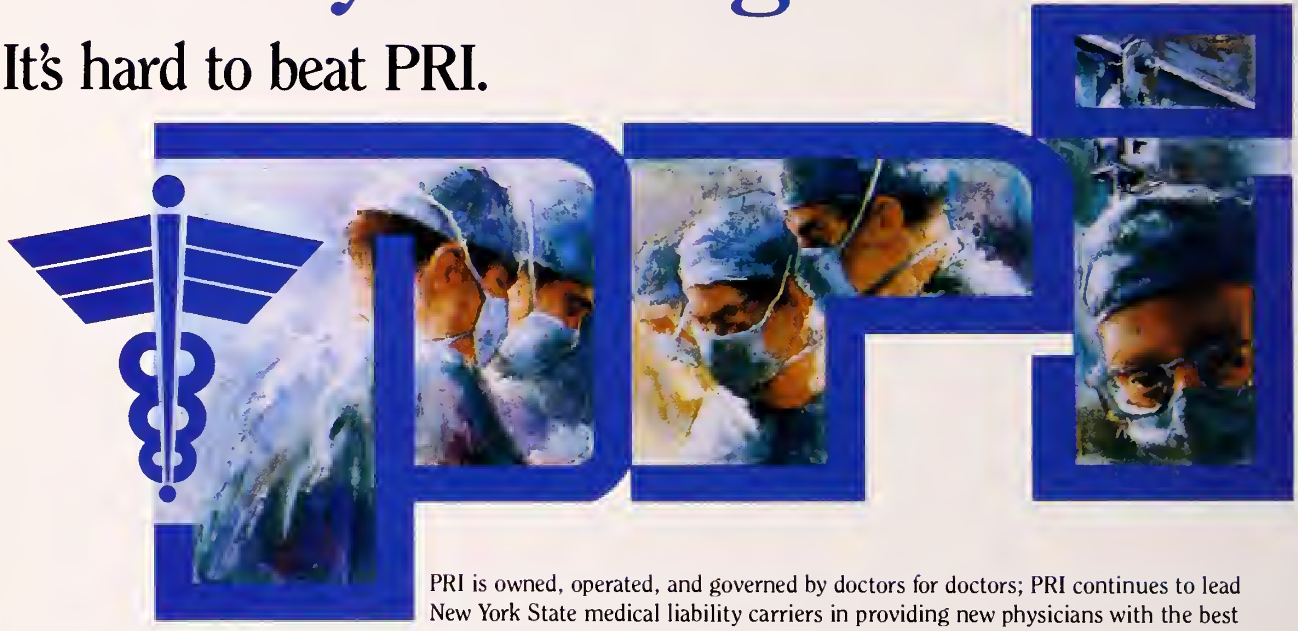

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New York State Journal of Medicine

AUGUST 1992 / Volume 92, Number 8

Contents

COMMENTARIES

329 **A public health campaign for New York State**
LLOYD F. NOVICK, MD, MPH

331 **The United States public health service and merchant seamen**
EDWARD J. HINMAN, MD

332 **Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome**
JENNIFER L. STOTKA, MD

RESEARCH PAPERS

334 **Alcoholism-induced bone necrosis**
BERNARD JACOBS, MD

338 **Lead screening and follow-up in an urban pediatric clinic**
DAVID C. CONE, MD

CLINICAL CONFERENCE

343 **Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman**

SPECIAL ARTICLE

349 **Regulated medical waste: Is any of it infectious?**
SEAN F. X. DUGAN, LLM

HISTORY

353 **Health care for seamen in the Port of New York—Part I: Recognizing the special needs of seamen**
FLORENCE KAVALER, MD, MPH; SHIRLEY A. ZAVIN, PhD

(continued on p 4A)

MEETINGS AND LECTURES

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SEPTEMBER 1992

AROUND THE STATE

EAST ELMHURST

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Sept 25–26. **Emergency Medicine Oral Board Review Course.** LaGuardia Marriott Hotel. Contact: Office of Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

AROUND THE NATION

CALIFORNIA

Sept 8–11. **The Second World Association of Sarcoidosis and Other Granulomatous Disorders Meeting.** Los Angeles. Contact: Dr Om P. Sharma, Room 11-900, LAC/USC Medical Center, 1200 N. State Street, Los Angeles, CA 90033. Tel: (213) 226-7923.

Sept 9–11. **Eighth Annual Sonography Update.** *Sept 12–13.* **Sonography Registry Review.** Hotel Del Coronado, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

Sept 10–13. **Uveitis and Retinal Frontiers.** Carmel Valley Ranch Resort, Carmel. Contact: Extended Programs in Medical Education, Room LS-105, University of California, San Francisco, CA 94143. Tel: (415) 476-4251.

PHILADELPHIA

Oct 1, 2, 3. **Innovative Technologies in Wound Healing: A Clinical & Scientific Symposium.** Registration deadline Sept. 16. Hotel Atop the Bellevue Philadelphia. Contact: Kate O'Neill, Wound Care Center, The Graduate Hospital, Philadelphia, PA 19146. Tel: (215) 448-1508. ☐

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References:
1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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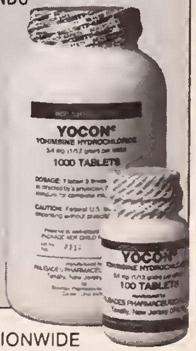

New York State
Journal of Medicine

CONTENTS, CONT'D

CASE REPORTS

359 **Epidemic Kaposi's sarcoma presenting with pleuropericarditis**
SCOT C. REMICK, MD; SAMUEL A. HOISINGTON, MD; JOSEPH A. MIGLIOZZI, MD, PHD

360 **Chronic recurrent multifocal osteomyelitis in adult women**
CATHERINE MARINO, MD; ELIZABETH McDONALD, MD; DANIEL MEGNA, MD; DEBRA BRENNESSEL, MD; KUMAR S. REDDY, MD; PRAMOD C. JAIN, MD

362 **Bilateral adrenal hemorrhage in polycythemia vera**
MAXWELL L. GELFAND, MD

364 **Large endometrioma with intrauterine pregnancy**
JERRY G. NINIA, MD

LETTERS TO THE EDITOR

365 **Appendiceal abscess presenting as an infected urachal cyst**
IHOR S. SAWCZUK, MD, WILLIAM BROWN, MD

365 **The importance of differentiating between anabolic steroids and glucocorticoids**
MICHAEL J. EDBAUER, DO; ANN MARIE LEVINE, MD; F. BRUDER STAPLETON, MD

365 **The physician and the movies**
RANDALL D. BLOOMFIELD, MD

366 **The Phi Lambda Kappa Medical Fraternity: Religion and medicine**
STANLEY ZASLAU

367 BOOK REVIEWS

370 BOOKS RECEIVED

371 OBITUARY LISTINGS

373 POETRY

374 GUIDELINES FOR AUTHORS

3A MEDICAL MEETINGS AND LECTURES

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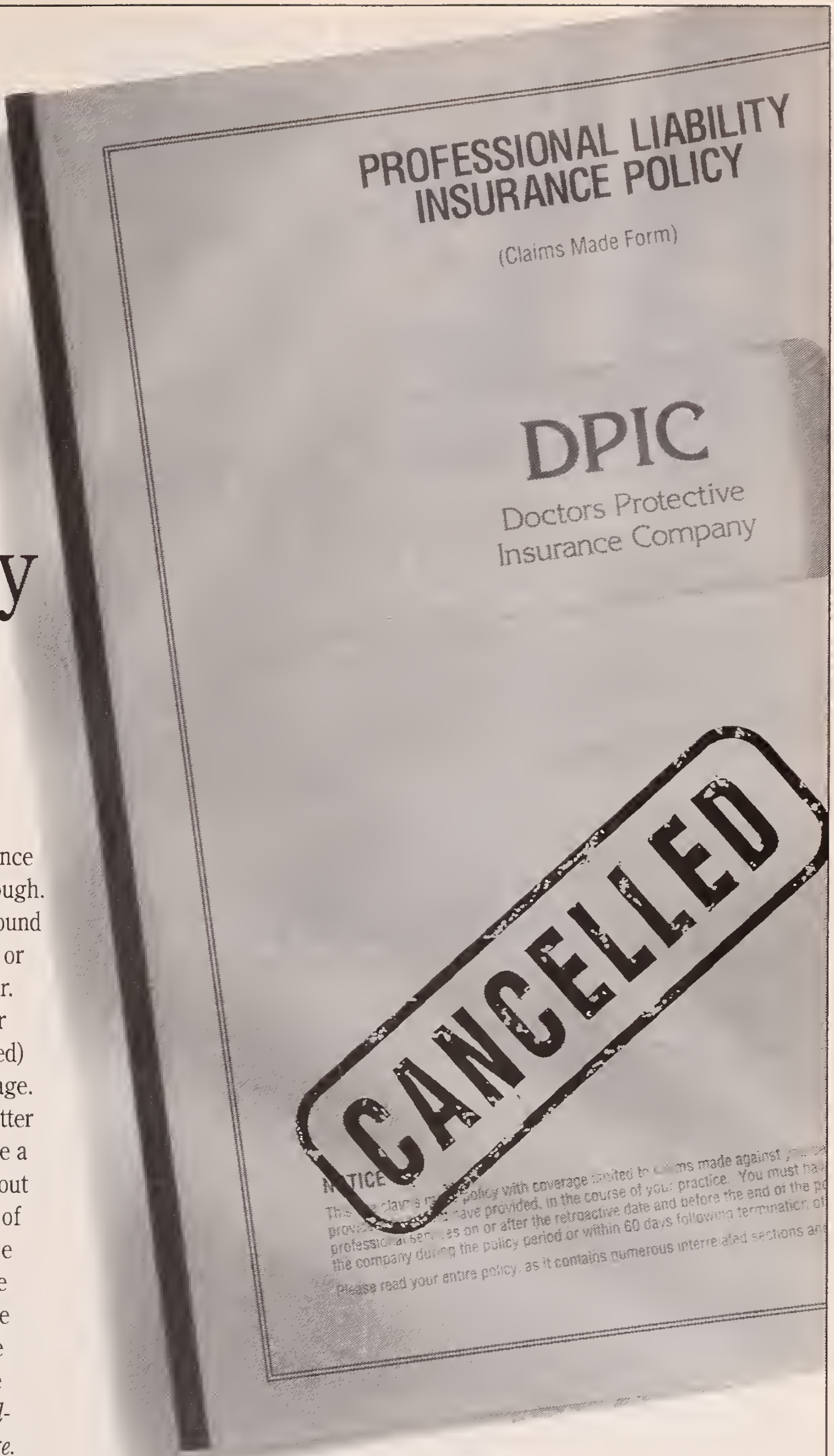
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COMMENTARIES

A public health campaign for New York State

Communities at high risk for widespread disease in New York State are now facing major epidemics for preventable diseases, primarily tuberculosis, syphilis, measles, human immunodeficiency virus (HIV) infection, and acquired immunodeficiency syndrome (AIDS). The same communities are plagued with endemic lead poisoning. Although a rising incidence of these diseases is a concern in urban areas of many other states, New York State usually exceeds them, both in absolute numbers of cases and in rates per 100,000 population.

These epidemics are most prevalent in New York City, but they touch all of New York State, especially having an impact on the Greater New York Metropolitan Region and on urban areas upstate. Some communities at high risk are defined not geographically but institutionally, as in the epidemics of tuberculosis, syphilis, HIV infection, and AIDS in prisons and jails.

The risk factors for these diseases are not limited to transmission vectors. Even where vaccines, antibiotics, and other preventive measures are available, conditions often exist which foster a rising incidence of disease. These conditions include poverty, inadequate access to primary care, prevalent substance abuse, homelessness, adolescent pregnancy, family dysfunctions, and environmental hazards.

Under the direction of Governor Mario Cuomo, a major Public Health Campaign is now under way in New York State to reduce the incidence, morbidity, and mortality of these preventable diseases. In his Message to the Legislature on January 8, 1992, the Governor emphasized the urgency of this public health crisis. Referring to the emergence of diseases such as AIDS and multiple-drug-resistant tuberculosis and to the re-emergence of diseases once thought to be eradicated, such as measles, he directed the State Department of Health and other agencies to mount a comprehensive Public Health Campaign.

More than $7 million in special funding for this campaign was provided in the Executive Budget and approved by the New York State Legislature. These funds will augment existing funds designated for prevention of tuberculosis, measles, sexually transmitted diseases, and lead poisoning. Additional new resources were allocated for media and public information activities.

The Public Health Campaign recognizes that epidemics occur in complex social contexts and are best addressed by large-scale efforts in which provider, community, and government resources are mobilized for collaborative action. In Governor Cuomo's words:

> Our approach to reform in public health must be synergistic; it should apply all our strengths to all of our vulnerabilities simultaneously. And it must involve all sectors within a community—hospitals, physicians, business, government, citizens, and community organizations.

This tradition of multifaceted community-health campaigns is centuries old. Charles-Edward Amory Winslow, a preeminent historian of public health, described campaigns from the Dark Ages to the early 20th century to ward off such scourges as leprosy, plague, cholera, and tuberculosis.[1] The current campaign is directed at the four scourges: tuberculosis, syphilis, measles, and lead poisoning.

Tuberculosis

Climbing rates of HIV infection, substance abuse, and homelessness have led to an ominous increase in cases of active tuberculosis. From a low of 2,078 new cases in New York State in 1978, the number increased by 114% to 4,176 new cases in 1990, the vast majority of which were in New York City. In the absence of organized public health activities to ensure the full course of therapy, noncompliance is frequent, and the problem is compounded by the emergence of organisms resistant to the most commonly used drugs.

The Public Health Campaign will mount a series of interventions to prevent transmission and ensure completion of the long course of drug treatment. The key to both objectives is a program of directly observed therapy for many individuals with active disease. Hospitals, clinics, methadone treatment centers, and other organizations at the community level will be enlisted in both outreach and administration of treatment.

SYPHILIS

A Syphilis Initiative targeted at seven epidemic counties and New York City was launched in June 1990 and is still in progress. The incidence of syphilis had doubled in New York City and quadrupled in the rest of New York State between 1978 and 1990. A shift to heterosexual transmission associated with cocaine use and anonymous sex had resulted in increasing rates among women and the tragedy of 1,200 cases of congenital syphilis in 1990.[2–5] By the following year, a decrease in syphilis cases among men was reported for the first time in four years. However, infection among women and congenital disease remain high.

The Public Health Campaign will further expand screening and treatment, especially for women of childbearing age, in high-risk communities. As in the Syphilis Initiative, the campaign will emphasize collaboration by state and local governments with providers and community organizations to implement intensive testing at sites likely to be frequented by the population at highest risk. These sites include family planning and prenatal care clinics, emergency rooms, ambulatory care facilities, sexually transmitted disease clinics, alcohol and drug treatment centers, HIV anonymous test sites, and local and county penal institutions.

This community-based strategy was strongly endorsed in the Report of the Metropolitan New York City Area Task Force on Syphilis, issued in October 1991, offering comprehensive recommendations for addressing the syphilis epidemic.[6]

MEASLES

In 1990 and 1991, New York City experienced its worst measles epidemic in 20 years. Almost all of the patients were children younger than five years of age, and more than one out of five cases required hospitalization. Some deaths occurred, primarily among children who were also infected with HIV. Although the epidemic subsided in late 1991, the root cause remains unchanged. Future large outbreaks of measles and other vaccine-preventable diseases will occur in New York City and other areas of the state where many young children live without the protection conferred by immunization.

The Public Health Campaign will expand on earlier initiatives in which the State Health Department, in concert with the Department of Social Services, the New York City Department of Health, and health facilities, increased the accessibility of immunization in communities at risk. In a special Measles Initiative in 1991 thousands of children were immunized in community-based organizations; women, infants, and children (WIC) facilities; and other nontraditional settings. Special efforts were also made to provide linkage with primary care. The strategy of that initiative will be enhanced for all immunizations in the Public Health Campaign.

LEAD POISONING

Lead is one of the most pervasive substances in the environment and has insidious effects on children's health and development. As many as half a million children in New York State under the age of six years may have blood lead concentrations that exceed the new standard (10 μg/dL) set by the federal Centers for Disease Control.

Reducing the incidence of lead poisoning is a key objective of the Public Health Campaign. Screening of children in communities at risk will be expanded with a more precise test for blood lead. Faster and more thorough follow-up and medical treatment will be made available for children with elevated blood lead. A registry of such patients is being established to facilitate case management. Expanded educational efforts, directed to the public and to health-care providers, will increase awareness about lead toxicity and the importance of testing all children as a component of primary care.

THE PUBLIC HEALTH CAMPAIGN

The Public Health Campaign is directed at pervasive, severe health problems that are preventable but have resisted noncommunity-based approaches. In this community-wide campaign, government agencies will collaborate with providers of health and social services and with other groups at the community level. HIV infection plays a crucial role in three of these epidemics, and relevant aspects of the campaign are being coordinated with activities of the New York State AIDS Institute.

The campaign has dedicated funding, but these resources will be combined with and influence the expenditures of much larger state and local public health allocations. An integral aspect of the campaign is that specific activities are to be implemented jointly by agencies of the state, New York City, and county governments. For each epidemic an agreement, called a New York/New York Agreement, is being negotiated for signature by Governor Cuomo and either the Mayor of New York City or the county executive.

If effective action is not taken to halt these epidemics now, they will endanger the health of all New York State residents for years to come. The Public Health Campaign will catalyze and coordinate preventive health actions by government, health and social services providers, and community groups to reduce the incidence of diseases that need not occur as the 20th century nears its end.

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1. Winslow C-EA: *The Evolution and Significance of the Modern Public Health Campaign*. New Haven, Yale University Press, 1923.
2. Imperato PJ: The current epidemic in New York State. *NY State J Med* 1991; 91:521.
3. Novick LF: Syphilis: A broad initiative to control New York State's epidemic. *NY State J Med* 1991; 91:524–525.
4. MacCubbin PA, Hipp SS, Murphy DP, et al: Syphilis: The epidemiology of change. *NY State J Med* 1991; 91:526–530.
5. Ong KR, Rubin S, Brome-Bunting M, et al: Early syphilis in New York City: 1985–1990. *NY State J Med* 1991; 91:533–536.
6. *Metropolitan New York City area task force on syphilis. Report of the New York State Department of Health*, Albany, New York, October 10, 1991.

The United States public health service and merchant seamen

A hundred and ninety years ago, on July 16, 1798, President John Adams signed an Act for the Relief of Sick and Disabled Seamen.[1] This Act represented the first federal health entitlement program in the United States. In this issue of the *Journal,* Kavaler and Zavin[2] chronicle the beginning of care for merchant seamen in the Port of New York and the establishment of the Seamen's Fund and Retreat Hospital.

Principal factors leading to this entitlement program were protection of the public against importation of communicable diseases such as cholera and the importance of shipping to the economic health of the new nation.[3] The New York port entitlement began as a state program with care provided by the New York Hospital with funds derived from a tax on seamen, officers, and passengers.[4] The federal program was initiated later in 1798 with a tax on seamen and eventual legislation mandating a tonnage tax.

The Marine Hospital Service, as it was initially called, was a forerunner of managed care programs such as HMOs, in that there was a defined population (merchant seamen), defined benefits, and a form of "community rating." Care was provided by a combination of contracts and employed physicians at dedicated facilities (Marine Hospitals). The health care entitlement was continued until repealed by Congress in 1981. Now, the United States is the only major maritime nation, which does not directly provide health care to its merchant seamen.

During the 19th century, the need for United States Public Health Service (USPHS) hospitals increased as more and more trade and fishing occurred. Early energy needs for the country were partially met by whale oil; this, coupled with commercial fishing, overseas shipping, passenger sailings, and coastal and inland waterway shipping, made the maritime industry one of the principal contributors to the national economy.[5] In 1836, there were 43,000 seamen on the Mississippi River and Great Lakes, and 63,000 in seagoing and coast-wise sailing. By 1848, the total had risen to 150,000. At the peak in the early history of the maritime industry (1873), there was 32,672 documented ships. However, the discovery of oil in Pennsylvania and the completion of the transcontinental railroad led to a diminution of the importance of shipping by the latter part of the 19th century.

Congress appropriated funds for the construction of many hospitals, with best estimates that only 53 were actually built. Numerous "contract" facilities were known as Marine Hospitals as well. Hawaii, the Gulf Coast, the Atlantic and Pacific coasts, the Great Lakes, and the Mississippi River, and its tributaries all had hospitals at major ports.

The early hospitals were involved in medical student and intern/resident teaching as well as research. The Venereal Disease Research Laboratory (VDRL), which developed the VDRL test to diagnose syphilis and which demonstrated the efficacy of penicillin in the treatment of syphilis, was under the leadership of Dr John F. Mahoney at the Staten Island facility in New York City. The forerunners of the National Institutes of Health were developed under Dr Joseph J. Kinyoun at the hospital as well.

The USPHS became the focus for all quarantine efforts when that became a national program in 1893. During periods of war, new duties and beneficiaries were added to the USPHS hospitals, culminating when the War Risk Insurance Bureau was established by congress and legislated medical care for veterans as a federal entitlement in 1917.[6] Until the Veterans Administration became a separate entity in 1921, the USPHS hospitals provided medical services to veterans.

During the mid-20th century, the merchant fleet was reduced and the maritime industry entered a period of decline. The hospitals were authorized to become more involved in providing community services, especially under the Great Society programs of the 1960s, but had fluctuating support in congress and in the various administrations. With the rising federal deficit and the elimination and/or reduction of many federal programs in 1981–1982, Congress repealed the 1798 act and the USPHS hospitals were closed. Some reverted to other federal agencies, some were transferred to community use, and some became community Treatment Facilities (to provide care for active duty and retired military personnel and their dependents). The Staten Island facility in New York City was converted to the Bayley-Seton Hospital operated by the Sisters of Charity.

Most of the current programs of the USPHS, such as research, health manpower development, protection of the health of the public, and infectious disease control, can be traced to beginnings in a USPHS hospital such as the one on Staten Island.

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1. *An Act for the Relief of Sick and Disabled Seamen*, 5th Cong, (Jul 16, 1798).
2. Kavaler F, Zavin SA: Health care for seamen in the Port of New York—Part I: Recognizing the special needs of seamen. *NY State J Med* 1992; 92:353–358.
3. Furmman B: *A profile of the United States Public Health Service 1798–1948*, DHEW Publication NIH 73-369, Washington, DC, 1973.
4. *An Act to Provide for Sick and Disabled Seamen*, April 22, 1831. NYS Leg.
5. *Historical Statistics of the United States 1789–1945*, US Bur Cen, Washington, DC, 1949.
6. *War Risk Insurance Bureau Act*, US Cong, Oct 6, 1917.

Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome

The basis of our knowledge regarding the pathogenesis of the human immunodeficiency virus (HIV), related opportunistic infections, and associated neoplasms continues to evolve. Cardiac complications, for example, are being identified more often clinically in patients with acquired immunodeficiency syndrome (AIDS).[1,2] Previous postmortem studies noted cardiac lesions in 50% to 70% of patients with AIDS.[1,3] Recent estimates suggest that as many as 5,000 HIV-infected patients per year may develop cardiac complications.[2] In fact, a two-dimensional echocardiographic study in San Francisco revealed that 64% of acutely ill, hospitalized patients with AIDS had cardiac abnormalities.[4] Cardiac complications encompass primary and secondary infections, neoplasms, non-specific myocarditis, and autoimmune dysfunctions. Most patients with AIDS have no obvious clinical signs of cardiac dysfunction.[2] When cardiac involvement occurs, the clinical presentation is often attributed to respiratory failure or infection. Fortunately, because of enhanced clinical awareness and newer diagnostic capabilities, cardiac problems are being detected and treated in a more timely manner. The antemortem diagnosis of cardiac Kaposi's sarcoma (KS) by Remick et al, as presented in this issue of the Journal is an example of this heightened awareness and earlier diagnosis.[5]

KS is a major neoplastic complication of AIDS.[6] The histopathology is characterized by a proliferation of small vessels with abdominal endothelial cells and tissue infiltration by spindle-shaped cells. AIDS-related KS is often wide-spread and progressive, affecting skin, mucous membranes, lymph nodes, and visceral organs. KS limited to the heart has been reported, but more commonly cardiac involvement is associated with disseminated disease.[1,2] Additionally, although there have been reports of serious morbidity and death related to cardiac involvement of this neoplasm in HIV-infected patients,[7,8] most of these patients do not succumb to the KS, but rather to opportunistic infections.[1] However, it is likely that the complications related to this neoplasm may be seen more commonly in the future because the advances made in the therapeutic modalities associated with many of the opportunistic infections are now affording patients increasingly longevity.

There are currently no unequivocal treatment strategies for AIDS patients with KS. The lack of uniform and precise criteria for KS disease evaluation, clinical staging, and response to treatment in patients with AIDS has hindered therapeutic trials. Yet, more progress has been made to standardize these criteria.[9] Various therapeutic approaches can be taken, and these are determined by the extent and location of the KS lesions and the severity of the tumor-related symptoms. These treatment options recently have been reviewed for this specific patient population.[6]

Local approaches have been attempted with surgical excision, laser or cryotherapy, and intralesional chemotherapies. Radiotherapy, either alone or in combination with radiosensitizing chemotherapeutic agents, has been used with varying success.[6] The use of cytotoxic chemotherapy with vinca alkaloids, bleomycin, actinomycin D, and dacarbazine, has resulted in a substantial number of partial and complete responses although in many instances the duration of the response is short lived. Interferons, in particular interferon-α have also been used alone and in combination regimens; positive objective response rates have been documented in several trials.[6,10] Unfortunately, many of the studies conducted thus far with combination therapies have provided little evidence for increased efficacy, yet have substantially increased toxicity.[6] There are many unanswered questions concerning the optimal use of local and systemic therapies in HIV-infected patients with KS.

At the present time, physicians have much to contemplate when considering what is appropriate treatment of a patient with disseminated KS involvement. Early diagnosis with aggressive therapy must be weighed against inducing complications related to the therapy itself. Aggressive therapy can ameliorate symptoms and improve the quality of life in these patients, but it can also place the patient at significant risk for life-threatening opportunistic infections. Additionally, in patients with cardiac compromise, the use of anthracycline chemotherapies could lead to further deterioration of cardiac function. It can be a difficult decision. If physicians follow the creed, "first, do no harm," careful regard for all the risks and benefits must be weighed. The patient's input on quality of life issues should be fundamental.

Fortunately, progress is being made in many aspects of AIDS treatment regimens. As the AIDS dilemma continues to unfold, physicians must remain vigilant for the many complications that potentially can be treated in a timely manner. They must also stay abreast of new therapeutic modalities that may improve the quality of life for their patients while remaining cognizant of the risk-benefit ratio of these therapies.

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REFERENCES

1. Francis CK: Cardiac involvement in AIDS. *Current Probl Cardiol* 1990; 15:575–639.

2. Kaul S, Fishbein MC, Siegel RJ: Cardiac manifestation of acquired immunodeficiency syndrome: A 1991 update. *Am Heart J* 1991; 122:535–544.

3. Paraf F, Py A, Pauly-Laubry C, et al: Pathologie cardiaque au cours de l'infection par le virus de l'immunodeficience humaine. *Arch Anat Cytol Path* 1990; 38:171–179.

4. Himelman RB, Chung WS, Chernoff, DN, et al: Cardiac manifestations of human immunodeficiency virus infection: A two-dimensional echocardiographic study. *J Am Coll Cardiol* 1989; 13:1030–1036.

5. Remick SC, Hoisington SA, Migliozzi A: Epidemic Kaposi sarcoma presenting with pleuropericarditis. *NY State J Med* 1992; 92:359–360.

6. Krown SE, Myskowski E, Paredes J: Kaposi's sarcoma. *Med Clin North Amer* 1992; 76:235–254.

7. Steigman CK, Anderson DW, Macher AM, et al: Fatal cardiac tamponade in acquired immunodeficiency syndrome with epicardial Kaposi's sarcoma. *Am Heart J* 1986; 116:1105–1107.

8. Stotka JL, Good CB, Downer WR, et al: Pericardial effusion and tamponade due to Kaposi's sarcoma in acquired immunodeficiency syndrome. *Chest* 1989; 95:1359–1361.

9. Krown SE, Metroka C, Wernz JC: Kaposi's sarcoma in the acquired immunodeficiency syndrome: A proposal for uniform evaluation, response, and staging criteria. *J Clin Oncol* 1989; 7:1201–1207.

10. Volberding PA, Mitsuyasu R: Recombinant interferon alpha in the treatment of acquired immunodeficiency syndrome-related Kaposi's sarcoma. *Semin Oncol* 1985; 12:2–6.

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PUBLIC HEALTH

Since the State Department of Health has been assigned the responsibility of securing compliance with health laws and regulations, it follows that one of its first duties is to acquaint the public with these laws and the basic principles underlying them in order that the necessity for strict obedience to these mandates may be fully understood. The methods employed in bringing health facts before the public vary according to conditions and the exact end sought. Broadly speaking, the method to be used in any given case is the one which will best carry the message and impress it indelibly on the mind of the people.

In any local health campaign, the State Department of Health is in position to render valuable assistance, but sometimes communities do not avail themselves of this service, probably because they are not familiar with the material and facilities which the Department has to offer. While it is undesireable that the Department should intrude where local initiative has resulted in the community developing its own plans, cooperation and assistance from the Department may often enable a local health agency to benefit greatly from the experience of others in problems of organization and of popular health education. Such local initiative and the needs of the individual community should be the basis for determining if assistance from the Department is desired in outlining a policy or in carrying out a program already determined upon. If such assistance is desired it will be rendered by the State Department as far as facilities permit. Sometimes questions of sanitary engineering are involved, sometimes a child welfare program is under way, in other cases tuberculosis clinics are desired or perhaps the community is interested in the venereal disease problem. Nearly every division of the Department is in a position to cooperate with any local health committee which will make its wants known.

EDITORIAL
(*NY State J Med* 1920; 20:203–204)

Alcoholism-induced bone necrosis

BERNARD JACOBS, MD

ABSTRACT. A total of 164 patients with alcoholism-induced osteonecrosis were seen over a 22-year period, from 1962 to 1984. Twenty-three percent of patients were female and 30.5% were black. The average duration of alcohol abuse was 9.5 years, ranging from 8 to 20 years. The presence of femoral head necrosis was diagnosed in patients aged 21–67 years; 28% of patients were under 40 years of age and 76% were under 50 years. Bilateral hip necrosis was present in 44.5% of patients and, within three years of the diagnosis of FHN, the presence of multifocal necrosis became evident in 23 cases at sites away from the hip (shoulders and knees).

Hyperlipidemia was found in 38.4% of cases, involving both cholesterol and triglycerides. Serum amylase was elevated in 33 patients; liver dysfunction was present in 50; hepatomegaly was found in 32; and biopsy-confirmed cirrhosis was present in 22 cases. Hyperuricemia was found in 22 patients, some of whom had received steroids. Disabling hip pain was the first manifestation of disability related to alcohol abuse in 158 patients, most of whom required total hip joint replacement.

This study supports the hypothesis that alcoholism-induced bone necrosis is caused by fat embolism linked to co-existent hyperlipidemia. The treatment of hyperlipidemia by dietary means or lipotropic medication and the cessation of alcohol abuse is advised. Multi-center studies employing such treatment should provide evidence of its effect on the evolution of necrosis as well as the incidence of bilateral hip femoral head necrosis and multifocal lesions.

(NY State J Med 1992; 92:334–338)

Bone necrosis, also termed osteonecrosis, had until recently also been called idiopathic necrosis. This condition most often afflicts the femoral head (Fig 1), but can also occur at other sites. It has long been assumed that necrosis is the result of circulatory embarrassment, to which the femoral head is especially vulnerable. The term "idiopathic" is now rarely used because the cause of femoral head circulatory occlusion appears to be well established. Currently, the favored explanation is that the femoral head circulation is interrupted by micro-fat emboli, which, in turn, have been linked to co-existent alcoholism-induced hyperlipidemia.[1]

An alternative theory, proposed by Boettcher et al,[2] is that the causative factor consists of a coagulation defect. The authors studied a group of patients using a battery of blood tests and found no consistent abnormality. Axhausen[3] is credited as having first suggested that osteonecrosis results from an embolic phenomenon. Vignon et al[4] was the first to report the connection between alcoholism and femoral head necrosis (FHN).

PATIENTS AND METHODS

Our study consisted of data collected over a 22-year period, from 1962 to 1984, on 164 patients diagnosed as having alcoholism-induced osteonecrosis. Many were patients at the affiliated Bronx Veterans Administration Hospital. At the outset, we included only compulsive drinkers of two or more "highballs" or the equivalent in beer consumption, and those with known liver disease. However, in the latter years of the study period, our criteria changed to include compulsive, daily drinkers regardless of quantity. Experience has confirmed that some patients do not readily admit to alcohol abuse, thus making the true incidence of alcoholism-induced osteonecrosis an elusive determination.

RESULTS

Thirty-eight (19.5%) of the 164 patients were female and 50 (30.5%) were black. Age at the time of diagnosis of osteonecrosis ranged from 21 to 67 years (Table I). The majority (76%) of patients were below 50 years of age. The average duration of alcoholism was 9.5 years and ranged from 8 to 20 years.

Bilateral hip necrosis was evident in 73 patients (44.5%). At the end of the study period, however, osteonecrosis was found at sites other than the hip in 40 patients (Table II), most frequently involving the humeral head (Table II, Fig 2) and knee regions and, least often, at the talus (Table II, Fig 3).

In consideration of the theory of micro-fat embolism and hyperlipidemia as possible causative factors, all but 12 patients underwent serologic testing that included total cholesterol, triglycerides, amylase, uric acid, alkaline phosphatase, and serum glutamic-oxaloacetic transaminase/serum glutamic-pyrovic transaminase (SGOT/SGPT). Hyperlipidemia was present in 63 patients (38.4%), with elevated cholesterol levels found more frequently than raised triglycerides (Table III). Serum amylase was elevated in 33 patients and evidence of liver dysfunction was present in 50 cases. Twenty patients had hyperuricemia and ten had used corticosteroids for clinical gout. Hepatomegaly was present in 32 patients and biopsy-confirmed cirrhosis or fatty liver disease in 22 cases.

Of interest, is that impairing hip pain was, in 23 cases, the first manifestation of disability related to alcoholism, and that all but 19 patients required some form of major hip surgery. Also noteworthy are the associated co-existent problems uncovered in some patients (Table IV), including steroid use, hyperuricemia,[5] pancreatitis,[6] diabetes, and sickle cell disease, each of which has been associated with osteonecrosis.

DISCUSSION

The association with alcoholism of liver disease and hyperuricemia has been well documented by Lieber et al .[7] Lipid studies similar to our own have been reported by

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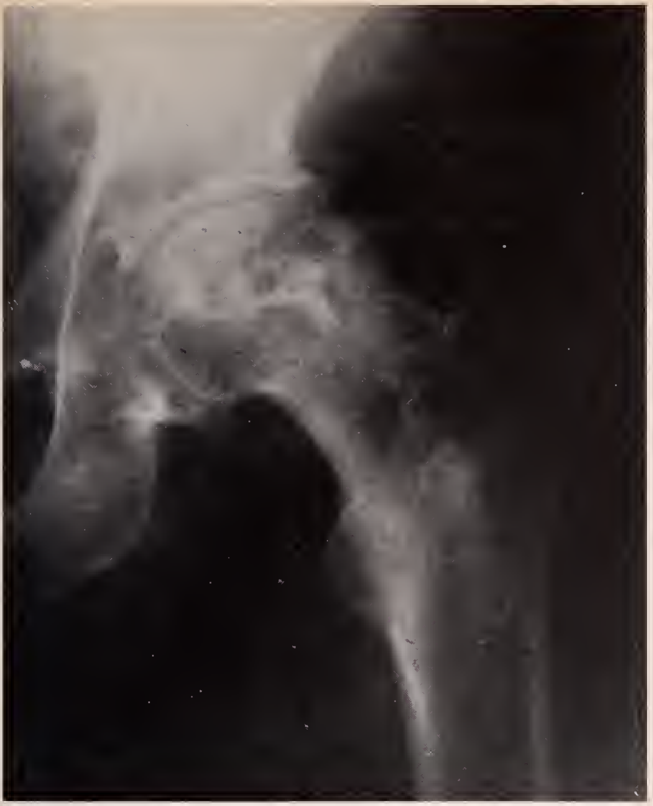

FIGURE 1. Typical appearance of unilateral femoral head necrosis.

other authors. In Jones's[8] study of 30 alcoholic patients, six of eight patients had hypertriglyceridemia, and, in a similar study, Roux et al[9] reported a high frequency of Types 2 and 4 Fredrickson lipid profiles, compared to a control group. Serre et al[10] found a 53% incidence of hyperlipidemia in 45 patients with alcohol-associated osteonecrosis, whereas Louyot et al[11] reported elevated pre-beta lipoproteins in 75% of their series of 150 patients with alcoholism-induced hip necrosis.

In 1983, this author supplied necrotic hips from alcoholic patients with hyperlipidemia to Boskey et al[12] for pathologic study. These colleagues found dense accumulations of cholesterol in the femoral heads and necks and proposed that these deposits were responsible for osseous cell death as a result of excessive pressure.

It is of interest that the sixth report to the Congress on Alcoholism, prepared by the National Institute on Alcoholism,[13] discussed the deleterious effects of alcoholism on all organ systems but failed to refer to the effect of alcoholism on the bones and joints and, moreover, made no specific mention of osteonecrosis or osteoporosis, conditions known to be aggravated by alcoholism. The same omission occurred in a comprehensive report on alcoholism issued by the New York State Department of Health to the state legislature.[14]

Clearly, there is a need to promote public and medical education on these associations. Valuable information would be gained from a multicenter study of alcoholism-induced osteonecrosis that would evaluate the incidence of the disorder and the effectiveness of treating associated hyperlipidemia by dietary modifications alone or in conjunction with lipotropic medication.

Visceral fat embolism associated with alcoholism has been well documented[15] and has been found to occur with both acute and chronic alcoholism. In a post-mortem study of 40 subjects with alcoholic liver disease, Lynch et al[16]

TABLE I. Clinical Characteristics of a Series of 164 Patients with Alcoholism-induced Osteonecrosis

Females	38
Males	126
Total Patients	164
Patients of Black Race	50
Age Range at Diagnosis	21–67
Duration of Alcoholism (yrs)	8–20 (mean = 9.5 yrs.)
Cases of Bilateral Hip Necrosis	73 (44.5%)
Cases of Multifocal Necrosis	40 (24.3%)
Hyperlipidemia	63 (38.4%)

TABLE II. Common Sites of Multifocal Lesions in 40 Patients with Alcoholism-induced Osteonecrosis

Site	Frequency	Bilateral Occurrence
Humeral Head	20	10
Knee Region (lower femur; upper tibia)	15	0
Lower Tibia	4	0
Talus	1	0
Totals	40	10

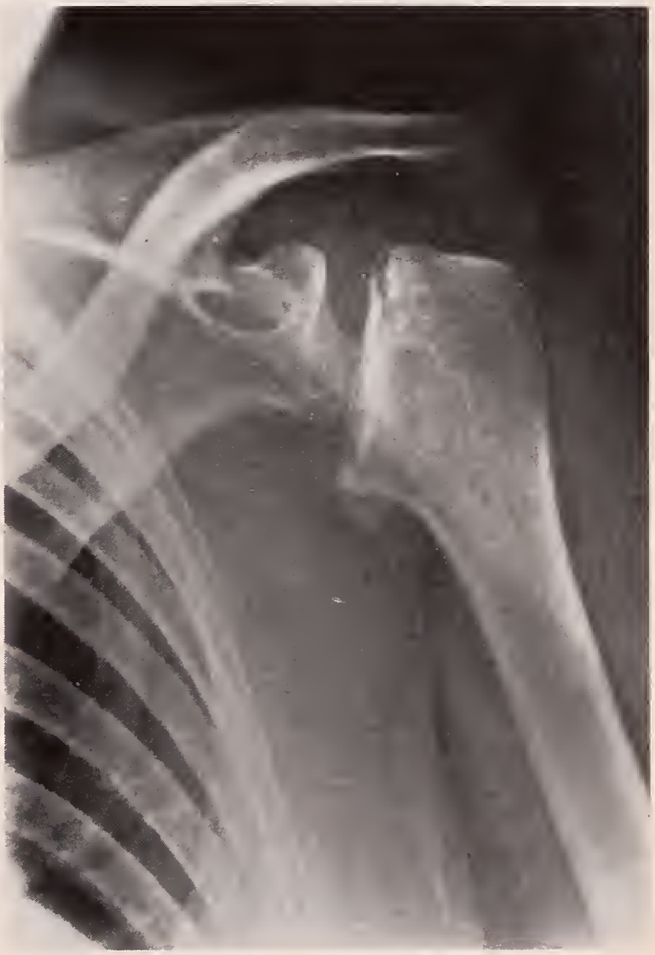

FIGURE 2. Humeral head necrosis in a 53-year-old patient.

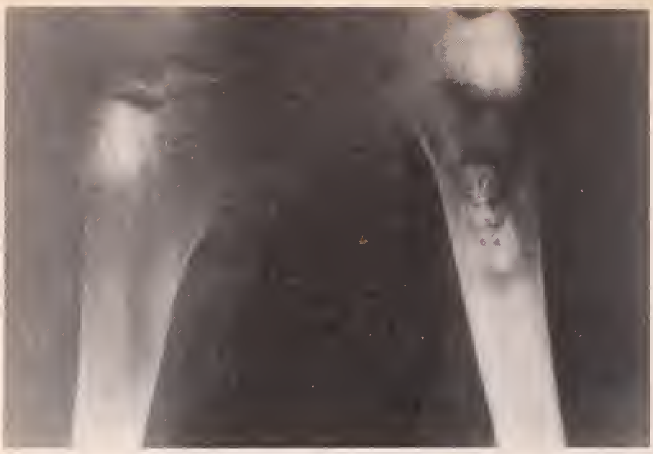

FIGURE 3. Lower femur infarcts in a 48-year-old patient.

found pulmonary fat embolism in 77.5% of cases and cerebral embolism in 68.6%.

The concept that fat embolism could also cause bone necrosis has been proposed by a number of authors, notably Wefling,[17] De Seze et al,[18] and Jones and Engelman.[19] The latter authors proposed that the alcoholism-induced fatty liver could be the source of embolic showers sufficient to cause bone infarction, and demonstrated intravascular fat globules in necrotic femoral heads. It is of interest that triglycerides are known to be the chief component of fatty livers and of the chylomicron aggregates that constitute emboli. Further support for the fat embolism mechanism accrued from Jones et al[20] animal experiments, in which iodized oil (Lipiodol) infusions consistently produced intraosseous emboli and obstruction of subchondral vessels in the femoral head.

Information concerning the incidence of alcohol-associated FHN has been provided by a number of authors (Table V). In an earlier review of 269 cases of FHN, of which 45% of patients were alcoholics and 10% were women, this investigator[21] found that the incidence of bilateral FHN was 50%. Although the reported incidence varies from 17% to 74%, all of the investigators[5,8,11,22] regarded the association as significant; there was also general agreement concerning the frequency of bilateral hip involvement and its incidence, ranging, in females, from 10% to 17%.[21]

The true incidence of alcohol-associated osteonecrosis is uncertain, but definitely does not mirror the prevalence of alcohol abuse. The lowest incidence of alcohol-associated FHN, reported by the Mayo Clinic,[23] was triple the frequency of alcoholism in the general patient population. A simple explanation might be the requirement for sustained bombardment of the femoral head with fat emboli. Hypertriglyceridemic subjects might be more prone to such exposure, since it has been shown that alcohol-induced hyperlipidemia is markedly exaggerated when alcohol is ingested with a fatty diet.

TABLE III. Serologic Test Results in 164 Alcoholic Patients with Osteonecrosis

Test	Normal Values	Cases with Elevated Values	Percentages
Cholesterol	267–425 mg	65	39.6
Triglycerides	197–940 mg	59	35.9
SGOT/SGPT	6–26 U/0–28 U	49	29.8
Alkaline phos.	6–9 Bu	36	23.7
Amylase	170–400 U	40	24.3
Uric acid	9–14 mg	30	18.2

TABLE IV. Associated Disorders Found in 44 Patients Among a Series of 164 Patients with Alcoholism-induced Osteonecrosis

Disorder	Cases	Percentages
Hyperuricemia	20	45.4
Steroid usage	10	22.7
Pancreatitis	9	20.4
Sickle cell disease	3	6.8
Diabetes	2	4.5

Circulatory vulnerability may also rest on recognized co-existent congenital variations in the blood supply to the femoral head. Finally, coagulation disorders known to exist in alcoholics, as proposed by Boettcher et al,[2] also may play a role in establishing vulnerability.

With the abundance of femoral heads made available by the increasing popularity of hip replacement surgery, evidence of underlying necrosis is frequently found masquerading as "idiopathic" osteoarthritis (Figs 1,2). The possibility that many osteoarthritic hips originate as osteonecrosis, obscured by superimposed degenerative changes, is by no means a novel suggestion. In 1940, Phemister[24] proposed a common cause for osteoarthritis and bone infarction. Three decades later, after an exhaustive study of osteoarthritic hips, Streda[25] concluded that most had underlying necrosis.

Although most often afflicting the femoral head, alcohol-associated osteonecrosis frequently occurs at other sites, substantiating its systemic nature. In Jones's[8] study of 30 alcoholic patients, a total of 77 lesions were found; the 31 non-hip lesions included the proximal tibia,[12] distal femur,[10] and humeral head.[9] These sites accounted for most of the 47 non-hip lesions found in our own series. Most of these lesions were either "silent" or idly symptomatic and were detected in the course of skeletal roentgenographic surveys. In addition to roentgenographic survey, radioisotope scintimetry has proved useful, permitting identification of disease in 20 asymptomatic contralateral hips with little or no initial roentgenographic changes.

In at least 23 cases, hip pain caused by necrosis was apparently the first symptomatic disability associated with alcohol abuse. Some patients, when informed of the interrelationship, terminated their habit and may have averted multifocal and contralateral hip involvement. Apart from an improved general well-being, another accrued advantage may have been the prevention of further loss of pelvic

TABLE V. Reported Case Series of Alcohol-associated Osteonecrosis of the Hip Joint

		Percentages		
Published Source	Total in Series	Alcoholic	Female	Bilateral Presence
Boettcher et al[2]	50	74	12	72
Jacobs[21]	164	100	23	44
Louyot et al[11]	150	22	13	?
McCollum et al[5]	68	40	15	34
Merle d'Aubigne et al[22]	125	Common	17	50
Patterson et al[23]	52	17	17	64

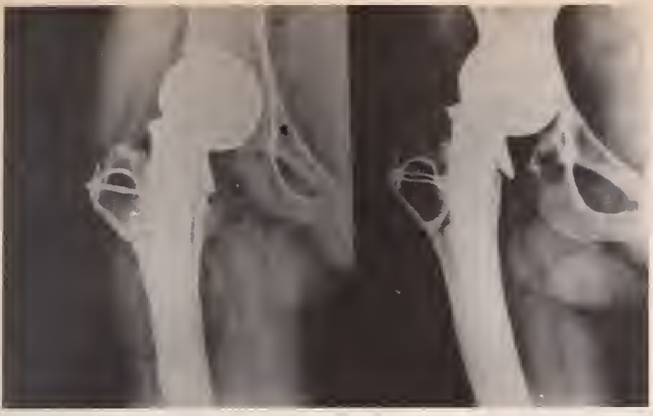

FIGURE 4. Appearance of hemi-arthroplasty performed in 1970 left showing complete loss of joint space and intra-pelvic protrusion six years later (1976, right).

bone mass (osteopenia), a factor which is believed to have an adverse effect on the outcome of femoral head replacement by means of hemi-arthroplasty.

The potential benefits of hemi-arthroplasty are that replacement of the acetabulum is not required and that total hip replacement may be indefinitely postponed. Unfortunately, in alcohol-associated hip necrosis, the longevity of hemi-arthroplasty is significantly reduced by gradual and progressive protrusion of the femoral head component through the soft acetabular cartilage into the osteoporotic pelvic bone (Fig 4). This complication usually is apparent within ten years; when it becomes sufficiently painful, a total hip replacement becomes inevitable (Fig 5).

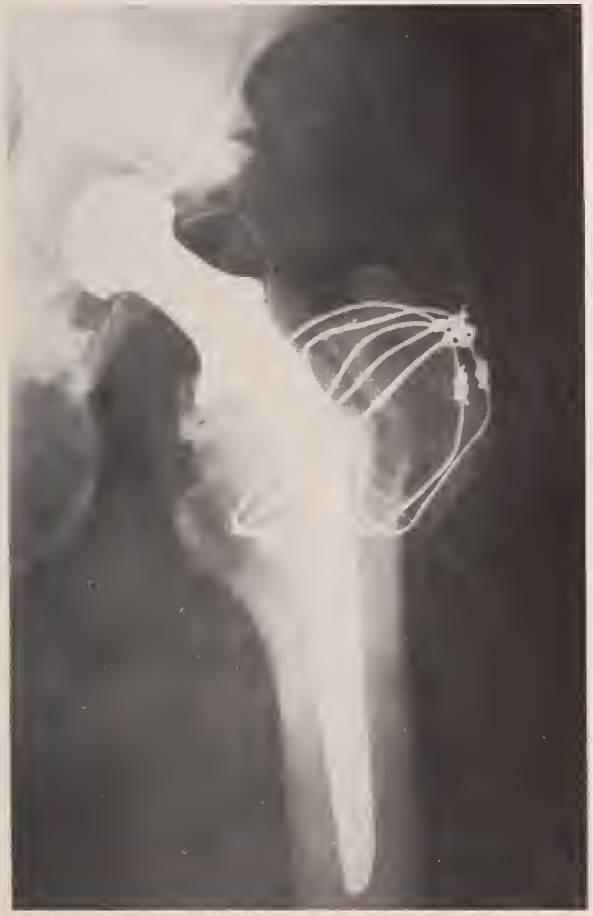

FIGURE 5. Total hip replacement in a 52-year-old patient.

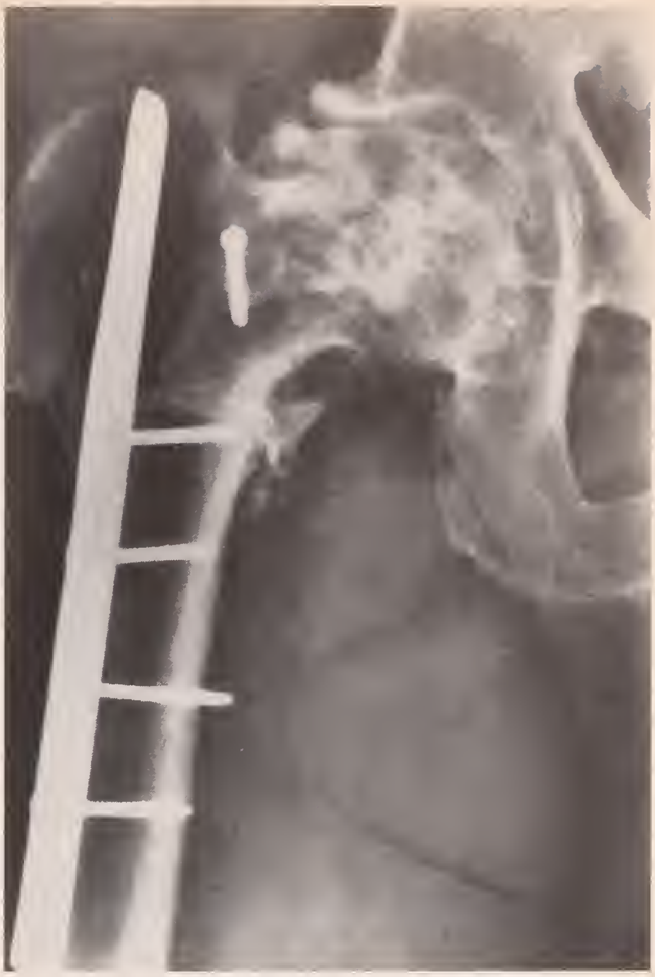

FIGURE 6. Appearance of osteotomy combined with muscle pedicle graft in a 56-year-old patient showing some preservation of the joint space six years after surgery.

Less radical treatment than total hip replacement, especially favored for younger patients, requires early evaluation and treatment of the hip, before significant collapse of the femoral head occurs. In recent years, we have accumulated evidence that magnetic resonance imaging (MRI) is a more efficient means of establishing an early diagnosis compared to conventional radiography.

Conservative treatment approaches include protective weight bearing, bone grafting, osteotomy, and externally applied electrical stimulation.

Experience with many patients who presented with acute onset of pain coincident with femoral head fracture of the necrotic segment, but without significant collapse, has demonstrated that a three-month period of protective weight bearing with the aid of crutches successfully relieves pain and is helpful in buying time before hip replacement surgery becomes the ultimate recourse. Similar benefits can accrue from bone grafting, provided diagnosis is made before significant femoral head collapse.

The goal of osteotomy (Fig 6) is to alter the angle of the femoral neck and reduce the stress of weight-bearing on the femoral head. It is estimated that about 70% of patients who receive this form of early intervention can gain ten or more years needing a hip replacement.

Electrical stimulation is applied externally in the hope of activating new bone production. However, a multi-center study, while demonstrating benefit through the reduction of hip pain, has not established the ability of electrical stimulation to defer the inevitable collapse of the necrotic portion of bone.

Regardless of the form of conservative management, early diagnosis and initiation of treatment ensure the best chance of success short of total hip replacement. Speedy treatment of associated hyperlipidemia is also important in the effort to forestall disease progression. Alcoholic patients who present with hip, knee, or shoulder pain should be assumed to be afflicted by osteonecrosis until adequate radiologic surveys and radioisotopic scintimetry have otherwise ruled out the diagnosis.

A treatment regimen that involves cessation of alcohol abuse, dietary modification, and the use of currently available medications is often successful in reducing hyperlipidemia. This combination approach may act to prevent contralateral hip necrosis and multifocal lesions. The disability resulting from osteonecrosis of the hip and humeral head ultimately requires total prosthetic replacement in the majority of cases. The best chance for less radical surgery, therefore, lies in early diagnosis. Multicenter studies are needed to collect data on the epidemiology of alcoholism-induced osteonecrosis, multifocal lesions, and the association of these with hyperlipidemia, and to evaluate treatment options.

REFERENCES

1. Hartmann G: The possible role of fat metabolism in idiopathic ischemic necrosis of the femoral head, in Zinn WM (ed): *Idiopathic Ischemic Necrosis of the Femoral Head in Adults*. Stuttgart, Germany, G Thieme, 1971, pp 140–144.
2. Boettcher WG, Bonfiglio M, Hamilton HH, et al: Non-traumatic necrosis of the femoral head. *J Bone Joint Surg* 1970; 52A:312–321.
3. Axhausen G: Uber anamishe infrakte an knochen system und ihre bedeutung fur die lehre von den primaren epiphysennekrosen. *Arch f Klin Chr* 1928; 151:72–98.
4. Vignon G, Duguesnel J, Drugue M, et al: Les nècroses primatie de la tete fèmorale chez l'adulte. *Rev Lyon Med* 1980; 9:12–15.
5. McCollum DE, Mathews RS, O'Neill MT: Aseptic necrosis of the femoral head. *South Med J* 1970; 62:241.
6. Inmelman EJ, Bank S, Krige H, et al: Roentgenologic and clinical features of intramedullary fat necrosis in bones in acute chronic pancreatitis. *Am J Med* 1964; 36:96–105.
7. Lieber CS, Jones DP, de Carli LM: Effects of prolonged ethanol intake. Production of fatty liver despite adequate diet. *J Clin Invest* 1965; 44:1009.
8. Jones JP Jr: Alcoholic hypercortisonism, fat embolism, and osseous avascular necrosis, in Zinn WM (ed): *Idiopathic Ischemic Necrosis of the Femoral Head in Adults,* Stuttgart, Germany, G Thieme, 1971, pp 112–133.
9. Roux H, Gastaut JA, Vovan L, et al: Exploration lipidique d'un groupe d'ostèonècrose. *Rev du Rhumatologie* 1974; 41:393–398.
10. Serre H, Simon L: L'ostèonècrose primatie de la tete femorale chez l'adulte. II. Etiologie et pathogenie. *Rev du Rhumatologie* 1962; 29:536–545.
11. Louyot P, Gaucher A, Pourel J, et al: De quelques problemes pose par l'ostèonècrose aseptique de la tete fèmorale. *Rev du Rhumatologie* 1971; 38:201–215.
12. Boskey AL, Raggio CL, Bulllogh PG, et al: Changes in the bone tissue lipids in persons with steroid and alcohol-induced osteonecrosis. *Clin Orthop* 1983; 172:289–295.
13. National Institute on Alcoholism: *Sixth Report to Congress on Alcohol and Health*. January 1987.
14. *Alcohol and Public Health Implications for New Division of Epidemiology*. Albany, NY, New York State Department of Health, 1989.
15. Fadell EJ, Sullivan BH: Fatty liver and fat metabolism. *US Armed Forces Med J* 1957; 8:114–119.
16. Lynch MG, Raphael S, Dixon TP: Fat embolism in chronic alcoholism. *Arch Pathol* 1966; 67:68–80.
17. Wefling J: Embolies micro-particulaires et nècrose de la tete fèmoral chez l'adulte. *Rev Du Rhumatologie* 1967; 34:126–130.
18. De Seze S, Wefling J, Lequesne M: L'ostèonècrose primitie de la tete fèmoral chez l'adulte. *Rev du Rhumatologie* 1960; 27:117–127.
19. Jones JP Jr, Engelman EP: Avascular necrosis of bone in alcoholism. *Arch and Rheum* 1967; 10:287–288.
20. Jones JP Jr, Sakovich L: Fat embolism in bone. *J Bone Joint Surg* 1966; 48A:149–164.
21. Jacobs B: The epidemiology of osteonecrosis. *Clin Orthop* 1978; 130:51–66.
22. Merle d'Aubigne R, Postel M, Mazabraud A, et al: Idiopathic necrosis of the femoral head in adults. *J Bone Joint Surg* 1965; 47B:612–633.
23. Patterson RJ, Bickel WH, Dahlin DC: Idiopathic necrosis of the head of the femora. *J Bone Joint Surg* 1964; 46A:267–282.
24. Phemister DB: Changes in bones and joints resulting from interruption of circulation. *Arch Surg* 1940; 41:2455–2482.
25. Streda A: Participation of osteonecrosis in the development of severe coxarthrosis (Monograph No. 42). Praque, Czechoslovaikia. Aetra Universitatis Carolinae Medica, 1971.

Lead screening and follow-up in an urban pediatric clinic

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ABSTRACT. The purpose of this study was to examine screening and follow-up procedures used in testing for lead toxicity at an urban pediatric clinic. A retrospective chart review of all patients with elevated zinc protoporphyrin levels was undertaken.

In the first four months of 1990, 66 of 438 patients screened (15.1%) had elevated zinc protoporphyrin levels. Sixty charts were located and reviewed to evaluate five key interventional strategies. Iron therapy was implemented in 31 (51.2%), and follow-up was scheduled in 21 (35.0%). Two exposure histories and two dietary histories were documented (3.3% each). Only 26 children (43.3%) had venous blood drawn for lead-level determination.

Eleven patients (18.3%) had none of these five steps documented, and an additional 26 (43.3%) received only one. Fifteen children (25.0%) received two interventions, six (10.0%) received three, and two (3.3%) received four. No patients received all five steps.

Better procedures are needed at this site to ensure the implementation and documentation of diagnostic and therapeutic interventions in children with elevated zinc protoporphyrin levels. Other screening programs may benefit from examination for similar methodologic problems.

(NY State J Med 1992; 92:338–342)

Lead is an element that has no biological value. As a result of industrialization, however, it has become ubiquitous in our environment. Lead toxicity has been and remains one of the most common, but preventable pediatric public health problems. Excess lead absorption is associated with disruption of hemoglobin (Hb) synthesis, impairment of central and peripheral nervous system function, neuropsychologic abnormalities, and proximal tubule injury in the kidney. A nationwide study conducted between 1976 and

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1980 by Mahaffey et al[1] found that 3.9% of children under the age of five years, or 675,000 children, had blood lead levels greater than or equal to 30 μg/dL, the value which at that time defined an elevated blood lead level. Furthermore, it was shown that about 2% of white children were affected, whereas 18.6% of urban black children from families with incomes of less than $6,000 per year were affected.

These figures are particularly alarming given the fact that recent studies have prompted a lowering of the threshold value to 25 μg/dL. Undoubtedly, there were thousands of children in the above study with lead levels between 25 and 30 μg/dL, who would today be considered to have elevated lead levels.

Significant progress has been made in reducing the amount of lead in the environment. The gradual phasing out of leaded gasolines has helped reduce lead levels in the air, and the 1977 banning of lead-based paint for household use has helped reduce domestic exposure. As a result of these and other efforts, levels of environmental lead have decreased. However, although cases of florid lead encephalopathy are rarely seen today and there have been no fatalities attributed to lead poisoning in New York City since 1974, the effects of low-level lead toxicity are still seen, and several important sources of lead remain. Old leaded pipes and lead solder used to join copper pipes continue to contaminate drinking water. Additionally, paints for specialized marine, industrial, and military uses still may contain more than the 600 parts per million allowed for household paint.

More importantly, some 27 million homes in the United States still have layers of leaded paints on their walls.[2] Peeling paint chips in dilapidated housing account for a large proportion of pediatric lead poisoning cases seen in this country. Even in buildings where the leaded paint is not peeling, paint dust containing significant amounts of lead can accumulate on surfaces, where it can find its way onto the fingers and eventually into the mouths of children.[3]

Lead inhibits two enzymes in the heme-synthesis pathway, ferrochelatase and δ-aminolevulinic acid dehydrase (ALAD), causing accumulation of several forms of erythrocyte protoporphyrin (EP) in the blood. Assays that test for protoporphyrins have thus become popular screening tools for elevated blood lead.

Since ferrochelatase needs iron to complete the conversion of protoporphyrin to heme, iron deficiency also results in the accumulation of protoporphyrins. EP testing thus provides an effective screen for iron deficiency. Indeed, it has been demonstrated that EP elevation can be detected even before a clinically detectable anemia develops.

Two methods of testing EP are currently available. Both are based on the detection and quantification of zinc protoporphyrin (ZNP). The first technique involves extraction of this metabolite from the erythrocytes in a fingerstick blood specimen. The extraction process converts the ZNP to free-erythrocyte protoporphyrin (FEP), which is then measured in a fluorometer and reported in μg/dL. The second method involves direct fluorimetry of a drop of fingerstick blood placed on a slide in a hematofluorometer, giving a ZNP result directly, again in μg/dL. The first method is generally more precise, especially at low EP levels, but the second technique offers the advantage of almost immediate determination of results; the extraction method is more time consuming and is generally done only in large, centralized labs.[4]

Until the mid-1970s, lead poisoning in children was defined by an FEP of 50 μg/dL or greater, and a blood lead of 30 μg/dL or greater. Since that time, research has conclusively demonstrated that physiologic effects occur at considerably lower levels.[5–7] From a biochemical standpoint, it has been shown that inhibition of ALAD occurs at blood lead levels below 10 μg/dL,[5] and that ferrochelatase is inhibited at a blood lead of 15 μg/dL.[6] In addition, a reduction in the circulating levels of the active form of vitamin D has been demonstrated at lead levels below 25 μg/dL.[7]

More importantly, it has been shown that neuropsychologic damage can be produced by lead levels below those previously felt to be safe. Pioneering work by Needleman et al in a group of asymptomatic children demonstrated that those with lead levels between 25 and 45 μg/dL had mean verbal IQ scores 4.5 points lower than children with lead levels of less than 25 μg/dL.[8] A later study by Smith et al[9] found significant differences in reading ability and IQ scores among three groups of children with even lower lead levels.

The Centers for Disease Control (CDC) currently defines an elevated blood lead level as 25 μg/dL or greater, with lead toxicity defined as a blood lead level of 25 μg/dL or higher accompanied by an EP level of 35 μg/dL or greater.[10] The American Academy of Pediatrics (AAP) also endorses this definition,[11] although the Academy is of the opinion that this definition may be again revised downward if further research demonstrates additional effects of low-level lead.[10]

At present, the recommendation of the CDC, endorsed by the AAP and the New York City Department of Health (NYC DOH), is that all children between the ages of nine months and six years be screened annually, with the first screening taking placed between 9 and 12 months of age. When EP screening tests identify a child with a value of 35 μg/dL or greater, immediate testing of venous blood is recommended to determine the child's serum lead level.[2] It is emphasized in the CDC recommendations that elevation of EP should not be considered diagnostic of lead toxicity until venous blood is analyzed, since iron deficiency also elevates EP.

Because of the abundance of dilapidated pre-World War II housing in New York City, free screening for all children in the city has been made available under the supervision of the Bureau of Lead Poisoning Control. A program has been in place for some years whereby clinics throughout the city send finger stick blood specimens on filter paper to the city laboratory for analysis of FEP and, if warranted, lead levels. More recently, hematofluorometers have been installed at many of the busier clinics in the city, allowing for rapid on-site determination of ZNP and the immediate implementation of follow-up procedures, such as the drawing of venous blood for lead-level determination. This obviates the need to recall patients when elevated ZNP levels are reported by the city lab several days after the initial appointment. Such recall has historically suffered from poor compliance rates, reported by some to be as low as 50%.[3]

The purpose of this study was to examine adherence to the guidelines established for the care and follow-up of children with elevated ZNP levels at a busy urban pediatric ambulatory care clinic. Attention was given to the demographics of the involved children, the immediate diagnostic and therapeutic steps taken, and provision for follow-up. An additional goal was to identify specific problems with the screening process at this site. Although the problems identified here clearly cannot be generalized to other screening programs, it is hoped that by highlighting the types of difficulties this site encounters, workers at other clinics may be encouraged to examine their own programs for similar types of problems.

METHODS

The pediatric ambulatory care clinic at St. Luke's Hospital in Manhattan served as the study site. This hospital serves a largely black and Hispanic population in west Harlem, Morningside Heights, and the area of Columbia University. The clinic is equipped with a hematofluorometer that analyzes a fingerstick blood specimen and provides a ZNP reading in several seconds. A centrifuge for determining spun hematocrit (Hct) levels is also available. All ZNP and Hct readings are recorded in a log book. This log was examined to determine the identities of all patients seen in the first four months of 1990 who had ZNP readings of 35 μg/dL or greater. The charts of these patients were obtained for review.

Each chart was analyzed for several characteristics: sex and ethnic background of the patient; ZNP, Hct, and venous lead levels (when obtained); and documentation of action taken on discovery of the elevated ZNP level. Five primary actions felt to be the most important in evaluating and following children with a high ZNP were considered. First, each chart was examined to determine whether a venous blood specimen was obtained for lead level analysis. Second, due to the high prevalence of iron deficiency in this population, and because elevated ZNP values more often indicate iron deficiency than lead toxicity, the provision of iron supplementation for those children with elevated ZNP levels was examined.

The third consideration involved scheduling a follow-up appointment for reassessment when the venous lead level became available from the city lab several days after the initial appointment. Follow-up also allowed for re-examination of the Hct in those patients who were in fact iron deficient. The final two items investigated were a review of potential exposures to lead in the child's environment, with particular attention to housing conditions, and a discussion of the child's diet, focusing on iron-rich foods and supplementation.

RESULTS

In the first four months of 1990, a total of 438 ZNP screening tests were performed. Of these, 66 (15.1%) resulted in values greater than or equal to 35 μg/dL. Charts of 60 of these patients were located and reviewed. The remaining six charts (9.1%) could not be located.

There were 39 (65%) females and 21 (35%) males studied. Thirty-seven (61.7%) children were Hispanic, 20 (33.3%) were black, and the ethnic background of the remaining three could not be determined from the charts. A two-sample test of means for independent samples was used to study these demographic groups. The mean ZNP value of the males (46.95 μg/dL) and females (45.10 μg/dL) were not significantly different ($t = 0.53$, $p = 0.60$), and while the mean ZNP value for Hispanics (48.0 μg/dL) was higher than that for blacks (42.65 μg/dL), the difference was not significant ($t = 1.51, p = 0.13$).

The primary focus of the study was on the five major interventions listed above. In total, 82 interventions were documented, or an average of 1.37 interventions per patient. A full 18.3% of the patients, 11 of the 60, received none of the five interventions. An additional 26, or 43.3%, received only one intervention. Fifteen, or 25.0%, had two interventions carried out, while six, or 10.0%, received three interventions, and two, or 3.3%, received four interventions. No patient received all five interventions (Table I).

TABLE I. Number of Interventions Received by Each Patient

Number of Interventions (Of 5 Studied)	Patients Receiving This Number of Interventions (%)	Mean ZNP (1 μg/dL)
0	11 (18.3)	39.27
1	26 (43.3)	44.23
2	15 (25.0)	45.07
3	6 (10.0)	54.67
4	2 (3.3)	79.50
5	0 (0.0)	
Total	60 (99.9)	

The average ZNP level for the 60 patients taken as a group was 45.75 μg/dL. The mean ZNP for those 11 patients who had received no interventions was 39.27 μg/dL. Patients receiving one intervention had an average ZNP of 44.23 μg/dL, whereas the mean ZNP values for patients receiving two, three, or four interventions were 45.07, 54.67, and 79.50 μg/dL, respectively (Table I). A scatter plot (Fig 1) of interventions and ZNP suggested a trend toward more interventions in patients with a higher ZNP. To examine this, a linear regression model for all 60 patients was constructed, with the number of interventions as the dependent variable and the ZNP value as the independent variable. An r value of 0.490 was obtained, a value which, by Student's t-test, is significantly different from zero ($t = 4.28, p < 0.005$). The r^2 value is 0.240, meaning that 24% of the variation in the number of interventions received can be accounted for by the magnitude of the ZNP.

The most frequently implemented intervention was the provision of iron therapy: 31 of the 60 patients, or 51.7%, were either already receiving iron supplements for previously diagnosed anemia (7 patients, 11.7%) or were started on iron at the visit in question (24 patients, 40.0%). Venous blood was drawn in 26 patients, or 43.3%. Adequate follow-up arrangements were documented in 21 visits, or 35.0%. Two patients, or 3.3%, had an exposure history documented, and a diet review was recorded for two others (Table II).

It was found that of the 26 patients who had venous blood drawn, results were eventually entered in the chart in 21 (80.8%). Ultimately, then, venous lead was determined for 21

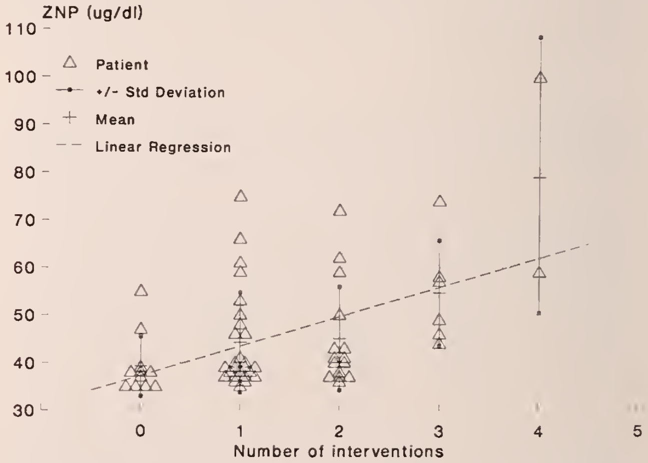

FIGURE 1. ZNP μg/dL vs number of interventions. Each triangle represents one patient, plotted to show the number of interventions received and the patient's ZNP level. The hash marks show the average ZNP for each cluster of patients. A linear regression for all 60 points is plotted. The correlation coefficient of the regression is 0.490, a value which is significantly different from zero ($t = 4.28$, $p < .005$). Note that no patient received all five of the interventions studied.

TABLE II. Number of Patients Receiving Each Intervention

Intervention	Number of Patients Receiving This Intervention (%)
Iron Started/Continued	31 (51.2)
Venous Blood Drawn	26 (43.3)
Follow-up Scheduled	21 (35.0)
Exposure History Taken	2 (3.3)
Dietary History/Advice	2 (3.3)

of the 60 patients, or 35.0%. The mean blood lead level for these 21 patients was 6.90 μg/dL, with a range of 2 to 17 μg/dL, all below 25 μg/dL, the CDC-defined level of elevated blood lead.

Discussion

In terms of detection of lead toxicity, drawing of a venous blood specimen for lead-level analysis and FEP confirmation is clearly the most important of the five interventions and is recommended in the guidelines set forth by the CDC, the AAP, and the NYC DOH. Only 26 (43.3%) of the patients in this study had venous blood drawn; furthermore, results of the lead level determination are not documented for five of these 26 patients.

Although none of the patients in this study were found to have lead levels above the limit of 25 μg/dL during the visits analyzed, two patients with elevated ZNP levels had elevated venous lead levels detected at other visits, and their situations illustrate the importance of ongoing screening. The first of these patients is a girl who was screened for the first time at age 17 months, during the study period. Her ZNP was 61 μg/dL, and she received only iron therapy; no venous blood was drawn. Three months later, during a routine return visit to the clinic, her ZNP was 46 μg/dL, and a venous blood specimen demonstrated a lead level of 35 μg/dL, with an FEP of 56 μg/dL, placing her in the category of lead toxicity. Had a venous lead been drawn at the time of the first visit, a toxic level might well have been discovered, and therapy implemented.

The second example is a boy who was eight years and seven months old at the time of the clinic visit in this study. He had been found to have lead toxicity 15 months earlier, with a lead level of 43 μg/dL. History revealed peeling paint in the bathroom of his family's apartment, and he was admitted for chelation therapy in December 1988. After discharge, his FEP remained elevated, in the 60–65 μg/dL range, although venous blood specimens taken in September and December of 1989 showed lead values of 23 and 22 μg/dL, respectively. At the clinic visit included in this study, his ZNP was 62 μg/dL. The chart indicates continuation of his iron therapy and the drawing of venous blood for determination of the lead level, but no result is listed. Fortunately, a repeat venous level drawn at his next regular appointment three months later demonstrated a lead level of 18 μg/dL. With lead values so close to the limit of 25 μg/dL on previous visits and a known history of lead toxicity, more vigorous follow-up would have been appropriate, as would discussion and documentation of abatement steps taken at the child's home.

The most frequently executed intervention in this study was the provision of iron therapy. Supplementation is important even in patients with normal Hcts for two reasons. First, as mentioned above, elevations in EP may be an early signal of iron deficiency, before true anemia develops. A study by Rajkumar et al[4] of 170 patients with lead levels greater than 30 μg/dL demonstrated that while only 9% were anemic, with a Hb of less than 11 g/dL, 29% were not anemic but showed biochemical iron deficiencies, with transferrin saturations of greater than 7%. Second, it has been demonstrated that iron-deficient patients have an increased rate of gastrointestinal absorption of lead,[12] and provision of iron supplements to these patients may help reduce their lead absorption. It thus seems reasonable to start iron therapy in all patients with elevated FEP values, at least until results of venous lead studies and perhaps iron studies become available.

Surprisingly, the least commonly documented interventions were those which would appear to be the easiest to carry out: discussion of potential exposures and diet, charted in only two patients each. It is certainly possible that some of these issues were discussed with other parents and not documented, and thus the 3.3% figure may well represent under-reporting of these interventions.

It is clear that with more than half of these 60 patients receiving none or only one of the five interventions, and none receiving all five, better implementation and documentation is needed. A standardized form or stamp, placed in the patient's chart by the lab technician when an elevated ZNP is found, could be used to help assure implementation and documentation. The physician or nurse practitioner would carry out and document on the form or stamped area the actions felt to be necessary. The form or stamp would have to be as simple and brief as possible to ensure good provider compliance, yet complete, to ensure that the necessary steps are taken.

It is recognized that the deficiencies detected in this study may well be site-specific, and that generalizations about other lead screening programs cannot be drawn from a study of one site. However, it seems possible that methodologic problems such as these may be affecting other screening programs, particularly those that, like the one studied, are extremely busy.

Federal legislation has recently been introduced calling for strict restrictions on the use of lead, including a ban on lead use in food cans and all paints, labeling and recycling of lead containing batteries, and a comprehensive program for the monitoring, detection, and removal of old lead-based paint and other lead hazards.[13] In announcing the proposed legislation, Senator Bill Bradley (D-NJ) emphasized the need for a three-pronged attack on the problem of lead in the environment: increased public awareness, research and testing, and restrictions on lead use.

Even with such sweeping restrictions, lead toxicity is likely to be an ongoing problem, particularly in inner-city areas with more sources for lead exposure. Screening programs will continue to play an important role in the detection of elevated lead levels, allowing children to be brought to medical attention before harmful effects develop. It is hoped that this study will highlight some of the potential problems with the screening process so that other lead screening programs can avoid problems such as these and can ensure high-quality care for our children.

Acknowledgments. The author thanks Joan Flanigan, MD, for her assistance in organizing and implementing this project, and Ernest Moy, MD, Kim-Thu C. Pham, MD, MPH, and E. Christopher Cone for their assistance in reviewing the manuscript.

REFERENCES

1. Mahaffey KR, Annest JL, Roberts J, et al. National estimates of blood lead levels: United States 1976–1980: Association with selected demographic and socioeconomic factors. *N Engl J Med* 1982; 307:573–579.

2. Centers for Disease Control: Preventing lead poisoning in young children. Atlanta: 1985.

3. *Guidelines for the detection and management of lead poisoning*. New York: 1988; NYC Department of Health publication no.

4. Rajkumar S, Geibel V, Devanagondi B, et al: The diagnostic significance of free erythrocyte protoporphyrin and the FEP/hemoglobin ration in plumbism. *NY State J Med* 1987; 87:542-545.

5. Hernberg S, Ikkanen J: Enzyme inhibition by lead under normal urban conditions. *Lancet* 1970; 1:63–64.

6. Piomelli S, Seaman C, Zullow D, et al: Threshold for lead damage to heme synthesis in urban children. *Proc Natl Acad Sci* 1982; 79:3335–3339.

7. Rosen JF, Chesney RW, Hamstra A, et al: Reduction in 1,2,5- dihydroxyvitamin D in children with increased lead absorption. *N Engl J Med* 1980; 302: 1128–1131.

8. Needleman HL, Gunnoe CF, Leviton A, et al: Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med* 1979; 300:689–695.

9. Smith M, Delves T, Lansdown R: The effects of lead exposure on urban children: The Institute of Child Health/Southhampton study. *Dev Med Child Neurol* 1983; 25(suppl 47):1–20.

10. Landrigan PJ, Graef JW: Pediatric lead poisoning in 1987: the silent epidemic continues. [editorial] *Pediatrics* 1987; 79:582–583.

11. American Academy of Pediatrics: Statement on childhood lead poisoning. *Pediatrics* 1987; 79:457–465.

12. Watson WS, Hume R, Moore MR: Oral absorption of lead and iron. *Lancet* 1980; 2:236–237.

13. Orr JS: Sweeping lead ban is proposed anew. *The Star-Ledger* [Newark, NJ], Feb 8, 1991, p 4.

Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman

CASE PRESENTATION

Robert N. Santella, MD (Attending Physician, Nephrology): This 72-year-old woman was well and active, regularly performing aerobics exercise two months prior to admission (PTA). Several weeks PTA, however, she became fatigued. Four weeks PTA, she noted a cough, with blood-tinged sputum. She was treated with oral erythromycin by her local physician. Her fatigue and leg weakness worsened, and diffuse arthralgias developed.

Two days PTA, shortness of breath, orthopnea, nausea, paresthesias, positional chest pain, and decreased urine output developed. She denied fever, chills, abdominal pain, and blood in the urine or stool. Her medications were spironolactone, isosorbide dinitrate, dipyridamole, aspirin, and ibuprofen, of which she took three or four doses.

Eleven months PTA, when in Florida, a computed tomography (CT) scan was obtained because of a palpable abdominal mass; there was radiologic evidence of cysts in both the liver and the kidneys. She had no history of renal disease; there was a question of chronic obstructive pulmonary disease and a question of coronary artery disease, neither of which we believe were objectively studied. At another hospital, shortly before she was transferred here, she was found to be uremic, with an elevated serum potassium of 8.4 mmol/L.

The patient was a smoker. Her mother had died of uremia, we believe. On admission physical examination at this hospital, she was alert and oriented. Her blood pressure was 180/90 mm Hg; pulse rate was 100 beats/min, but bradycardia developed rapidly; her temperature was normal. The positive physical findings included myoclonic jerking, elevated jugular venous pressure above 15 cm, diffuse wheezing throughout both lungs, a pericardial rub, a cardiac third sound (S3) gallop, carotid and abdominal bruits, a trace of peripheral leg edema, and non-palpable lower extremity petechiae. There was no adenopathy or synovitis.

The hematocrit was 28%; white blood cell count was 26,400/μL; prothrombin time was 12.1 sec, and partial thromboplastin time was 23.8 sec. Liver function tests were normal. The serum electrolyte levels were as follows: sodium, 120 mmol/L; potassium, 7.4 mmol/L; chloride, 87 mmol/L; and bicarbonate, 19 mmol/L. Her blood urea nitrogen (BUN) level was 116 mg/dL, and the serum creatinine was 9.2 mg/dL.

Soon after arriving in the Intensive Care Unit, severe wheezing and acute respiratory acidosis with CO_2 retention developed. The arterial blood gas determination indicated a pH of 7.12, a Pco_2 of 58 mm Hg, a Po_2 of 77 mm Hg, and bicarbonate was 18 mmol/L Hg. Prompt intubation was performed. The acute respiratory acidosis worsened the hyperkalemia, resulting in symptomatic bradycardia responding to calcium and immediate hemodialysis.

She produced less than 20 ccs of grossly bloody urine per day, with no urine on some days. She underwent hemodialysis and had several hypoxic episodes, most of which were associated with severe bronchospasms, and several were associated with a wide-QRS complex tachycardia, either supraventricular or ventricular. Several of these episodes precipitated cardiac arrest with good recovery except for the terminal episode.

The initial chest film was interpreted as showing mild, congestive heart failure; subsequent chest films demonstrated persistent interstitial edema and right-lower lobe consolidation consistent with pneumonia. The hemodynamic values as determined by Swan Ganz monitoring after her initial dialysis were essentially normal; the central venous pressure was 6–10 mm Hg, main pulmonary artery pressure was 24–31 mm Hg, pulmonary capillary wedge pressure was 14–16 mm Hg, cardiac output was 6–8 L/min. An echocardiogram showed normal left ventricular function, with no evidence of pericardial effusion or valvular disease. An abdominal ultrasound revealed normal appearance of both kidneys, one cyst in the right hepatic lobe, and no abdominal aortic aneurysm.

A nuclear renal scan indicated normal renal blood flow but very poor function, as one might expect from the elevated serum creatinine. Two antinuclear antibody (ANA) tests were ordered but did not reach the laboratory. The serum protein electrophoresis was essentially normal; a test for cryoglobulins was not ordered. The third component of serum complement (C3) was slightly low at 44 mg/dL (normal range, 50–120 mg/dL); the fourth component (C4) was 18 mg/dL (normal range, 15–50 mg/dL); the CH_{50} assay for total complement activity was 123 U/L (normal range, 100–300 U/L). Serologic tests for hepatitis were negative.

The patient underwent extubation and reintubation twice because of the previously described bronchospasm and the cardiac problems. Her unstable condition did not permit percutaneous renal biopsy or bronchoscopy. Daily, she produced purulent sputum not containing pathogenic organisms and, which for the first six hospital days, was not bloody. Broad-spectrum antibiotic therapy was begun; all bacterial cultures were negative for pathogens.

On the sixth hospital day, an antineutrophil cytoplasmic autoantibody (ANCA) test performed by indirect immunofluorescence on a serum specimen obtained on the first hospital day was reported positive at 1:128 titer in a cytoplasmic pattern. The test for serum antiglomerular basement membrane (anti-GBM) antibody was negative.

Based on a clinical conference held at Mary Imogene Basset Hospital, Cooperstown, NY, on March 5, 1991.

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Intravenous methylprednisolone, 500 mg/day, was administered on the sixth and seventh hospital days; intravenous pulse cyclophosphamide, 500 mg/m^2 of body surface, was administered on the sixth day. An open renal biopsy had been scheduled but was cancelled due to cardiovascular instability. Fever, hypotension, and hemoptysis developed rapidly, and on the seventh hospital day, the patient sustained a cardiac arrest with ventricular fibrillation. Resuscitation was unsuccessful.

Radiologic Presentation

Lawrence Barnowsky, MD (Attending Radiologist): The admission roentgenograms showed the heart to be within normal limits; the costophrenic angles were fairly clear; there were no signs of vascular congestion or of infiltrates. By the third hospital day, the chest film indicated the presence of congestive heart failure; vascular redistribution; and a small, right pleural effusion. By the next day, the infiltrate was unchanged, but the effusion had increased slightly; the cardiac silhouette remained unchanged, but the vascular congestion had begun to clear.

By the fifth hospital day, the chest film revealed more consolidation in the right lower lobe (RLL), together with an increase in the pleural effusion. There was no pleural fluid on the left side, and little in the way of vascular redistribution. At this point, the clinicians were beginning to suspect the presence of an inflammatory process rather than pure edema.

In the last available film, the patient was intubated; the pleural fluid persisted as a hazy shadow in the RLL area. The heart was no further enlarged, and there was very little in the way of vascular congestion.

This series of chest films indicated that the patient was progressing into congestive heart failure, developing an RLL ground-glass haziness which began to clear. The differential diagnosis of the RLL infiltrate would include localized edema and pneumonia, but we know that there was a question of vasculitis. Another possibility would be the presence of localized hemorrhage.

The ultrasound of the left kidney showed a reasonably good renal cortex, with a normal hypoechoic appearance and normal echosonicity in the renal sinus. There was no evidence of hydronephrosis. The right kidney also showed a hypoechoic cortex, with an essentially normal appearance and no hydronephrosis. The sonogram of the liver showed a solitary liver cyst, probably 3–4 cm in size.

Dr Santella: In summary, we have an elderly woman with a severe, acute systemic illness dominated by renal failure and a pulmonary process. One of the major questions was the etiology of her pulmonary disease. Did she have an infectious process; a pneumonia with a superimposed, acute tubular necrosis (ATN); or did she have a systemic illness involving the lungs and the kidneys?

I will talk briefly about the differential diagnosis of acute renal failure, and during the remaining time I will discuss the ANCA test. We briefly considered that this patient's illness might be related to some sort of cystic renal disease, given the family history and the report of previous abdominal CT scan; but our imaging studies readily dismissed that possibility.

The differential diagnosis of acute renal failure can be divided into prerenal, postrenal, and intrinsic renal disease including ATN, acute interstitial nephritis, and acute glomerulonephritis.[1] The patient had no evidence of prerenal disease; although she had symptoms and signs and an initial chest film consistent with congestive heart failure, no other objective measurements supported[3] this diagnosis. The cardiac output was not low, the pulmonary capillary wedge pressure was not high, and the echocardiogram was normal. The patient's intravascular blood volume was not depleted, and there was no evidence of the hepatorenal syndrome. The hemodynamic picture was consistent with sepsis, which can cause shunting of the blood away from the kidneys, although multiple bacterial cultures did not grow organisms.

We entertained the possibility of renal artery occlusion only because she was anuric. The nuclear study, however, indicated reasonable renal blood flow. Similarly, there was no evidence for postrenal disease, as evidenced by the normal ultrasound of both kidneys.

Turning to renal causes, vascular disease was a possibility. Atheroembolic disease can occur spontaneously in the setting of severe atherosclerosis, but usually follows aortic surgery or catheterization. She did have vascular bruits, but spontaneous atheroemboli are not common, and the condition tends to be a slowly progressive disease, relentless but not very acute.

The patient did not have malignant hypertension. Her illness did not fit the clinical picture of the hemolytic-uremic syndrome. Acute renal vein thrombosis was a possibility, but there was no reason to suspect it.

Interstitial nephritis is a common cause of acute renal failure and can be idiopathic. There can be allergic causes, but the patient was not taking drugs that commonly cause classic allergic interstitial nephritis, such as non-steroidal anti-inflammatory drugs (NSAIDS). Ibuprofen is a NSAID know to induce interstitial nephritis but the patient had taken only a few doses. Typically, interstitial nephritis caused by NSAIDS occurs after the patient has been taking the drug for many days to months; 70% of such patients have significant edema, with the nephrotic syndrome a common result. She did not have this clinical picture. NSAIDS can cause acute renal insufficiency due to functional microvascular changes in the kidney, but that condition should be rapidly reversible on withdrawal of the offending agent. In addition, if such a condition had developed, she should have had some predisposing prerenal type of illness.

The patient could have had an infectious interstitial nephritis, that is, pyelonephritis; but this condition does not cause the degree of renal insufficiency seen in this patient, unless accompanied by renal papillary necrosis, for which there were no risk factors; or by urinary tract obstruction. There was no obvious evidence of infiltrative renal disease, not the slowly progressive sort that rarely causes acute renal failure, such as that caused by lymphoma.

ATN is a common cause of acute renal failure, but uncommon as a condition presenting initially outside the hospital. In a recent series of patients with acute renal failure that presented outside the hospital, ATN was the cause in only one of 70 patients.[2] Furthermore, the patient we are discussing today did not have an obvious cause for ATN, such as exposure to a toxin or evidence of rhabdomyolysis; there was, however, the possibility of ischemic-induced ATN on the basis of a large-vessel vasculitis.

Rapidly progressive glomerulonephritis (RPGN) can cause acute renal failure in the elderly.[3] The causes of RPGN include membranoproliferative glomerulonephritis, post-infectious glomerulonephritis, systemic lupus erythematosus (SLE), cryoglobulinemia, Goodpasture's syndrome, IgA/Henoch-Schonlein purpura, the hemolytic-uremic syndrome, polyarteritis, and Wegener's granulomatosis. The latter two conditions have recently become known as ANCA-associated glomerulonephritis.

Several aspects of this patient's disease led us to believe that a systemic vasculitis was the cause of her acute renal failure, ie, the systemic nature of the disease, the lack of objective findings of heart failure, the purulent sputum with negative Gram's stains and negative cultures for organisms, the other negative bacterial cultures, and a high-titer positive ANCA test.

I will not discuss the entire differential diagnosis of RPGS because I wish to devote the remainder of my discussion to the ANCA test and its relationship to the differential diagnosis of glomerulonephritis.

The ANCA test is a serologic marker for a spectrum of disease entities, ranging from multisystemic vasculitis to renal-limited disease. The former includes such entities as Wegener's granulomatosis with lung involvement, whereas the latter includes conditions such as idiopathic or crescentic glomerulonephritis, so-called renal vasculitis. The pathologic features of the ANCA-positive diseases are uniform, in that they present with necrotizing vascular inflammation with essentially no immunocomplex deposits (pauci-immune disease).

The ANCA autoantibodies react with the cytoplasmic antigens of human neutrophils and monocytes. Typically, the ANCA test, performed by indirect immunofluorescence, shows two staining patterns: the autoantibody stains either the cytoplasm of the neutrophils (C-ANCA) or the perinuclear region (P-ANCA). The antigen for which the cytoplasmic pattern offers major specificity is a 29-kD protein, protease 3, which is found in the primary granules of the neutrophil. For the perinuclear pattern, the antigen is myeloperoxidase.[4]

In addition, an enzyme-linked immunosorbent assay (ELISA) test is thought to be confirmatory and possibly more specific. The patient we are discussing today had a high-titer cytoplasmic pattern ANCA by immunofluorescence, with confirmation by a positive ELISA test.

Drs Falk and Jennette at the University of North Carolina at Chapel Hill (UNC) have reported the largest number of patients with either positive C-ANCA or P-ANCA tests. They initially studied 70 patients, all with positive ANCA tests and with biopsy-proven pauci-immune necrotizing and crescentic glomerulonephritis;[5] they now follow 90 such patients.[6] The mean age in their series was 55 years; the percentage of male patients was approximately equal to that of female patients. The white race was more heavily represented than the black, in a ratio of 6:1, much higher than the 2–3.5:1 ratio of white-to-black in the Medical Center's referral area. The onset of illness had a seasonal variation, being much more common in the winter than in the summer months. SLE was ruled out by negative ANA and negative anti-DNA testing.

Twenty percent of this patient group developed both renal and extrarenal disease but without lung involvement; one might designate this condition as microscopic polyarteritis nodosa (PAN). One third of the patients had renal and pulmonary disease without sinus involvement; 18% had renal, pulmonary, and sinus disease, which we might think of as Wegener's granulomatosis. In 29% of the patients, the disease was limited to the kidneys, presenting as idiopathic RPGN or renal vasculitis.

The clinical manifestations in this group of patients varied: 94% of the patients experienced a flu-like prodromal illness that occurred days to weeks before the overt vasculitis or glomerulonephritis was diagnosed; 25% had upper respiratory tract disease; almost half of the patients had arthralgias; 25% had myalgias; and 7% had myocardial infarctions at or close to the time of presentation. One half of the patients had pulmonary involvement, and 21 of these 37 patients experienced hemoptysis accompanied by pulmonary infiltrates on chest films. Chest films in the 16 patients without pulmonary hemorrhage showed nodules, cavities, or persistent infiltrates unexplained by infection or congestive heart failure. Of the 11 lung biopsy specimens obtained, four showed necrotizing granulomatous inflammation, and seven showed capillaritis. In the overall group, the mean serum creatinine was elevated, at 6 mg/dL; 25% of the entire group required dialysis.

A larger series of patients with antineutrophil cytoplasmic autoantibodies, C-ANCA only, has been reported jointly by investigators from the Mayo Clinic and from Germany.[7] They reported 277 patients with Wegener's granulomatosis (222 biopsy-proven cases) and 1,657 controls (1,033 normal or without related disease). They attempted to determine the sensitivity and specificity of C-ANCA for Wegener's granulomatosis.

The sensitivity and specificity of the ANCA test vary depending on the definition of disease for which one is attempting to establish a diagnosis, the extent of the disease and its activity, and the pattern of ANCA. The true sensitivity and specificity of the ANCA test have not yet been clearly established. Many of the controls used in the Mayo/German study were either normal volunteers or patients with unrelated disease, rather than patients in whom one might be seriously considering the diagnosis of Wegener's granulomatosis, a vasculitis, or RPGN. The UNC group reported patients with glomerulonephritis and known positive ANCA tests; there was no control group in this study. The investigators looked at the spectrum of clinical manifestations rather than the sensitivity and specificity of the test.

If one uses only the C-ANCA pattern as a positive indicator, the test is highly sensitive and specific for a narrow disease spectrum, namely classic Wegener's granulomatosis. In a specific patient, we may be suspecting active generalized Wegener's granulomatosis, with necrotizing granulomatous pulmonary disease, respiratory insufficiency, diffuse glomerulonephritis with crescent formation, and rapidly progressive renal failure. In such a case, the sensitivity and specificity of the ANCA test are extremely high, at 93%–96% and 98%–99%, respectively, when the C-ANCA is positive, as reported by the Mayo/German group.[7] In that report, the sensitivity of the C-ANCA test depended on disease activity and extent, being 60%–67% positive for patients with active loco-regional disease. In the UNC series, all four patients with classic Wegener's granulomatosis, with biopsy-proven, necrotizing granulomatous inflammation in the lungs, tested positively for C-ANCA.[5]

The UNC group has looked at a broader spectrum of disease centered around the presence of glomerulonephritis; in this patient group, both the C-ANCA and P-ANCA tests were positive. In this series, Falk et al found that 50% of the patients with both renal and pulmonary disease had a positive perinuclear pattern.[5] Of the 18 patients in this series who presented with renal vasculitis, that is, crescentic glomerulonephritis without extra-renal lesions, 15 had the perinuclear pattern; of the patients with PAN, that is, glomerulonephritis with nonpulmonary extra-renal disease, 50% had a perinuclear pattern. As a consequence, if one ignored the implications of a positive P-ANCA test and assumed that only a positive C-ANCA test was significant, serious vasculitis warranting aggressive immunosuppressive treatment might be missed.

Among patients with crescentic glomerulonephritis, Falk found the specificity of the ANCA test for pauci-immune glomerulonephritis to be 92%, distinguishing it from immunocomplex renal disease.[8] The ANCA test was found to be positive in 87% of patients with pauci-immune glomerulonephritis with crescents or necrosis; positive in 11% of patients with non-lupus, immunocomplex glomerlonephritis; and negative in all 24 patients with anti-GBM glomerulonephritis.[9]

The ANCA test has been reported positive in cases of lupus erythematosus, most often with a perinuclear pattern, but at times also with a cytoplasmic pattern. There are also individual case reports of ANCA positivity in inflammatory bowel disease, Churg-Strauss syndrome, anti-GBM disease, Behcet's disease, hydralazine-induced vasculitis, and Kawasaki disease.

There appears to be no difference in survival in renal-limited disease compared with the entire group or with those who have pulmonary disease. There is no difference in survival between patients demonstrating the C-ANCA pattern and those with the perinuclear pattern. The limited data available suggest that treatment of a patient with widespread Wegener's granulomatosis and a C-ANCA pattern would have the same outcome as treatment of a patient with renal-limited disease and a perinuclear pattern.[5]

Half of the deaths in the series studied occurred very early in the course of the disease, before or just at the start of therapy, as was the case with the patient under discussion today. The UNC group found only two significant predictors of outcome in Wegener's granulomatosis: the initial serum creatinine level in relation to poor long-term outcome and the presence of massive pulmonary hemorrhage in relation to poor short-term prognosis.[5] Age, sex, severity of pathology, immunofluorescent staining pattern, duration of disease, clinical group, and treatment modalities did not assist in predicting the outcome. There was no difference in outcome between various treatment regimes: oral steroids with intravenous cyclophosphamide, oral steroids with oral cyclophosphamide, or corticosteroids alone. There was a 75% patient survival and renal recovery based on a 24-month follow-up of treated patients; results of patients with renal-limited disease fared as well as the patient population as a whole, and as well as with those with pulmonary and renal disease.[5] Most experts in the field would treat ANCA-associated vasculitis with steroids plus cyclophosphamide; however, there is an ongoing debate as to which route of cyclophosphamide therapy administration is preferable.[6,10,11]

Eric J. Del Giacco, MD (Attending Physician, Internal Medicine): Were there patients in that study with as severe renal disease as the patient we are discussing today, but who experienced a return of renal function?

Dr Santella: The data offered were insufficient to answer that question. The mean serum creatinine in the group of patients was 5.6 mg/dL, ±4 mg/dL; the reported range among the patients was 0.7–21.6 mg/dL. There was a higher mortality among those who presented with the worst renal function.

Dr Del Giacco: Do we know if any patient in those studies recovered with treatment, after having experienced the same degree of renal failure as did this patient?

Dr Santella: We do not know that. In the North Carolina experience, six of the 12 patients requiring dialysis during the initial phase of therapy recovered renal function. There are case reports of patients with renal-limited vasculitis who required dialysis and were treated with pulse steroids, and then recovered enough renal function so that dialysis was discontinued. These cases preceded the availability of the ANCA test, and therefore we cannot be sure that they necessarily represented the same disease as occurred in this patient.

The ANCA test is changing the way we think about rapidly progressive glomerulonephritis. One can divide RPGN into three categories:

1. Immunocomplex disease, such as lupus nephritis or post-streptococcal glomerulonephritis. Most of these instances are associated with depressed serum complement levels. The ANCA test is negative in this group.
2. Anti-GBM disease such as Goodpasture's syndrome. The histologic feature in such cases is linear immunofluorescent staining, and a circulating anti-GMB antibody is often present.
3. ANCA-associated glomerulonephritis. This is associated with a spectrum of diseases recently termed ANCA-associated vasculitis, including necrotizing crescentic glomerulonephritis with or without systemic vasculitis and with or without a range of pulmonary involvement.

CLINICAL DIAGNOSIS

ANCA-associated vasculitis with rapidly progressive glomerulonephritis and pulmonary hemorrhage or inflammation.

PATHOLOGY PRESENTATION

Harlan D. Alpern, MD (Pathologist-in-Chief): The postmortem examination of the kidneys demonstrated slight enlargement to 180 g each. Each kidney contained multiple small cysts, measuring up to 1 cm in diameter. The cause of the renal failure was evident on microscopic analysis (Fig 1). More than 95% of the glomeruli demonstrated crescentic, rapidly progressive glomerulonephritis, characterized by a striking proliferation of the cells lining Bowman's space and in the glomeruli, together with fibrin deposition, focal necrosis, a relatively mild endocapillary proliferation,

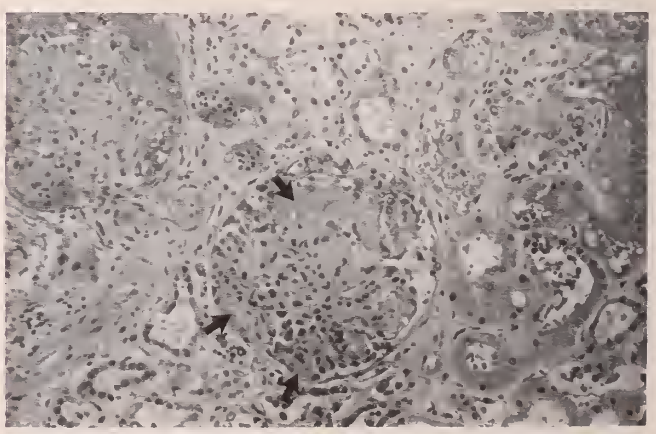

FIGURE 1. Crescentic, rapidly progressive glomerulonephritis, with proliferation of cells lining Bowman's space (lower arrows) and fibrin deposition (upper arrow). (hematoxylin and eosin stain; original magnification × 100)

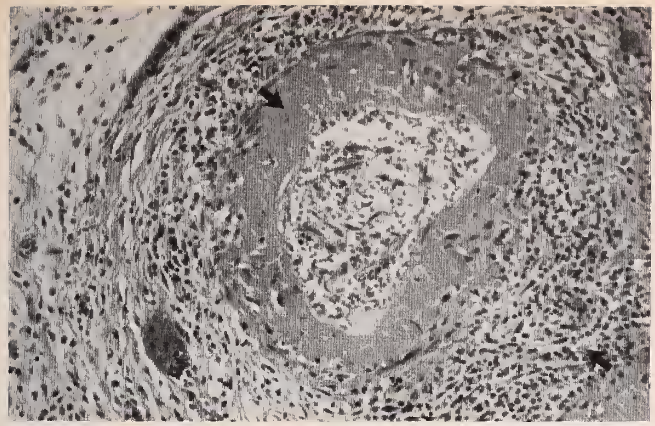

FIGURE 2. Vasculitis with fibrinoid necrosis of the vessel wall (upper arrow) and surrounding inflammation (lower arrow). (hematoxylin and eosin stain; original magnification × 100)

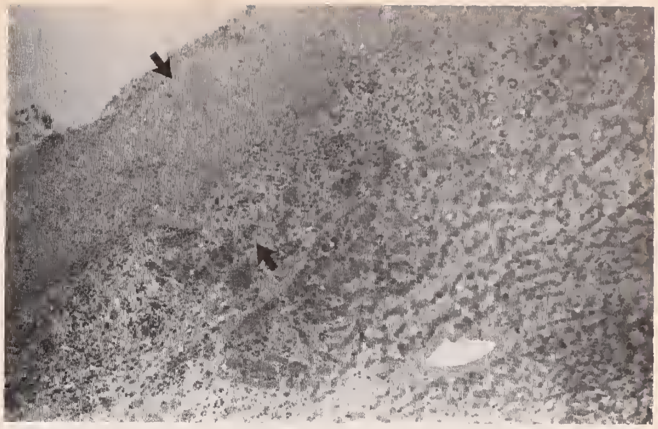

FIGURE 4. Wall of large hepatic cyst lined by fibrin (upper arrow) and inflammatory debris (lower arrow). (hematoxylin and eosin stain; original magnification × 40)

and occasional neutrophils. There was also secondary ATN, with necrotic cells lining the tubules and with necrotic material in their lumens. In the interstitium, the slight inflammatory infiltrate was dominated by lymphocytes and edema. The capillary vasculature was congested.

Vasculitis was not seen in the kidneys or in the lungs but was present in vessels in two extrarenal locations: in tissue adjacent to one oviduct in the mesosalpynx, and in the perivesicular fat of the urinary bladder (Fig 2). These small arteries demonstrated changes associated with arteritis, with fibrinoid necrosis of the vessel walls, and with transmural and especially adventitial inflammatory reaction.

The lungs were heavy, the left having a mass of 810 g, and the right having a mass of 1,040 g. The cut surfaces were dark red, and the microscopic examination disclosed widespread intra-alveolar hemorrhage (Fig 3), as well as abundant iron in alveolar macrophages in the alveoli, indicating that the blood was not all recently extravasated. There was also evidence of pulmonary emphysema, suspected clinically, with large dilated alveolar air spaces, principally in the apices of the lungs.

As clinically suspected, there was severe coronary heart disease; the lumen of the right coronary artery was markedly compromised by thick atherosclerotic plaques, with fibrosis, calcification, and deposition of lipids. As a result of the coronary heart disease, the heart had a mass of 580 g, approximately twice normal. There was dilatation of the right ventricle representing acute right heart failure; there was marked hypertrophy of the left ventricular wall.

Histologic examination did not demonstrate acute or old myocardial infarction, but did identify areas of chronic ischemic myocardial fibrosis with numerous patchy areas of myofibers replaced by scar, and hypertrophy of adjacent fibers.

In the pylorus, an ulcer, 2.5 cm in diameter, had perforated through the gastric wall, so that the base of the ulcer was in the omentum; along the surface of the ulcer was a fibrino-purulent pseudomembrane.

The liver contained numerous cysts, measuring up to 4 cm in dimension, with one exception; they contained a clear serous fluid. Histologic study of these cysts demonstrated fibrin connective tissue with benign biliary glandular cells along some surfaces, and normal liver tissue subjacent to the cyst walls. In the left lobe of the liver, there was a large cyst, 14 cm in maximum dimension, positioned so that it blocked the perforation of the pyloric ulcer. A probe could be passed through this cyst into the perforation and thereby into the lumen of the stomach. This cyst contained a thick greyish fluid; its wall demonstrated fibrin and inflammatory debris along its inner surface (Fig 4). A section stained by GMS method demonstrated large numbers of budding yeast forms and pseudohyphae of *Candida* that had colonized the cyst (Fig 5).

Dr Santella: There was no evidence of systemic candidiasis?

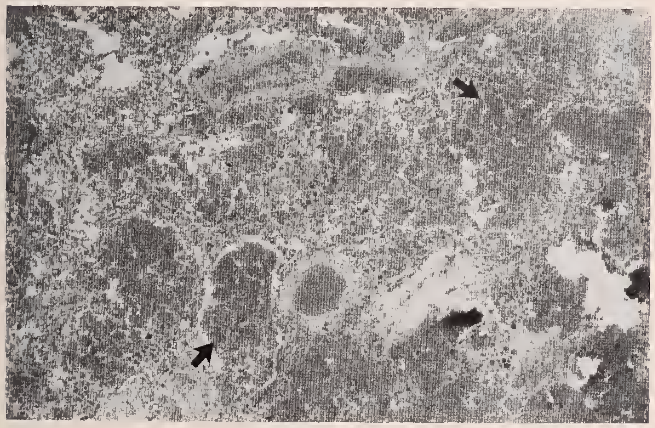

FIGURE 3. Intra-alveolar pulmonary hemorrhage (typical areas at arrows). (hematoxylin and eosin stain; original magnification × 40)

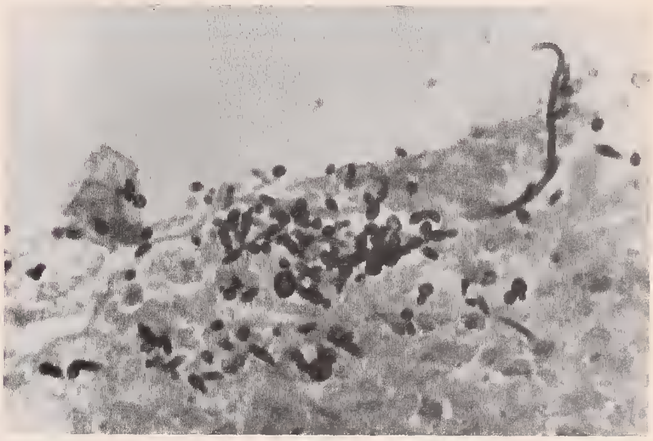

FIGURE 5. Fungal organisms (*Candida* species) colonizing inner aspect of large hepatic cyst. (hematoxylin and eosin stain; original magnification × 100)

Dr Alpern: There was no evidence of systemic candidiasis or of infection in any other location, other than this large cyst in the liver. In summary, the pathologic features are those of an anti-neutrophilic, cytoplasmic, antibody-associated, crescentic, and rapidly progressive glomerulonephritis, accounting for the renal failure. An associated extra-renal arteritis of the polyarteritis type was seen only in two locations, both in the genitourinary tract. Intrapulmonary hemorrhage was present. The patient's final demise resulted from an arrhythmia.

William Guiney, MD (Attending Pathologist): What do you think produced the pulmonary hemorrhage? Could she have aspirated blood?

Dr Alpern: The blood did not appear to have been aspirated. The cause of the pulmonary hemorrhage remains uncertain. Undetected pulmonary vasculitis is the most probable cause; cardiac failure as a cause is less likely.

FINAL ANATOMIC DIAGNOSES

The final diagnoses includes the following:

- ANCA-associated rapidly progressive crescentic glomerulonephritis involving both kidneys, with secondary ATN.
- Focal extrarenal arteritis, polyarteritis type, perivascular, of the urinary bladder fat and mesosalpinx.
- Diffuse intra-alveolar pulmonary hemorrhage in both lungs.
- Atherosclerosis of the right coronary artery, with myocardial fibrosis, left ventricular hypertrophy (580 g), and right ventricular dilatation.
- Pyloric ulcer, with perforation walled off by a large hepatic cyst, with colonization by *Candida*.
- Multiple cysts in the liver and kidneys.

REFERENCES

1. Brezis M, Rosen S, Epstein FH: Acute renal failure, in Brenner BM, Rector FC Jr (eds): *The Kidney,* ed 4, Philadelphia, WB Saunders Co, 1991, pp 993–1061.
2. Levison SP: Renal disease in the elderly: The role of the renal biopsy. *Am J Kidney Dis* 1990; 16:300–306.
3. Kaufman J, Dhakal M, Patel B, et al: Community-acquired acute renal failure. *Am J Kidney Dis* 1991; 17:191–198.
4. Jennette JC, Falk RJ: Antineutrophil cytoplasmic autoantibodies and associated disease: A review. *Am J Kidney Dis* 1990; 15:517–529.
5. Falk RJ, Hogan S, Carey TS, et al: Clinical course of antineutrophil cytoplasmic autoantibody-associated glomerulonephritis and systemic vasculitis. *Ann Intern Med* 1990; 113:656–663
6. Falk RJ, Jennette JC: ANCA-associated glomerulonephritis (letter). *Ann Intern Med* 1991; 114:430.
7. Nolle B, Specks U, Ludemann J, et al: Anticytoplasmic antoantibodies: Their immunodiagnostic value in Wegener's granulomatosis. *Ann Intern Med* 1989; 111:28–40.
8. Falk RJ: ANCA-associated renal disease. *Kidney Int* 1990; 38:998–1010.
9. Jennette JC, Wilkman AS, Falk RJ: Antineutrophil cytoplasmic autoantibody-associated glomerulonephritis and vasculitis. *Am J Pathol* 1989; 135: 921–930.
10. Hoffman GS, Leavitt RY, Fleisher TA, et al: Treatment of Wegener's granulomatosis with intermittent high-dose intravenous cytophosphamide. *Am J Med* 1990; 89:403–410.
11. Cupps TR: Cyclophosphamide: To pulse or not to pulse? *Am J Med* 1990; 89:399–402.

Regulated medical waste: Is any of it infectious?

Sean F. X. Dugan, LLM

A federal judge recently sentenced defendants found guilty of improperly disposing of medical waste in Staten Island. The Court noted that it would cost at least $15 million, and probably something closer to $30 million, to clean up the resulting illegal dump site. The infectious waste generators were New York physicians, and two New York hospitals. Judge Motley wrote that "by disposing of the medical waste illegally, defendants exposed the waste generators to both civil and criminal liability, although defendants had assured such generators that they were duly licensed."[1]

What is it about infectious medical waste that makes innocent physicians and hospitals generating this waste absolutely liable for its improper disposal?

AN HISTORICAL LEGAL PERSPECTIVE

In 1976, Congress ordered the Environmental Protection Agency (EPA) to develop environmentally sound methods to manage solid waste which, because of its infectious characteristic, may either significantly contribute to an increase in death, or an increase in serious irreversible or incapacitating reversible illness, or pose a substantial present or potential hazard to human health or the environment when improperly managed.[2] Infectious waste is defined as waste containing pathogens of sufficient virulence and quantity that exposure to the waste by a susceptible host could result in an infectious disease.[3]

The EPA subsequently could identify no correlation between infectious medical waste and disease among persons who were exposed to that waste,[4] and regulations were never promulgated to implement the 1976 Act. The EPA did issue a *Draft Manual for Infectious Waste Management* in the fall of 1982, which was refined in the summer of 1986 with the publication of *EPA Guide for Infectious Waste Management.* Nevertheless, the EPA still issued no regulations dealing with infectious waste management which had the binding effect of law, nor did the EPA come forward with any evidence that there was in fact a relationship between the management or mismanagement of infectious medical waste and infectious disease produced in the population that came into contact with that infectious waste.

There matters remained until the summer of 1988 when needles, insulin-type syringes, blood bags, vials, and bandages washed up on some New York and New Jersey shorelines. Only about 1% of the waste found on the beaches consisted of insulin-type syringes and blood vials. The remaining 99% of this solid waste consisted of household trash and garbage, wood, and miscellaneous debris.[5] However, the public outcry over this waste prompted Congress to pass the Medical Waste Tracking Act of 1988 (MWTA),[6] which added a new Subtitle J to the Resource Conservational and Recovery Act (RCRA). The implementing regulations by the EPA [40 CFR Parts 22 and 259 (1989)] specifically note that "currently available data suggest that medical waste does not generally pose a significant potential for disease transmission."[7]

The sunset provision which Congress wisely wrote into the MWTA caused that Act to expire in June of 1991. One of the mandates of the MWTA was for the EPA to obtain information referable to any present or potential threat to human health posed by medical waste.

GOVERNMENT REPORTS

In October of 1988, the Office of Technology Assessment (OTA) of the US Congress investigated the management of infectious waste. OTA was able to point to no evidence that the management or mismanagement of infectious medical waste posed risks to the community.[8]

The General Accounting Office (GAO) is the auditing and investigative arm of Congress. The GAO studied the issue of risks posed by medical waste. It concluded that the existence of any threat to the general public has not been determined. GAO further stated that whether medical waste is any more infectious than ordinary garbage is an open question.[9]

The New York State Department of Health observed that "truly infectious waste (as defined by scientific potential for causing infection and disease) . . . is routinely handled every day inside and outside of health facilities all over the world without causing disease."[10]

In May 1990, pursuant to a directive contained in the MWTA, the EPA presented its First Interim Report to Congress, on *Medical Waste Management in the United States.*[11] The EPA identified the key question as which, if any, components of the medical waste stream pose true health hazards and therefore require regulatory control. In this Interim Report, the EPA simply raised the question and recognized that most experts in the field were of the opinion that actual threats to the general public are not likely to be posed by medical waste, even when such waste is mismanaged or improperly disposed. However, the EPA noted the obvious: that some medical waste may contain pathogens in concentrations sufficient to cause disease in susceptible individuals. Thus, the EPA deferred to the Agency for Toxic Substances and Disease Registry (ATSDR) of the Public Health Service, US Department of Health and Human Services, which had been ordered by Congress, when it passed the MWTA, to report to Congress on the health effects of medical waste. However, the EPA would itself investigate the issue, in conjunction with the ATSDR.

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ATSDR presented its Report to Congress in September of 1990.[12] The Agency flatly stated that aside from used sharps, "no HIV infection reported in the scientific literature is associated with medical waste," and that "to date, no cases of medical waste—related HBV infection or disease have been reported in the scientific literature." The Agency went on to state that the "scientific literature does not report any injury data for medical waste management except injuries associated with sharps."[12(p4.6)] In summary, ATSDR concluded from its investigation that the "scientific literature does not document a single infection resulting from contact with non-sharp medical waste."[12(p4.10)] The ATSDR concluded that the public health is not likely to be adversely affected by medical waste generated in the traditional health care setting. Although communicable diseases theoretically could be transmitted by medical waste, the probability of such transmission is remote.

The EPAs Second Interim Report to Congress, *Medical Waste Management in the United States* (pursuant to the MWTA of 1988), simply noted that its own studies and analyses concerning disease transmission from regulated medical waste are continuing and will be reported to Congress in its final report, now scheduled to be issued this winter.

Non-Sharp, Regulated Medical Waste

It is well documented that current hospital waste disposal practices, still leaving aside the sharps issue, have not caused disease in the hospital or in the community.[13] There is no epidemiologic evidence that hospital waste disposal practices have caused disease in the community.[14,15] The Centers for Disease Control (CDC) reiterated its finding, in August of 1987, that "there is no epidemiologic evidence that hospital waste has caused disease in the community as a result of improper disposal."[16]

As has often been stated in the literature, for disease to be transmitted from infectious waste to humans, several links in a chain of events must all be present. The factors in the chain of infection that are necessary for disease transmission include dose, susceptible host, portal of entry, presence of viable pathogens, and virulence.[17] Transmission will occur only when all these factors are present simultaneously. Dose is the presence of a sufficient quantity of infectious agents to cause disease in a susceptible host. New York defines infectious agents as those organisms that cause disease in humans (Environmental Conservation Law [ECL] Section 27-1501). A susceptible host offers a favorable environment for survival of the pathogens. The portal of entry permits the infectious agent to gain entry into a susceptible host. A portal of entry for the disease organisms is denied when universal precautions are observed, for example by placing an appropriate barrier between the person exposed to infectious waste and the pathogens associated with that waste. This barrier includes, for instance, the wearing of gloves when regulated medical waste is handled.[18]

Since 1983, at least, it has been conceded that hospital waste is no more infectious than normal household garbage.[19] This finding was supported by a separate study demonstrating that hospital wastes were not more contaminated than household refuse.[20] In 1984, these results were confirmed by research conducted for the EPA by the University of Cincinnati.[21]

There is no evidence that mismanaged infectious laboratory waste has caused human disease.[22(p573–586,579)] The CDC and the National Institutes of Health jointly reported that laboratories working with infectious agents have not been shown to represent a threat to the community.[23]

The disposal of infectious wastes in New York's 177 landfills has been banned in New York State since 1988 (Chapter 654 of the Laws of 1988). However, the scientific evidence indicates that land-filling of infectious waste is environmentally safe, as most pathogens are inactivated by the increase in temperature and by the acidic environment found in landfills, as well as by salt in the landfill which inactivates many pathogens.[12(ppE11,7.12),22(pp573–586)]

None of the twelve municipal solid waste incinerators operating in New York accepts infectious waste. No presently operating on-site hospital incinerator can meet the air pollution regulations which went into effect in New York State on January 1, 1992 (ECL Section 19-0306; 7 the Official Compilation of the Codes, Rules and Regulations of the State of New York [NYCRR] 219). Thus, the 300 or so hospitals now operating incinerators in New York must either spend from $500 thousand for a 300 bed hospital to several million dollars for a large institution to upgrade their incinerators to meet the state's specifications, or pay exorbitant fees to have regulated medical waste shipped off-site for disposal.

There are approximately 5,000 medical waste incinerators operating in United States hospitals. There is not one documented case of an individual becoming ill from infectious disease as a result of exposure to the incineration process of medical waste disposal. Whether or not the regulated medical waste is sterilized as a result of the incineration process,[12(p7.5)] the pathogen level in the waste is clearly reduced to a level from which infection is neither likely nor documented in persons exposed to the emissions from the flue, or exposed to the ash.[24,25]

Whether or not some viable pathogenic organisms survive the incineration process is irrelevant; all environments except those maintained under sterile conditions harbor some populations of pathogenic microorganisms. To be capable of producing an infectious disease, the microorganisms must be of sufficient virulence and of sufficient dose, and they must have a portal of entry to the susceptible person. There is no evidence that this is the case with reference to incinerated regulated medical wastes. The residue from the incineration process has not been reported to cause infectious disease in man.

Pathogens may be released into the environment from incineration so as to come in contact with man basically by two routes, discharge air streams and post-incineration residue. The EPA Office of Air and Radiation, Office of Air Quality Planning and Standards, issued a Final Report entitled "Hospital Waste Combustion Study—Data Gathering Phase" in December of 1988 (EPA-450/3-88-017). It noted that no statistically significant difference could be found between the ambient air mean level of bacteria as opposed to the pathogenic bacteria found in the gas of a hospital waste incinerator stack.

As there is no evidence that infectious disease is transmitted from infectious medical waste, the CDC sanctions the disposal of infectious medical waste by grinding and flushing into the sewer.[16(p12S)] The EPA recommends the same disposal practice, as long as secondary treatment is available.[26] The ATDSDR acknowledges that infectious medical waste discarded into the sanitary sewer does not present any public health effects over and above those associated with the disposal of non-infectious medical waste into the sewer.[12(p7.13)] The State codified this scientific fact in the Public Health Law (PHL), which permits the discharge of regulated medical waste into the sewerage system (PHL Section 1389-dd[1][b]).

There is neither epidemiologic nor microbiologic evidence supporting the contention that waste from hospitals, other health care facilities, or clinical or research laboratories is more infectious than residential wastes. There is no documented epidemiologic evidence implicating hospital waste with disease transmission in the community. Neither is there evidence that presently accepted waste disposal methods pose a risk of human immunodeficiency virus (HIV) transmission.[27]

Even in view of the above-referenced, generally accepted scientific and medical evidence, fees obtained from the medical community by infectious waste management services continue to burgeon. Before the summer of 1988 beach wash-ups, fees collected in the United States from infectious waste management services totalled slightly more than $500 million. As of 1990, these fees totalled in excess of $1 billion. It is anticipated that if this senseless regulation continues, infectious waste service fees will exceed $2 billion in 1996.[28] Of course, as more and more states across the country outlaw the land-filling of infectious medical waste and make the incineration of those wastes prohibitively expensive, the fees the medical community will have to pay to dispose of these wastes will increase astronomically.

CONTAMINATED SHARPS

A portal of entry for pathogens into a person may be created when a contaminated sharp is mishandled in such a way as to cause a puncture wound or cut in the skin, permitting a sufficient dose of pathogens of sufficient virulence to enter a susceptible host. This may be the case with the hepatitis B virus (HBV), based on information gathered by the CDC. CDC has estimated that 12,000 health care workers whose jobs entail exposure to blood become infected with HBV each year. Of them, 500–600 are hospitalized as a result of that infection. Of those, 700–1,200 infected become HBV carriers. Approximately 250 of the infected health-care workers die each year.[30] It is established that HBV is transmitted perinatally from infected mothers to their newborns, is transmitted through sexual contact, and is transmitted through parenteral exposure to contaminated blood or blood products.[12(p2.18)] It is unknown how many of the infected health-care workers were exposed to waste blood or to waste blood contaminated items, as opposed to being exposed to infected blood in the treatment of patients or in the process of engaging in laboratory experimentation.

There is only one documented case of infectious disease transmission possibly associated with a medical waste sharp reported in the literature: A hospital housekeeper developed staphylococcal bacteriaemia and endocarditis after a needle injury.[29]

The mode of transmission for HBV virus is similar to that of the HIV. However, the potential for HBV transmission is greater than for HIV transmission for several reasons, including the fact that HIV does not live long or well outside its host. There is also a significantly lower concentration of virus in the blood of HIV-infected persons than of HBV-infected persons. The CDC estimates, for instance, that 6%–30% of persons who receive a needlestick exposure from an HBV-positive individual will become infected, whereas the risk of infection with HIV after one needlestick exposure to blood from a patient known to be infected with HIV is approximately 0.3%.[27,29]

Since HIV is transmitted in the same way as HBV, and several hundred health care workers die from HBV infections every year, there is a theoretical possibility that a portion of those deaths is due to waste sharp injury, permitting transmission of the virus from infected medical waste to the person parenterally exposed to the same. Since HIV is transmitted in the same manner, the same theoretical possibility exists that HIV could be transmitted to a person handling HIV-infected blood or blood contaminated objects.

The ATSDR has calculated theoretical, upper-limit estimates indicating that persons handling blood-contaminated waste sharps have a potential of becoming infected with HBV and HIV. For example, the ATSDR gives upper-limit, theoretical estimates that the potential number of HBV disease cases annually for the following exposed occupational sub-groups within the almost 6,000 hospitals in this country are 12–45 for janitorial and laundry workers, 10–18 for registered nurses, 12 for hospital engineers, 1–8 for laboratory workers, 3–4 for licensed practical nurses, and less than one for physicians, dentists, and interns.[12(pp5.19–5.25)]

The upper-limit, theoretical estimates of the annual number of HIV infections in hospital employees as a result of medical waste-related injuries from sharps are less than one to three for janitorial/laundry workers, less than one for registered nurses, less than one for hospital engineers, less than one for laboratory workers, less than one for licensed practical nurses, and less than one for physicians-dentists-interns.[12(pp5.19–5.25)]

The same agency also gives a theoretical, upper-limit estimate that less than one refuse worker per year would acquire an HIV infection from medical waste-related injuries from contaminated waste sharps.[12(pp5.19–5.25)]

It must again be emphasized, however, that there is not one case of a waste-handler acquiring HIV from that infected waste documented by the CDC or documented in the literature. This is true even in view of the fact that the AIDS epidemic has raged for a decade.[31]

CONCLUSIONS

There is no greater risk of infection arising out of the management, or mismanagement, of regulated medical waste than from any other type of waste. Therefore, infectiousness should not be a criterion used to regulate medical waste, since regulation based on that provides no benefit to the public health. Non-sharp medical waste should be treated for the purpose of regulation based on infectiveness exactly as residential waste is treated.

There may be a possibility that waste-used sharps could transmit deadly disease. Therefore, until more precise data are obtained and studied, the disposal of used sharps should be regulated on a nation-wide basis to prevent the possible transmission of blood-borne disease.

Of course, there are many reasons other than infectiousness for the desirability of the regulation of medical waste. National minimal standards governing the handling, transportation, and disposal of regulated medical waste would effectively destroy the Balkanization of this issue which has occurred over the past several years. Clear definitions and criteria imposed on a national level would make the interstate shipments of medical waste much less costly, as compliance with only one set of regulations, as opposed to different and conflicting state, county, town, and city regulations, would be required.

The fact that regulated medical waste constitutes only 0.3% of the total waste generated in this country is a crucial fact that must be kept at the forefront of all considerations for dealing with the disposal of this waste. Regulated medical waste is nothing more than a tiny percentage of the overwhelming problem of waste disposal generally in this country. The science involved in regulated medical waste clearly supports the position that there is no danger of people becoming infected as a result of its handling, other than possibly from contaminated sharps. For this reason, infectiousness should not be a criterion for disposal practices. Other criteria which may be valid, though, include the aesthetic affront caused by the sight of pathological waste, the lack of landfill capacity, the physical hazard posed by sharps, and the possible emission of metals and incompletely combusted chlorinated products from the stacks of older incinerators.

REFERENCES

1. *United States of America v Paccione, Vulpis & McDonald*, 751 F Supp 368 US DC, SD NY, 1990.
2. *The Resource Conservation and Recovery Act of 1976*, 42 USCA 6903 5.
3. *EPA Guide for Infectious Waste Management.* US Environmental Protection Agency, Office of Solid Waste publication no EPA/530-SW-86-014, NTIS PB86-199130.
4. *Resource Conservation and Recovery Act: Subtitle C-Hazardous Waste Management. Section 3001 and 250.14-Infectious Waste Background Document*, Washington, DC: 1978; US Environmental Protection Agency publication no PB89-102594.
5. New York Department of Environmental Conservation: *Investigation—Sources of Beach Washups in 1988*, 1988.
6. Pub L No 100-582,102 STAT. 2950, 42 USCS 6992 (1988).
7. Federal Register: Vol 54, No 56, March 24, 1989, p 12327.
8. US Congress, Office of Technology Assessment: *Issues in Medical Waste Management—Background Paper.* Washington, DC: 1988; US Government Printing Office publication no OTA-BP-O-49.
9. *Report to the Chairman, Subcommittee on Regulation, Business Opportunities and Energy, Committee on Small Business, House of Representatives. Medical Waste Regulation. Health and Environmental Risks Need to be Fully Assessed.* US General Accounting Office: 1990; publication no GAO/RCED-90-86.
10. *A Statewide Plan for Treatment and Disposal of Regulated Medical Waste.* NYS Department of Health, approved by State Hospital Review and Planning Council. August 1989, p iii.
11. *First Interim Report to Congress: Medical Waste Management in the United States.* 1990; US Environmental Protection Agency, Office of Solid Waste, publication no EPA/530-SW-90-051A.
12. *The Public Health Implications of Medical Waste: A Report to Congress. Agency for Toxic Substances and Disease Registry.* 1990; Public Health Service, US Department of Health and Human Services publication no PB91-100271.
13. Rutala WA: Infectious Waste: A Growing Problem for Infection Control. *Asepsis* 1987; 9:2N6.
14. Centers for Disease Control: *Guidelines for Hand-washing and Hospital Environmental Control, 1985.* Section 4: Infective Waste, p 15.
15. A Report of the Nelson A. Rockefeller Institute of Government. Perspectives on Medical Waste. Sponsored by the Medical Waste Policy Committee, State University of New York. June 1989.
16. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987: 36(suppl2S):12S.
17. Rutala WA, Odette RL, Samsa GP: Management of infectious waste by US hospitals. *JAMA* 1989; 262:1635–1640.
18. Sulmer PJ: *Defining and Managing Infectious Waste: Plant, Technology and Safety Management Series. Medical Waste Disposal.* 1989; The Joint Commission on Accreditation of Healthcare Organizations publication no 4.
19. Althaus H, Sauerwald M, Schrammeck E: Waste from hospitals and sanatoria. *Zentralbl Bakteriol Mikrobiol Hyg* 1983; 178:1–29.
20. Kalnowski G, Wiegand H, Ruden H: The Microbial Contamination of Hospital Waste. *Zentralbl Bakteriol Mikrobiol Hyg* 1983; 178:364–379.
21. Donnelly, JA, Scarpino PV: Isolation, Characterization and Identification of Microorganisms from Laboratory and Full-Scale Landfills. Cincinnati, Ohio; US. 1984; Environmental Protection Agency, Municipal Environmental Research Laboratory, Office of Research and Development publication no EPA 600/2-84-119.
22. Hoeltge GA: Managing hazardous waste in the clinical laboratories. *Clin Lab Med* 1989;9:573-586,579.
23. US Department of Health and Human Services. *Public Health Service: Biosafety in Microbiological and Biomedical Laboratories*, ed 2. 1988; HHS publication no NIH 88-8395 p 2.
24. Blenkharn JI, Oakland D: Emission of viable bacteria in the exhaust flue gases from a hospital incinerator. *J Hosp Infect* 1989; 14:73–78.
25. Allen RJ, Brenniman GR, Logue RR: Emission of airborne bacteria from a hospital incinerator. *J Air Waste Manag* 1989; 39:164–168.
26. *EPA Guide for Infectious Waste Management.* Washington, DC: 1986; Office of Solid Waste and Emergency Response publication no EPA/530-SW-86-014.
27. Becker CE, Cone JE, Gerberding J: Occupational infection with human immunodeficiency virus (HIV): Risks and risk reduction. *Ann Intern Med* 1989; 10:653–656.
28. Management of Infectious Waste Called Regulation-driven Growth Industry. *J Air Waste Manag* 1991; 41:1028.
29. Guidelines for prevention of transmission of human immunodeficiency virus and hepatitis B virus to health-care and public-safety workers. *MMWR* 1989; 38(suppl6):5.
30. Jacobson T, Burke JP, Conti MT: Injuries of hospital employees from needles and sharp objects. *Infect Control* 1983; 4:100–102.
31. Recommendation for preventing transmission of human immunodeficiency virus and hepatitis B virus to patients during exposure—prone invasive procedures. *MMWR* 1991; 40:4.

Health care for seamen in the Port of New York

Part I: Recognizing the special needs of seamen

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Health care for merchant seamen was of paramount importance, as was the maritime industry, for the growth of our nation in the 18th century. The occupational hazards of transporting cargo laden with infectious diseases set the seamen apart for special consideration, including financial arrangements and provision of health services.[1–6] Colonial, state, and congressional legislation promoted local and national support for the earliest health maintenance organization (HMO), with eligibility restricted to merchant seamen and eventually extended to other beneficiary groups such as Native American Indians and military veterans.

State port entry taxes on seamen and passengers—to support quarantine services—and the head tax imposed by the Congress made seamen entering the Port of New York in the 18th century subject to double taxation for health-related purposes (Fig 1). After two centuries of health care entitlements, merchant seamen were completely disenfranchised in 1981 with respect to health care services. An historical review of the significant developments in health care services for merchant seamen in the Port of New York is traced from the erection of a facility in Staten Island, where the National Institutes of Health (NIH) began, to the disappearance of services developed specifically for merchant seamen.

CONGRESSIONAL SEAMEN'S HEAD TAX AND ROLE OF NEW YORK HOSPITAL

In 1798, the Fifth Congress of the United States passed "An Act for the Relief of Sick and Disabled Seamen,"[7] which imposed on seamen a head tax of 20¢ a month deducted from their wages to cover any future costs of their medical care.

Information concerning hospital care for seamen in the Port of New York during the early 19th century is found in several letters written by Christopher Prince.[8] He was a former sea captain, and he served as Superintendent of Seamen at the one hospital under federal contract to treat seamen—the New York Hospital. Located at what was then the periphery of the city, on the west side of Broadway between today's Duane and Worth Streets, the hospital received its first patients in 1791. Demolished in 1869 after its move uptown, the original building is preserved in a number of 19th-century prints and photographs (Fig 2).[9]

Originally, seamen were neither considered nor treated differently from the general hospital population. In 1826, however, a decision was reached to house the seamen in a separate facility, and a building that had been constructed in 1807 to serve as a lunatic asylum was converted and designated The Marine Department. This building appears on the south or left side of the main hospital building in the 19th century view of New York Hospital.[9]

New York Hospital's early Annual Reports are relatively brief, but they reveal that seamen accounted for 40%–60% of the patient population. Receipts for the year 1818 included $12,500 from the State of New York (an annual annuity), $10,762 from the US Custom House for Seamen, $3,491 from the almshouse, and $4,316 from paying patients in the lunatic asylum. Although individual admissions of seamen numbered well over the 100 limit established by the Secretary of the Treasury for the port of New York, total admissions did not exceed 100 for a duration of four months, or approximately 12,000 days of care, with no one individual to remain in the hospital for more than the four-month period.

MOUNTING DISSATISFACTION

In his 1821 letter to the Secretary of the Treasury,[8] Prince observed that very few of the seamen admitted to the New York Hospital were diagnosed as incurable or were in the last stages of "consumption;" these cases were always sent to the almshouse. Prince pointed out that he was following Treasury Department policy of restricting admissions, for had he not, there would have been more than 100 incurable seamen in the hospital at that time. From another letter written the same year by Jonathan Thompson[8] to the US Collector of Customs at the Port of New York (whose duty it was to collect the monthly tax from seamen), Prince cited several factors that had contributed to seamen seeking admission to the New York Hospital in numbers far greater than could be accommodated with the funds available under the 1798 Act.

Not only had commerce in the port significantly increased since that date, but many hundreds of seamen were semi-permanently employed on shore in loading and unloading vessels, work that earlier in the century had been done only by a vessel's own crew. Seamen, particularly those recovering from afflictions of various sorts, often worked in port for as long as six months before shipping out again. Prince estimated their number to be between 1,500–2,000. Injuries were frequent, as were accidents sustained in the

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FIGURE 1. The Port of New York, c 1872, Currier and Ives. (The New York Historical Society, New York City)

process of rigging the vessels, another task undertaken by many of the shore-bound seamen. Noted by Prince, as well, was the large increase in the number of non-whites employed at sea as stewards, cooks, and seamen; they were in need of as much, or more, medical assistance. The coastal trade had grown, and with it came a related rise in the cases of "agues, fevers and rheumatism"[10] contracted in southern ports. Prince was compelled to admit that he had been "under the necessity of rejecting many for admission who have been exposed and actually have become victims of an untimely death."[8]

Supportive of these views, a petition protesting the inadequate hospital care available to merchant seamen in the nation's largest port, the Port of New York, was sent to the Congress in 1822.[8] As noted in the report of the Secretary of the Treasury, the petitioners, Isaac Waite and Thomas B. Vermilye, officers of the Nautical Society, pointed out that although an Act had been passed by Congress in 1798 establishing a hospital fund to provide for the temporary relief of sick and disabled seamen, seamen entering the Port of New York had been "cruelly disappointed" in their efforts to obtain medical assistance.

Strong objections were raised in the petition to the policies established by the hospital fund's administrator, the Secretary of the Treasury. The principal complaint addressed the arbitrary restriction of the numbers of seamen who could apply for hospital care annually, "no more than sixty":

FIGURE 2. View of the New York Hospital, c 1811. (Reprinted from, Stokes IN, *The Iconography of Manhattan Island, 1498–1909*. Vol 3, plate 88, New York, 1915)

> Given the fluctuations in commerce, 1000 to 2000 might be in need. Seamen who have paid hospital money for twenty years and more are compelled in the dark and desolating hour of sickness and misfortune to ask for relief at the doors of the hospital. . .and to ask in vain. . . . Hundreds in a course of a year may be rejected.[8]

Protested as well were the Treasury Department's four-month limit on hospital care and the exclusion of those who were "suffering under venereal disease, afflicted with mania" or considered incurable.[8] The petitioners asked:

> Is this providing for sick and disabled seamen? . . . are any reasons furnished why American seamen should be shut out from relief, from comfort, from humanity . . . an asylum where the last pulse of life shall beat? . . . such principles of public policy would disgrace the page of this our nation's history.[8]

Although the petitioners committed certain inaccuracies in their zeal to emphasize the seamen's plight, hospital admissions in the Port of New York were limited to 100 rather than the stated 60, and those afflicted with venereal diseases were not specifically prohibited from admission—the general thrust of their remarks is confirmed by other contemporary sources. Secretary of the Treasury William Crawford in several policy directives and letters of the 1820s had himself noted that the funding made available under the 1798 Act was inadequate, even if the regulations restricting the number of admissions had been strictly observed. In spite of efforts on his part, additional funding had not been enacted; therefore, exclusionary policies were necessary to prevent the legislated monies from being totally expended on the care of "maniacs and incurables."[8] Economic necessity dictated both the formulation of the 1798 Act and subsequent directives: the fund was to be "applied to the temporary relief of sick and disabled seamen," so that "their usefulness might be restored." Responsibility for maniacs and incurables must rest, Crawford said, with local charitable agencies, a policy firmly stated in a directive of 1821: "Seamen when incurable must be considered paupers."[8]

Despite the relief and services for seamen provided at the New York Hospital, the health care available in the Port of New York could not be considered adequate. To address the need for additional funds, the Nautical Society proposed that the tax on seamen be raised from 20¢ to 40¢ a month. In addition, a tonnage tax, a contribution the ship owners could well afford, should be introduced. They further proposed that the general federal hospital fund be applied "with no discrimination. . .made as to disease. . . no apprehension of death should be regarded in affording care. . .that mania should not be viewed as out of the compass of God's judgments and that the term of giving four months of assistance should be discountenanced forever."[8]

Implementation of the petitioners' proposals regarding increased federal funding would not occur until well toward the end of the 19th century. The less restrictive admission policies they proposed and the provision of long-term care, however, were respected by the Seaman's Retreat, when in 1831 it first opened its doors under provisions made by New York State.

It was in 1821 also that the need for a separate hospital facility for seamen in the Port of New York was first suggested. The US Collector of Customs, in a letter to the Secretary of the Treasury,[8] described his efforts to locate a building that could serve as a federal marine hospital; and cited two possible candidates, both former factory buildings, one on an island near Hell's Gate (probably Ward's Island) and the other in Brooklyn. A separate hospital was not attained until more than 80 years later, when the US Marine Hospital Service finally acquired the Seaman's Retreat hospital in 1903.

New York Port Entry Taxes and the Quarantine Station

Public agitation for improved and more accessible health care for seamen in the Port of New York did not abate during the 1820s. In the early 1830s, however, in addition to Congressional pressure, attention turned toward another potential source of funding—the New York State imposed head tax collected since 1797 from all persons entering the harbor. These funds were applied to the administration of port quarantine regulations and the construction and operation of hospital facilities for the confinement of those suffering from infectious diseases.

Quarantine restrictions in the Port of New York had been in effect at least since 1738, when the Common Council of New York City, in response to fears of small pox and "malignant fevers" introduced from South Carolina, Barbados, and Antigua, passed an ordinance requiring all vessels to anchor off Bedloe's Island until inspected by "physicians from the city."[10] In 1758, the Colonial Legislature passed "An Act to prevent the bringing in and spreading of infectious distempers in the Colonies;" it too required that vessels carrying "small pox, yellow fever and other contagious diseases" remain in quarantine at Bedloe's Island (Bedloe's Island is currently Liberty Island, the site of the Statue of Liberty).[11,12] In 1784, after the Revolutionary War, the New York State Legislature renewed these quarantine restrictions, and a physician was appointed to inspect the vessels on a regular basis. The inspection system was further formalized in 1796 with the appointment of a Commissioner of Health, the official granting by federal authorities of Bedloe's Island for use as a quarantine facility, and the appropriation of funds by the New York State Legislature to repair existing buildings there for hospital use. Funds were also appropriated for the construction of a new lazaretto (hospital for those affected with contagious or loathsome diseases) on Nutten or Governor's Island.[11,12]

In 1797, the number of Health Commissioners was increased to three, and on March 30, the act that would eventually provide the funds for the Seaman's Retreat was passed, "An Act for the Establishment and Support of a Lazaretto." For the new building on Nutten Island, $4,500 was appropriated, and to maintain it a port entry tax in the following amounts was imposed on all seamen and passengers entering New York from foreign ports: captains, $1.00; mates, 50¢; cabin passengers, $1.00; steerage passengers, 50¢; and sailors (seamen), 25¢. These monies were to be applied to the "relief of sick seamen and foreigners who may be received into the said lazaretto." In 1798, the contribution of seamen was raised to 50¢.[2]

How far construction had advanced on the Nutten Island lazaretto is not clear by the time a decision was reached in 1799 to move the quarantine station to a location more distant from the city proper. Thirty acres of land on Staten Island were acquired by the State of New York near the Watering Place, the area to the north of today's Victory Boulevard; it extended almost to today's Hyatt Street at its western boundary and to the present St. Mark's Place paralleling the shoreline. Construction of hospital facilities for quarantine purposes began around the early site of the village of Tompkinsville.

Expectations of growing demands for quarantine resulted in an increase in the port-entry tax. Authorized in April 1800 by the New York State Legislature were the following fees: $1.50 to be collected from each cabin class passenger and the ship's captain, 75¢ from each steerage class passenger, mariner and mate. These taxes remained in effect until revised by an act of 1813; steerage passengers and mariners thereafter contributed $1.00, and a 25¢ tax was imposed on the masters, mates, and sailors of "coasting" vessels, (ie, those sailing from local ports). The tax on captains and cabin passengers remained at the levels established in 1800.[2]

Little is known about the earliest buildings constructed at the Staten Island quarantine. A description of the services provided there in 1800 indicated that the 945 persons ill with ship fever, yellow fever, and dysentery admitted between May 1 and December 1 far exceeded the capacity of the buildings ready to receive them. Tents were put up, and the well passengers were "put to work making their own shelters."[2] The first hospitals, probably semi-permanent buildings of wood, were later replaced by larger, more substantial buildings of brick and stone. It is these later buildings that appear in the 19th century views of Quarantine (Fig 3). On the left of the 1859 print, "View of the Quarantine Grounds and Buildings," is the two-story brick structure crowned by a two-stage cupola, the first of its three major hospitals. Designated by various titles at different times, it has been called "the long hospital," the Fever Hospital, the Female Hospital, and the Marine Hospital. The center building, St. Nicholas Hospital, was the largest and most imposing of the three. Intended as a general hospital, it was also used for patients with smallpox, cholera, and yellow fever. The smaller building to its right was known as the Smallpox Hospital. As the 1859 print shows, the Quarantine grounds also included a large number of other structures including staff residences and service buildings of various types.[13]

In spite of the fact that one of the hospitals at the Quarantine was called the Marine Hospital, the contribution of the medical facilities there to the improvement of the general health care available to seamen was relatively negligible according to the arguments of advocates for the seamen.

Pressure on the State Legislature

All sectors of the New York City's merchant marine community, both the seamen's societies and the shipmas-

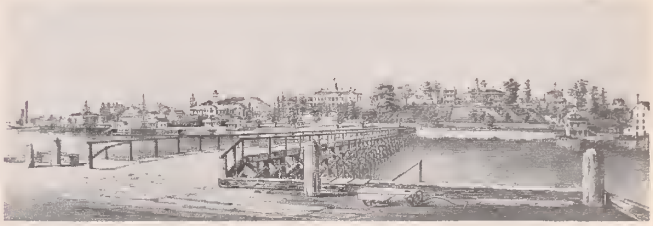

FIGURE 3. View of the quarantine buildings and grounds, Staten Island, May, 1858. (Valentine CT: *Manual of the Corporation of the City of New York for 1859*, Staten Island Institute of Arts and Sciences.)

ters, as well as local elected officials, continued to press New York State for more equitable use of the funds generated by the port-entry taxes and the expansion of services to the seamen contributors. Captain James Morgan of the Nautical Society and Captain Asa Weldon for the Seaman's Association had succeeded in being elected to the New York State Legislature with the intent of lobbying there on behalf of seamen.[14] Sent to Albany, also, was the report of a special committee appointed by the New York City Common Council in 1830 to investigate the use of the funds collected in the port.

The report first pointed out that although seamen were taxed throughout the year, care was available at the quarantine hospitals only during the months when the quarantine laws were in effect (ie, during the warm months of summer). When the seamen were most in need, "... during these eight more inclement months—vast numbers arriving during the winter, more especially, some frost-bitten and others laboring under smallpox and various pulmonary affections. . ."[14] it was then that the hospitals were closed.

The report goes on to estimate the sums collected at the port under the New York State tax from March 30, 1797, to January 1, 1831. Of the $605,933 collected, half was judged to be the contribution of approximately 434,756 seamen. However, the report concluded that:

> Estimating the number of seamen annually received as patients into the marine hospital at Staten Island at two hundred, which is a large estimate, there have been during the space of thirty-four years, 6,800 seamen only who have enjoyed the advantages of that institution; while there has been paid towards that institution and establishment, in the same space of time by the seamen contributors to that tax who have received no benefit from the fund so accumulated.[14]

Furthermore, the report estimates the sums collected in the port between 1802 and January 1, 1831, from the United States tax, to have been $431,155. At the rate of $3.00 per day paid for seamen at the New York Hospital, if the average stay is two weeks, an estimated 56,859 persons have been provided with care, leaving 328,127 seamen without benefit. Summarizing the results of its inquiry, the committee's report concluded as follows:

> The whole amount of tax therefore collected upon seamen of this port, during the last thirty-four years, by the law of the general government, which was passed July 16, 1798, and since the passage of the act of the legislature of this state, March 30, 1797, amounts to $613,676 paid by 434,756 seamen; out of which number of seamen, 63,659 *only* have received medical care and treatment; while it is a well-known fact, that numbers of *sick and disabled seamen* have every winter during that period, for want of some suitable place to accommodate them, become paupers upon the charity of the city authorities of New York.
>
> In conclusion, under the state tax, add to the above amount of $613,676, the fees and salary of the health officer for thirty-four years, $222,287 and the tax on passengers for the same period, 302,966, and we have the sum of *one million one hundred and thirty-eight thousand nine hundred and twenty nine dollars* paid to the United States and State governments, principally by the seamen and shipowners employed in the commerce of this port during the last *thirty-four* years.
>
> The committee are at a loss to conceive that any argument could be presented, in behalf of the seamen and shipping interest of this port, more powerful and conclusive that the simple arithmetical computations herewith submitted to your consideration.
>
> They beg leave therefore to offer for your adoption, the following resolution.
>
> RESOLVED: That the Common Council of this city do respectfully recommend to the Honorable Legislature of this State, that the present law, imposing a tax upon seamen arriving at this port, be so amended that the whole amount collected upon that class of persons be appropriated to their sole and exclusive benefit.[14]

Almost a decade of effort on the behalf of seamen was culminated on April 22, 1831, when the New York State Legislature approved the bill entitled, "An Act to Provide for Sick and Disabled Seamen."[2] In accordance with the resolution passed by the New York City Common Council, the act's first section declared that henceforth the funds collected from seamen under the act of 1797 were to be differentiated from those contributed by passengers:

> From and after the passage of this act the respective sums of money now levied and collected by law upon masters, mates, mariners, and seamen arriving at the city and port of New York, shall be collected and paid over to the Trustees by this act constituted, to be denominated "The Trustees of the Seaman's Fund and Retreat in the City of New York."[15]

With this act begins the history of the first hospital devoted solely to the care of seamen in the Port of New York, The Seamen's Retreat.

THE SEAMAN'S FUND AND RETREAT HOSPITAL: 1831

On May 9, 1831, just a little more than two weeks after "An Act to Provide for Sick and Disabled Seamen" had been approved, the Board of the Seaman's Fund and Retreat held its first meeting in the office of the Mayor of New York City. Admissions to the retreat peaked at 3,150 in 1852; thereafter, until it ceased to operate in 1882, restricted funding as well as other factors contributed to a decline in admissions and a cessation of its growth.

Present at the board's first meeting, as stipulated by the act, were the Mayor, the Collector of Customs, the Health Officer of New York (Commissioner of Health), and the presidents of the Seamen's Savings Bank, the Shipmasters Society and Nautical Institution, and the Marine Society, together with the five sea captains who had been appointed to complete the membership of the board. They included Captain James Morgan, who had been so effective as a spokesman for the seamen's cause in Albany; he was named Secretary of the Board, the only compensated position.[16] The board met almost weekly during the summer and early fall of 1831, enabling the hospital to open its doors by October 1.

Dr Peter Townsend, principal author of the Common Council's special committee report of 1830, was appointed as the institution's first Chief Physician (the resident physician). Townsend was one of the city's most eminent physicians; he had earlier served as a Health Commissioner, and in 1823, he published an extended account and case study of the yellow fever epidemic that had occurred in the city during the preceding summer and autumn.[17]

SITE SELECTION

The new institution's first funds, a sum of $12,197.68—that being the amount collected in the Port of New York since the passage of the legislation—was received by Captain Morgan from the Comptroller of the State of New York. By late May 1831, a site search committee of five sea captains was charged with obtaining "within the counties of

New York, Richmond or Kings . . . some suitable and healthy spot . . . for the construction of a convenient building or buildings."[18] The committee quickly located a farm in Williamsburg (Brooklyn), and the Trustees authorized its purchase for $20,000. This offer was rejected and another committee was instructed to proceed to Staten Island and purchase a farm at a cost not to exceed the funds then on hand. By the middle of June, the committee had decided to purchase 36 acres of a bay-front farm on the island's east shore belonging to Cornelius Corson for a price of $10,000.[18]

The choice of this site was soon to be criticized for its inconvenient distance from the city, but no fault was ever found with its "surpassing beauty and commanding prominence." Thus, was it praised by Chief Physician Thomas Moffatt in his 1862 history of the hospital.[18] "In nothing," he said, "did the founders of this institution evince more taste and judgement than in the selection of its location."

In choosing a waterfront location for the Seaman's Retreat, a long-established. tradition for marine hospital siting had been followed. As stated by Dr William Barton in his 1814 *Treatise Containing a Plan for the Internal Organization and Government of Marine Hospitals in the United States*, ". . . a marine hospital should be on an elevation . . . having a good command of water;"[19] there it would provide easier access for patients, often transported there directly from their vessels.

Other early actions by the board made clear its intention to develop the Seaman's Retreat as the primary hospital facility for seamen in the Port of New York, using both state and federal funds. The first resolution to be passed by the Trustees stated that until the new hospital was ready, seamen would continue to be received at the New York Hospital and the Quarantine, but the cost of their maintenance would be assumed by the Seamen's Fund.[18] The Retreat's first book of accounts reveals that this resolution was, in fact, put into effect.[20]

By June 22, 1831, the board had sent to Washington DC a memorandum asking that the 20¢ a month collected from seamen in the Port of New York under the Congressional Act of 1798 be turned over to the trustees of the Seaman's Retreat.[18]

Cited as the advantages to be offered by the new hospital were the larger number of patients that could be accommodated, the "beauty and salubrity" of its location, the view of harbor traffic, and diminished costs. In late September, Dr Townsend and Captain Reuben Brumley, authorized by the Board to contract with the federal government for the care of seamen at the cost of $2.00 per day, traveled to Washington to press their case with the Secretary of the Treasury.[18] The vision of what the Trustees hoped to accomplish in seeking to join the federal and state funds was best expressed in Secretary James Morgan's report to the State Legislature in December 1831:

> The Trustees are so well convinced of the propriety and advantages that will accrue to seamen by their having one hospital exclusively appropriated to themselves . . . so that all moneys paid by seamen may be placed in the hands of one set of men, and disbursed by them for seamen only. Should this be accomplished, the State of New York may justly claim honor of being the first in the Union that has stepped forward and provided a retreat for her sick and worn-out mariners."[2]

Just days after the hospital officially opened, the agreement with the Secretary of the Treasury had been concluded, and the Seaman's Retreat would assume responsibility for all seamen entering the port as of January 1832, when the existing contract between the federal government and New York Hospital terminated.[18]

PROTEST BY NEW YORK HOSPITAL

During this same period, the Governors of New York Hospital undertook action that was to result in the denial of federal money collected under the Act of 1798 to the trustees of the Seaman's Retreat. The Retreat hospital's first 50 years of operation were thus to be financed by state funds only, and it would not be until the early 1870s, after the reorganization of the United States Marine Hospital Service, that the hospital in Staten Island would begin to receive federal financial assistance as one of several area facilities contracted to provide care for merchant seamen.

In late 1831, the Governors of New York Hospital addressed a formal remonstrance to Washington objecting to the agreement that had been signed between the Retreat's trustees and the Secretary of the Treasury, an agreement that would have resulted in the loss of a substantial portion of New York Hospital's patient population. One of the Governors' principal arguments pertained to the inconvenient location of the new hospital on Staten Island and the difficulty of transporting the extremely sick and seriously injured, particularly during the winter when harbor icing frequently interrupted communications. They pointed out as well that the facilities for medical and surgical consultation were far greater in the city than on the distantly located island.

Although the trustees of the Retreat had announced plans to open a satellite medical facility in Manhattan, the Secretary of the Treasury noted that the objections of the New York Hospital Governors were well taken and suggested that the administrators of the two hospitals work out a compromise, and that seamen be allowed to choose which of the two institutions they preferred. The trustees apparently made several attempts to enter into such negotiations with the New York Hospital, but no agreement could be reached and the situation remained unresolved for more than a year.[18]

Another visit to Washington in May 1832 produced an agreement with the Secretary of the Treasury to appoint a committee to inspect and evaluate the Retreat's facilities; should the committee conclude that the care provided at the Retreat was equal to that available at the New York Hospital, the Secretary was prepared to order the US Collector of the port to contract with the Staten Island Hospital for the care of all seamen entering the port.[18] In a letter dated February 16, 1833, the Secretary informed the trustees that the committee had, in weighing the advantages offered by the two institutions, decided against the withdrawal of seamen from the New York Hospital.[18]

One suspects that the struggle between the Seaman's Retreat and the New York Hospital may have been somewhat acrimonious. Part of New York Hospital's desire to remain the sole federal contractor is certainly related to the fact that seamen generated a significant part of the hospital's income. New York Hospital's annual reports for the early 1830s indicated that between a third and a half of its patients were seamen, and a similar proportion of its revenue was obtained from the federal head tax.

In fact, the retreat did not at that date have the capacity to provide care to all the sick and disabled seamen who entered this port and the division of health care between the Retreat and the New York Hospital probably accrued to the seamen's benefit during the middle decades of the 19th century.

REFERENCES

1. Furman B: *A Profile of the United States Public Health Service, 1798–1948.* DHEW Publication No (NIH) 73-369. US Government Printing Office, Washington, DC, 1973.
2. Hasse AR: *Index of Economic Material in the Documents of the State New York, 1789–1904.* Kraus Reprint Corp, New York, 1965.
3. Healey JC: *Foc's'le and Glory-hole: A Study of the Merchant Seaman and his Occupation.* New York Merchant Marine Publishers Association, New York, 1936.
4. Clark EW. Haddock HS: *Medical and Hospital Care for Merchant Seamen: Historical Review of the Marine Hospitalization Program.* Labor-Management Maritime Committee, vol I, Washington DC, 1964.
5. Strauss R: *Medical Care for Seamen: The Origin of Public Medical Service in the US,* (Published for the Department of Sociology) Yale University Press; New Haven, Connecticut, 1950.
6. Williams RC: *The United States Public Health Service, 1797–1950.* Commissioned Officers Association of the United States Public Health Services, Washington DC, 1951.
7. Baxter AE: An Act for the Relief of Sick and Disabled Seamen, Fifth Congress of the United States, July 16, 1798. *General Index to the Laws of the New York State, 1777–1901, 3 vols, Lyon JB, Albany, New York, 1902.*
8. Report of the Secretary of the Treasury Showing the Application of the Fund for the Relief of Sick and Disabled Seamen and the Description of Seamen Relieved Thereby. US Treasury Department, Washington, DC, January 4, 1822.
9. Stokes IP: *The Iconography of Manhattan Island, 1498–1909,* 6 vols, Arno Press, New York, 1915.
10. Osgood HL, Keep AB, Nelson CA (eds): *Minutes of the Common Council of the City of New York, 1675–1776,* 8 vols, Dodd, Mead and Co, New York 1905.
11. Fernow B (ed): *Documents Relative to the Colonial History of the State of New York,* 15 vols, Albany, NY 1856–1861.
12. Laws of the State of New York, in *General Index of the Laws of the New York State, 1777–1901,* 3 vols, Lyon JB, Albany, NY, 1902.
13. Proceedings of the Commission for ascertaining and collecting damages caused by the destruction of the quarantine. Hasse AR. *Index of Economic Material in the Documents of the States of the United States: New York, 1789–1904.* Kraus Reprint Corp, New York, 1965.
14. The Seaman's Retreat, *The Sailor's Magazine,* vol 10, November 10, 1837.
15. New York State: An Act to Provide for Sick and Disabled Seamen, 1831. New York, Board of Trustees, Seaman's Retreat, 1845.
16. Special report of committee appointed to reprint the report of the institution published in 1846 and also to add thereto the transactions of the intitution up to the present time. *New York, Seaman's Retreat,* 1853.
17. Townsend PS: An account of the yellow fever as it prevailed in the City of New York in the summer and autumn of 1822. New York, O Halsted, 1823, New York Historical Society, Manuscript Division, Medical History Pamphlet Collection.
18. Moffatt T: A brief history of the seaman's retreat, Staten Island, from its origin in the year 1831 to the present time by its physician-in-chief. New York, 1863.
19. Barton WC: A treatise containing a plan for the internal organization and government of Marine hospitals in the United States together with a scheme for amending and systematizing the medical department of the Navy. (Printed for the author; sold by Parker E, Nicklin PH, Philadelphia, PA), 1814.
20. United States Public Health Service Hospital, Staten Island, NY Archies. Department of buildings and grounds, 1831–1851.

Epidemic Kaposi's sarcoma presenting with pleuropericarditis

SCOT C. REMICK, MD; SAMUEL A. HOISINGTON, MD; JOSEPH A. MIGLIOZZI, MD, PHD

Kaposi's sarcoma associated with human immunodeficiency virus (HIV) infection demonstrates multisystem involvement, yielding cutaneous and gastrointestinal lesions.[1] Recently, there has been increased recognition that patients with the acquired immunodeficiency syndrome (AIDS) often have significant cardiac abnormalities.[2] Cardiac manifestations of HIV infection have been noted in up to 30% of AIDS patients at autopsy[3] and in up to 64% of hospitalized AIDS patients having echocardiography.[4] Lesions of Kaposi's sarcoma have been found to occur in all layers of the heart[2,3] and should be considered when evaluating cardiac dysfunction in an HIV-seropositive patient.

We present the case of an AIDS patient with epicardial Kaposi's sarcoma. This is one of the few reported cases in which a definitive antemortem diagnosis of cardiac Kaposi's sarcoma has been made in conjunction with pericardial effusion and tamponade.[5–7]

CASE REPORT

A 42-year-old, homosexual man was admitted to the hospital for evaluation of progressive fatigue, dyspnea on exertion, and orthopnea of two months duration. There was associated chest tightness when recumbent. There was no earlier history of AIDS-defining illness.

On physical examination, his blood pressure was 120/80 mm Hg and pulse rate was 80/min and regular without paradox; respiration rate was 20/min. The patient was afebrile. Several nodular and violaceous lesions were noted over the head and neck. Breath sounds were diminished at the lung bases. The heart sounds were distant, and there was a three component pericardial friction rub. There was no jugular venous distension, Kussmaul's sign, or peripheral edema. The remainder of the examination was unremarkable.

Pertinent laboratory data on admission included the following: hemoglobin, 10.5 g/dL; hematocrit, 31%; platelet count, normal; white blood cell count, 4,800/mm^3 with 84% neutrophils and 8% lymphocytes; CD^4 and CD^8 lymphocyte percentages, 1% and 33%, respectively; absolute CD^4 lymphocyte count, 4/mm^3. The chest radiograph demonstrated moderate bilateral pleural effusions and marked cardiomegaly; the electrocardiogram showed a sinus rhythm with a rate of 90/min; and the echocardiogram showed a large pericardial effusion with associated right atrial and right ventricular diastolic compression, suggestive of tamponade.

Pericardiocentesis was performed the day after admission. Approximately 400 cc of serosanguinous fluid was removed. The results of pericardial fluid analysis were as follows: white blood cell count, 600/mm^3 with 66% polymorphonuclear leukocytes, 31% lymphocytes, and 3% mononuclear cells; glucose level, 110 mg/dL; protein count, 3.9 g/dL; lactate dehydrogenase level, 1,000 IU/L. Cytologic evaluation was negative for malignancy. The patient also underwent bilateral thoracenteses, yielding an exudate with cytologic findings consistent with a reactive inflammatory process. All cultures of pericardial and pleural fluid and subsequent cultures of blood, urine, and sputum were negative for bacterial, fungal, mycobacterial, and viral pathogens. Viral cultures of pericardial fluid were negative for cytomegalovirus, herpes virus, echovirus, Coxsackie virus, and enterovirus. Because of persistent pleuropericardial effusions, a pericardial window was placed. There was histopathologic evidence of Kaposi's sarcoma within the pericardium (Figs 1,2). The HIV serology was positive by enzyme-linked immunosorbent assay and was confirmed by Western blot analysis.

The patient was initially treated with low dose radiation, 15 Gy to the pericardium. Approximately two months later, there was significant reaccumulation of the pleural effusions, for which the patient was treated with a variety of single agent chemotherapeutic regimens including etoposide, doxorubicin, and dacarbazine. At no point was there objective evidence of response, and multiple therapeutic thoracenteses were required.

Approximately eight months after initial presentation, the patient died from progressive Kaposi's sarcoma. At autopsy, dissection of the pleural and pericardial cavities revealed massive adhesions over the pleural, pericardial, and diaphragmatic surfaces, with the heart totally encased in these adhesions. Histologic examination of the pleural, pericardial, and diaphragmatic surfaces and adhesions revealed diffuse and extensive involvement with Kaposi's sarcoma. There was also evidence of Kaposi's sarcoma throughout the lymphoreticular system.

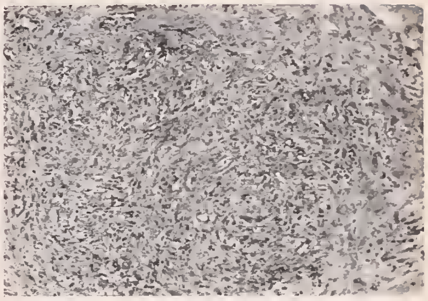

FIGURE 1. Kaposi's sarcoma. This well-developed lesion in the pericardium exhibits numerous blood vessels and aggregates of spindle cells arranged in interweaving fascicles. (hematoxylin and eosin stain; original magnification × 100).

DISCUSSION

Pericardial effusion in AIDS is not uncommon. The etiologic agents are varied and have included infection by *Mycobacterium tuberculosis, Cryptococcus neoformans, Toxoplasma gondii,* herpes virus, and cytomegalovirus.[5,8–10] Neoplastic involvement of the heart leading to pericardial effusions has been seen with malignant lymphoma[11] and recently with Kaposi's sarcoma.[5–7] Those cases not attributable to a particular

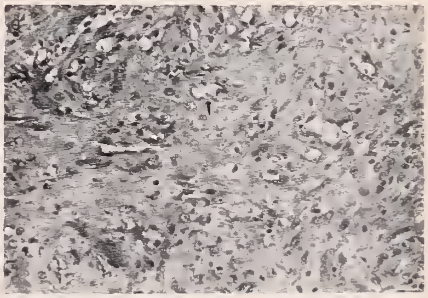

FIGURE 2. Thin-walled, dilated, irregularly shaped blood vessels (arrow) are present throughout. Extravasated red blood cells and accompanying inflammatory cell infiltrate made up of plasma cells and lymphocytes are also seen. (hematoxylin and eosin stain; original magnification × 200).

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agent have been termed nonspecific myocarditis and have been in the majority.[2–4] This case is the fifth reported in which AIDS-related pericardial Kaposi's sarcoma has led to a massive effusion with tamponade[5–7] and the first in which this manner of cardiac dysfunction was the presenting illness. In four of the five cases, chest roentgenogram demonstrated an enlarged cardiac silhouette and effusion and tamponade were confirmed with echocardiography.[5,7] In the remaining case, pericardial effusion was demonstrated by computed tomography, and the patient expired shortly thereafter.[6] In all cases, pericardiocentesis yielded a serosanguinous fluid, negative for malignant cells. In the patient described here, the diagnosis of Kaposi's sarcoma was made after the placement of a therapeutic pericardial window, whereas the diagnosis was made at autopsy in the other patients.

Considering the multiple etiologic agents involved in pericardial effusion, it is not an easy task to differentiate possible causes clinically. The only consistent finding in all cases of cardiac Kaposi's sarcoma associated with pericardial effusion was the presence of a hemorrhagic rather than straw-colored effusion,[5–7] although such effusions have also been seen in association with *Toxoplasma gondii* infection and cardiac lymphoma.[10,11] No method of treatment was successful in those effusions found to be related to cardiac Kaposi's sarcoma.

With the increasing awareness that HIV infection may lead to life-threatening cardiac abnormalities, it is apparent that more attention should be paid to the appraisal of cardiac function in affected individuals. We believe that symptomatic cardiac dysfunction should lead to echocardiographic evaluation, as this modality has demonstrated a high sensitivity for the early cardiac manifestations of HIV infection.[4] Kaposi's sarcoma should not be overlooked as a causative agent when evaluating pericardial effusion in a patient with AIDS as management of the effusion and prevention of tamponade will depend on the causative factors.

REFERENCES

1. Krigel RL, Friedman-Kien AE: Kaposi's sarcoma in AIDS: Diagnosis and treatment, in DeVita VT, Hellman S, Rosenberg SA (eds): *AIDS* Philadelphia, JB Lippincott Co, 1985, pp 245–261.
2. Cammarosano C, Lewis W: Cardiac lesions in acquired immune deficiency syndrome (AIDS). *J Am Coll Cardiol* 1985; 5:703–706.
3. Welch K, Finkbeiner W, Alpers CE, et al: Autopsy findings in the acquired immune deficiency syndrome. *JAMA* 1984; 252:1152–1159.
4. Himelman RB, Chung WS, Chernoff DN, et al: Cardiac manifestations of human immunodeficiency virus infection: A two-dimensional echocardiographic study. *J Am Coll Cardiol* 1989; 13:1030–1036.
5. Stotka JL, Good CB, Downer WR, et al: Pericardial effusion and tamponade due to Kaposi's sarcoma in acquired immunodeficiency syndrome. *Chest* 1989; 95:1359–1361.
6. Steigman CK, Anderson DW, Macher AM, et al: Fatal cardiac tamponade in acquired immunodeficiency syndrome with epicardial Kaposi's sarcoma. *Am Heart J* 1988; 116:1105–1107.
7. Langer E, Mischke U, Stommer P, et al: Kaposi's sarcoma with pericardial tamponade in AIDS. *Dtsch Med Wochenschr* 1988; 113:1187–1190.
8. D'Cruz IA, Sengupta EE, Abrahams C, et al: Cardiac involvement, including tuberculosis pericardial effusion, complicating acquired immune deficiency syndrome. *Am Heart J* 1986; 112:1100–1102.
9. Cegielski JP, Ramiya K, Lallinger GJ, et al: Pericardial disease and human immunodeficiency virus in Dar es Salaam, Tanzania. *Lancet* 1990; 335:209–212.
10. Adair OV, Randive N, Krasnow N: Isolated toxoplasma myocarditis in acquired immune deficiency syndrome. *Am Heart J* 1989; 118:856–857.
11. Gill PS, Chandraratna PA, Meyer RP, et al: Malignant lymphoma: cardiac involvement at initial presentation. *J Clin Oncol* 1987; 5:216–224.

Chronic recurrent multifocal osteomyelitis in adult women

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Chronic recurrent multifocal osteomyelitis (CRMO) is an uncommon entity, characterized clinically and radiologically by multiple osteomyelitic-type bone lesions, with sclerosis and bone pain, which may be present over a course of several months to years. The etiology is unknown, and the age of onset is typically in children and young adults. We present three cases of CRMO that occurred in adult women (30–54 years of age) and review the literature.

CASE REPORTS

Case 1. A 30-year-old woman presented with recurrent fever to 103°F and left knee pain in 1976. She had a 12-year history of ulcerative colitis with a colectomy and ileostomy.

On admission, her knee was warm and swollen and contained a bland effusion. Laboratory studies revealed a white blood cell count of 8,800/mm^3; hematocrit of 34.4%; and hemoglobin of 10.7 g/dL. Radiologic examination of the left lower leg revealed multiple lucencies of the proximal three quarters of the left tibial shaft as well as the head of the fibula and patella. On radiologic examination of the right distal femur, similar changes were noted (Fig 1).

A bone biopsy of the left medial proximal tibia was performed, and yellow purulent material oozed from the wound as soon as the cortex was removed. On histologic evaluation, granulation tissue infiltrated with lymphoid cells, polymorphonuclear leukocytes, and a few plasma cells were noted. The bony trabeculae were partly necrotic. Cultures for bacteria mycobacteria and fungi were negative. She was treated with acetaminophen, as needed, and over two months the pain abated.

FIGURE 1. Radiologic examination of distal femurs and proximal tibias revealing multiple lucencies (case 1).

The patient was next seen in 1977, at which time she complained of pain in the left knee and recurrent fever. A skeletal survey noted lucent changes similar to those seen in 1976. A bone biopsy and culture of the left midtibia were negative for organisms. The patient was treated empirically with clindamycin and amikacin for two weeks with no real change in her bone pain.

Her condition improved over time, and she was asymptomatic when she was again seen in 1983. Repeat technetium bone scans done in 1983 and again in 1986 revealed increased nucleide activity in both tibias and the right femur. In 1989, another scan was remarkable for the aforementioned areas as well as the left posterior elbow and tarsal joints of both feet. Radiographs of the left knee revealed only changes consistent with osteoarthritis. Left elbow radiographs showed marked demineralization. The patient, however, when last seen in 1990, was asymptomatic.

Case 2. A 54-year-old woman presented with anemia and low back pain in 1986. She had undergone a right nephrectomy two months before because of renal calculi and

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infection. During surgery, she received intravenous antibiotics. One month before to the current admission, she noted constant back pain which was exacerbated by sitting and movement. A low-grade fever and night sweats were present during this time. She had experienced a 30-lb weight loss since the renal surgery.

On physical examination she was afebrile. She had a well-healed right nephrectomy scar. The examination was otherwise normal, except for tenderness over the sacral and coccygeal areas. On neurologic evaluation, there were no sensory or motor deficits. A complete blood cell count remarkable for a white cell count of 12,300/mm^3 with 70% polymorphonuclear leukocytes, a hemoglobin of 5.3 g/dL, and a mean corpuscular volume of 86 red cells/fL. There was no evidence of bleeding or hemolysis. The erythrocyte sedimentation rate was 140 mm/hr.

Radiographs of the spine revealed disc narrowing between L5-S1, and a technetium 99 bone scan showed areas of increased uptake in the left ninth rib, midshaft of the right femur, and distal end of the sternum. A corresponding radiograph of the right femur, however, was normal, whereas the rib series demonstrated "unhealed fractures" of the right third, fourth, and fifth ribs along the mid-axillary line. An extensive malignancy workup, including bone marrow aspiration and biopsy, was negative. Computed tomography (CT) of the spine revealed multiple "lytic" areas in the vertebral bodies of L1 and L5, with reactive sclerosis and destruction of the cortex of the body of L5.

An open biopsy of the ribs revealed diffusely fibrotic bone with abnormal cartilage arrangement and a mild inflammatory response. Cultures were negative for fungi, bacteria, and mycobacteria. A bone biopsy of L5 revealed only inflammatory cells. Here, too, special stains and cultures were negative.

The patient's pain persisted for months; she was treated with analgesics and nonsteroidal anti-inflammatory drugs. When last seen, five months after her initial presentation, the patient's pain had disappeared, and she had gained 20 lbs. Repeat laboratory values, at one month, revealed a erythrocyte sedimentation rate of 60 mm/hr and improvement in the hemoglobin level. She moved to Florida, and details of subsequent medical care are unknown.

Case 3. A 53-year-old woman presented in 1990 with pain and swelling in the sternoclavicular (SC) joint that had been present for one year. The pain began insidiously in the left upper sternal area and became more severe with time. She recalled having had fever and generalized fatigue at the onset of these symptoms. She also noted pain over the anterior pubis with sensitivity to touch. This pain spontaneously decreased in intensity, but the pain and swelling of the left SC joint worsened. She was treated with prednisone 5–10 mg daily, for several weeks with clinical improvement of the left SC joint.

On physical examination, the left sternoclavicular area was swollen and erythematous with tenderness and warmth to palpitation. The bone and joint examination was otherwise unremarkable.

Radiologic examination of the left sternoclavicular area was remarkable for irregular articular margins with widening of the joint space and sclerosis (Fig 2). Periosteal reaction was noted at the medial end of the clavicle. Radiographs of the pelvis revealed widening of the symphysis pubis with an irregular joint space and sclerosis (Fig 3). A CT scan of the left SC joint (Fig 2) revealed a soft tissue mass and focal osseous destruction.

Increased radionuclide uptake (technetium 99) was demonstrated in the vicinity of the left SC joint, the anterior aspect of the left rib, and the symphysis pubis. Laboratory evaluation included a white blood cell count of 13,200/mm,^{3} hematocrit of 40.6%, Westergren sedimentation rate of 34 mm/hr, and a negative test for rheumatoid factor.

A biopsy of the left medial clavicle demonstrated marrow space with polymorphonuclear leukocytes, lymphocytes, and fibrous tissue. Focal extension of inflammatory cells into adjacent soft tissue and occasional areas of new bone formation were also noted. A gram's stain and cultures for bacteria, mycobacteria, and fungi were negative. HA typing revealed the presence of A1, B7 and 8, Cw7 and DR3. The patient was treated with analgesics, and a diagnosis of suspected chronic recurrent multifocal osteomyelitis was made. Seven months later, the patient returned with pain in the pubic area, but no pain in the sternoclavicular joint. We are continuing to follow-up this patient.

Discussion

CRMO is a rare clinical entity, first described by Giedion et al[1] and characterized by a self-limited inflammation of bone. Recurrent clinical episodes interspersed with periods of symptomatic improvement occur over months to years. Multiple osseous sites may be affected at different times, with recurrence at the same site of involvement being a typical feature. The age of onset is reported as 4–27 years, although one case has been documented in a 55-year-old woman.[2] The disease is more common in women than men (ratio 2:1) and no racial associations have been noted.[3]

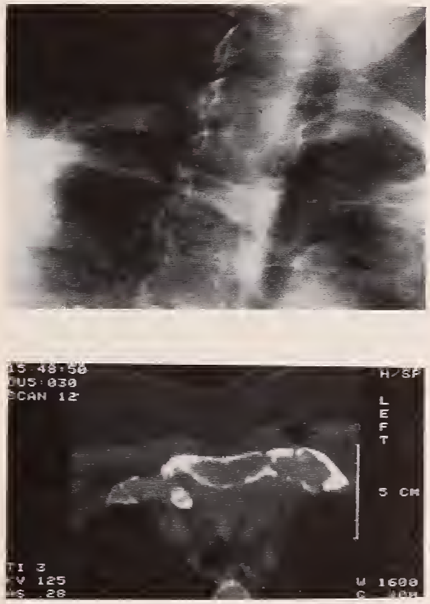

FIGURE 2. Top, widening of the left sternoclavicular (SC) joint with an irregular articular margin of the medial left clavicle and sclerosis (arrow). Bottom, CT scan of left SC joint. Focal bone destruction involving superior medial aspect of the sternum and medial left clavicle with associated ill-defined soft-tissue mass (arrow) (case 3).

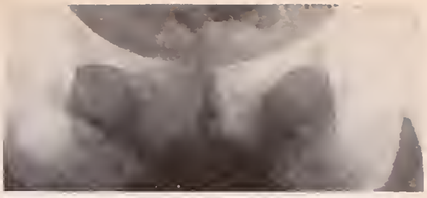

FIGURE 3. Widening of the pubic symphysis with irregular margins and sclerosis (case 3).

The onset of CRMO is usually insidious. Half of the patients with this entity are febrile. Local pain and swelling in affected bones or joints may persist for months or years. Joint effusions, however, are unusual. Elevation of the erythrocyte sedimentation rate is typical,[4] and white cell counts tend to be normal. Blood, joint, and tissue cultures are routinely sterile.

The multifocal radiographic abnormalities are often symmetrical and usually juxtaepiphyseal in the metaphysis of the long bones[3] and sternal ends of the clavicles. The proximal and distal ends of the tibia are the most common sites of involvement,[4] but lesions in the femur, pelvis, spine, and mandible have been described. The usual number of lesions is five to six, although as many as 10–12 lesions have been reported in a single patient. [5]

Patient complaints are usually focused on a single location, but the radiologic picture is most frequently multifocal.[6] Rarely, multifocal lesions are only detected over time. Roentgenographic findings include bone lesions, soft tissue swelling, and rarely, joint effusions. Lesions may initially be interpreted as malignant.[7]

Asymptomatic bone lesions are detected by imaging techniques, such as bone scan, as noted in Cases 1 and 3, followed by plain radiographs. Chronic stages demonstrate small radiolucencies adjacent to metaphyseal growth plates and surrounded by areas of sclerosis.[8] In flat bones and clavicles, the sclerotic reaction may be more diffuse involving the whole diameter of the affected bone.[7]

Histologically, CRMO begins as an inflammatory process with osteoblastic bone resorption. Neutrophils dominate in the acute phase. Lymphocytes, granulation tissue (noncaseating granuloma), and new bone formation are associated with chronicity.[2] Later lesions show fibrosis surrounding foci of inflammation. Bacteriologic and fungal cultures are uniformly negative. Differentiation from subacute bacterial osteomyelitis is difficult and requires bone biopsy and cultures.[9]

Palmoplantar pustulosis has been previously described in association with this entity, as has psoriasis vulgaris.[10] About 20% of patients develop a pustular rash on their palms and soles. One of the patients in our study had pre-existing ulcerative colitis, and this association has been reported by Kahn.[11] King et al[12] postulated that CRMO might be related to the spondyloarthropathies because of the association with psoriasis. In adults, sternocostoclavicular hyperostosis as well as sacroilitis, spondylitis, and peripheral arthritis have been reported in association with palmoplantar pustulosis.[10] However, none of the patients presented here had any of these findings. Giedion et al [1] proposed that CRMO may be an autoimmune process in which "cryptantigens" are released. Investigation into possible immunodeficiency in four CRMO patients was unrewarding.[11] Lastly, a fastidious organism of low virulence has been postulated as a causative factor, but this organism has yet to be isolated. Routine cultures for bacteria, fungi, and mycobacteria are consistently negative[13].

The prevalence of HLA-B27 in CRMO has not yet been ascertained, but it was absent in five previously reported cases[1] in addition to Case 3. HLA-Cw4 antigen has been found with increased frequency in pustulotic arthro-osteitis and acne arthro-osteitis[14] but was absent in the patient tested here. The genetics of this disorder may be an area for investigation, especially when CRMO is seen in association with seronegative arthropathies and psoriasis. Other predisposing factors are not known.

In summary, CRMO is a benign, self-limited disease with osteomyelitis-like lesions essentially resolving over a period of 1–15 years.[15] This entity should be considered in all patients, regardless of age, who have multiple lytic and sclerotic lesions with or without systemic manifestations. CRMO remains a diagnosis of exclusion as vascular, inflammatory, and neoplastic bone disorders must also be considered. In one patient described here, weight loss was marked and extensive; prolonged evaluation was performed to rule out a malignant etiology. Therefore, a bone biopsy is still required, as there is no other current way to confirm the benign nature of the lesions.

The correct diagnosis of CRMO is important to avoid unnecessary diagnostic procedures or therapy. Antibiotic therapy seems to have no effect on the course of the disease. Corticosteroids and anti-inflammatory agents have been tried during exacerbations, with and without improvement.[15] Although treatment with steroids may yield initial improvement, recurrence may occur once the dose is tapered.

CRMO has a favorable diagnosis overall, although lesions may persist for years and children with CRMO have had complications.[15] The recommended treatment for CRMO is largely supportive with the use of analgesics, physical modalities appropriate for each patient, and the avoidance of invasive diagnostic procedures after the diagnosis is established.

REFERENCES

1. Giedion A, Holthusen W, Masel LF, Vischer D: Subacute and chronic "symmetrical" osteomyelitis. *Ann Radiol* 1972; 15:329–342.
2. Bjorksten B, Boquist L: Histopathological aspects of chronic recurrent multifocal osteomyelitis. *J Bone Joint Surg* 1980; 62B:376–380.
3. Meller Y, Yagupsky P, Elitsur Y, et al: Chronic recurrent multifocal osteomyelitis: Report of two cases in Bedouin infants. *Am J Dis Child* 1984; 138:349–351.
4. Van Howe RS, Starshak RJ, Chusid MJ: Chronic recurrent multifocal osteomyelitis. *Clin Pediatr* 1989; 28:54–59.
5. Solheim LF, Paus B, Liverud K, Stoen E: Chronic recurrent multifocal osteomyelitis: A new clinical radiologic syndrome. *A Orthop Scand* 1980; 50:37–41.
6. Murray SD, Kehl DK: Chronic recurrent multifocal osteomyelitis: A case report. *J Bone Joint Surg* 1984; 66A:1110–1112.
7. Probst FP, Bjorksten B, Gustavson KH: Radiological aspect of chronic recurrent multifocal osteomyelitis. *Ann Radiol* 1978; 21:115–125.
8. Wiener MD, Newbold RG, Merten DF: Chronic recurrent multifocal osteomyelitis (case report). *AJR* 1986; 146:87–88.
9. Yu L, Kasser JR, O'Rourke E, et al: Chronic recurrent multifocal osteomyelitis. Association with vertebra plana. *J Bone Joint Surg* 1989; 71:105–112.
10. Laxer RM, Shore AD, Manson D, et al: Chronic recurrent multifocal osteomyelitis and psoriasis. *Semin Arthritis Rheum* 1988; 17:260–270.
11. Kahn MD: Letter to the Editor. *J Bone Joint Surg* 1990; 72:305–306.
12. King SM, Laxer RM, Manson D, et al: Chronic recurrent osteomyelitis: A non-infectious inflammatory process. *Pediatr Infect Dis* 1987; 6:907–914.
13. Pelkonen P, Ryoppy S, Jaascelainen J, et al: Chronic osteomyelitis-like disease with negative bacterial cultures. *Am J Dis Child* 1988; 142:1167–1173.
14. Huaux JP, Esselinckx W, Rombouts J, et al: Pustulotic arthro-osteitis and chronic recurrent multifocal osteomyelitis in children. *J Rheumatol* 1988; 15:95–100.
15. Case records of the Massachusetts General Hospital (Case 28, 1986). *N Engl J Med* 1986; 315:178–185.

Bilateral adrenal hemorrhage in polycythemia vera

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A frequent cause of adrenal hemorrhage is anticoagulant therapy.[1,2] Other precipitating factors include shock (Waterhouse-Friderichsen syndrome),[3] surgery,[4] sepsis,[3] thrombocytopenia,[5] adrenal metastases,[6] trauma,[7] and coagulopathy.[8] Early recognition of this unusual condition is necessary for successful treatment. Before the use of computed tomography (CT), the diagnosis of this condition was difficult. With CT,[9] however, it is possible to visualize and diagnose the adrenal hemorrhage, enabling early administration of steroid therapy. This report presents the first case to my knowledge of spontaneous bilateral adrenal hemorrhage with recovery in a patient with polycythemia vera who did not receive anticoagulants.

CASE REPORT

An 85-year-old man with a history of polycythemia vera and stable exertional angina pectoris was admitted to the Tisch Hospital at New York University because of precipitous syncopal attack that lasted several minutes. He complained of severe pain in the chest and back, with marked weakness, dizziness, and faintness. The patient stated that his medication consisted of diltiazem hydrochloride, 60 mg once a day, atenolol, 25 mg twice a day, and enalapril maleate, 5 mg once a day.

On physical examination, he had an oral temperature of 96°F, which subsequently rose to 102°F. The pupils were equal and regular and reacted to light with normal accommodation. The neck was supple, and there was neither thyromegaly or lymphadenopathy. The chest was symmetrical and the expansion was equal. The heart was enlarged to the left on percussion, and a grade II-III diastolic murmur was heard at the right of the sternum, indicating aortic insufficiency. The blood pressure initially was 190/80 mm Hg, and after resting was 140/90 mm Hg. The lungs were clear to percussion and auscultation. The abdomen was slightly distended, and there was mild tenderness to palpation in the right upper quadrant. The liver and spleen were not palpable. The extremities were not edematous, and the deep reflexes could not be elicited in the lower extremities. Motor power and sensation were intact. The rectal examination revealed no masses and the guaiac test was negative. The prostate was slightly enlarged, but no nodules could be felt.

The urine tested negative for protein and blood. The sediment contained 1-2 white blood cells and no bacteria per high-power field. The specific gravity was 1.015.

The hematocrit was 45%; the red blood cell count was 5.69 10^{12}/L; the white cell count was in the range of 14.8–23.3 10^{9}/L, with 64% neutrophils, 25% lymphocytes, 8% monocytes, and 2% eosinophils; the platelet count was 538,000/μL, and they were clumped. Before the attack, the patient's hematocrit reached 60%; the red cell mass reached 51.3 cc/kg or 1,909 cc/m^2; whole blood volume

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reached 95.4 cc/kg or 3,500 cc/m^2; and erythropoietin level was 2 mIU/mL.

The serum sodium was 136 mg/dL; potassium was 5.5 mg/dL; chloride was 100 mg/dL; carbon dioxide was 26 mEq/L. The blood urea nitrogen level was 39 mg/dL; creatinine was 1.5 mg/dL; glucose was 100 mg/dL; total protein was 6.5 g with 3.5 mg albumin. The blood calcium count was 8.5 mg/dL; total bilirubin was 0.5 mg/dL; and direct bilirubin was 0.1 mg/dL. The alkaline phosphatase level was 78; creatinine phosphokinase was 25; serum glutamic oxaloacetic transaminase was 26; and amylase was 164 U. The lactate dehydrogenase count was elevated at 331 U/L, but the isoenzymes were not tested. Several blood cultures were negative.

A chest film was within normal limits. An ultrasound examination of the gallbladder revealed slight "sludging," a normal liver and pancreas, and bilateral renal cysts but no hydronephrosis.

Because of the suspicion that there had been intra-abdominal bleeding, shortly after admission a CT scan of the abdomen with oral contrast and with and without non-ionic contrast was performed. It demonstrated bilateral high-attenuating homogenous adrenal masses which did not enhance with contrast and were interpreted as being diagnostic of bilateral adrenal hemorrhages (Fig 1).

The plasma cortisol level was found to be 7.0 μg/dL, within the "normal" range, but inappropriately low for a patient with acute illness. To treat the adrenal insufficiency that resulted from the adrenal hemorrhage, the patient was first given 100 mg of intravenous hydrocortisone sodium succinate daily for two days. Then he was switched to 25 mg of oral hydrocortisone acetate twice a day. After one day, this dose was reduced to 25 mg in the morning and 12.5 mg in the evening. The hydrocortisone acetate dose was gradually tapered, and the patient currently takes a maintenance dose of 20 mg once a day because of mild signs of adrenal insufficiency. A CT scan performed 18 months after the adrenal hemorrhage showed complete resolution (Fig 2).

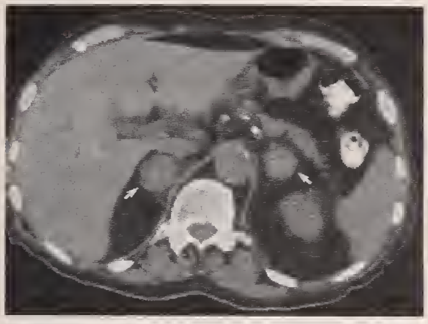

FIGURE 1. CT scan shows masses in both adrenal glands (arrows). The masses are of slightly higher attenuation than the adjacent tissues, and they did not enhance with intravenous contrast media, indicating that they represented bilateral adrenal hematomas.

DISCUSSION

Bilateral adrenal hemorrhage in the absence of anticoagulant therapy is an uncommon phenomenon. Reports of this entity are more common in patients who received heparin. An early report of heparin-related adrenal hemorrhage was documented by Thorn et al in 1956.[10] Bilateral adrenal hemorrhage in a patient with polycythemia vera, without the administration of anticoagulants, has never been reported to my knowledge. Marcus et al,[2] however, did report a patient with polycythemia vera who received heparin and then experienced bilateral adrenal hemorrhage. Although it is well known that hemorrhage and thrombosis are common features in polycythemia vera,[11] no connection has been shown until now between polycythemia vera and hemorrhage within the adrenal glands. In polycythemia vera, an increase in the red cell mass and expanded blood volume often result in hematocrit and hemoglobin elevation. Bleeding is often seen from the gastrointestinal tract, cerebrum, and nose.[11]

The three major risk factors for bilateral adrenal hemorrhage are thromboembolic disease, coagulopathy, and the postoperative state.[12] Additional risks that may lead to adrenal hemorrhage are underlying chronic diseases, such as arteriosclerotic heart disease and emphysema, or an underlying condition such as complicated pregnancy or postpartum state.[13]

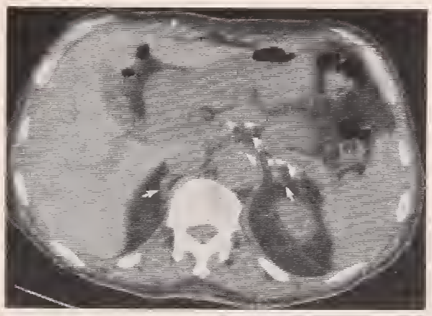

FIGURE 2. Follow-up CT scan reveals that the hematomas have resolved and the adrenal glands appear normal.

The patient presented here demonstrated none of the classic findings of polycythemia vera, such as splenomegaly, hepatomegaly, elevated uric acid, or increased hemoglobin or hematocrit. However, he did have angina and an increased platelet count with clumping. Coagulopathy is what appears to link polycythemia vera with bilateral adrenal hemorrhage.

When clinical symptoms suggest adrenal hemorrhage, a CT scan of the abdomen can confirm the diagnosis. The finding of a unilateral adrenal mass or bilateral adrenal masses of high attenuation (~ 50–60 HU) that do not enhance with intravenous contrast is diagnostic of adrenal hematoma and can be used to detect and confirm this condition. It is essential to perform CT early on, and this early scan is the gold standard that helps prevent a fatal outcome.

Acknowledgments. The author thanks Arthur C. Fox, MD, Morton A. Bosniak, MD, and Manfred Blum, MD, for their generous assistance.

REFERENCES

1. Ognibene AJ, McBride H: Adrenal hemorrhage: A complication of anticoagulant therapy—a case history. *Angiology* 1987; 38:479–483.
2. Marcus AO, Duick DS, Little D, et al: Bilateral adrenal hemorrhage during anticoagulant therapy. *Ariz Med* 1982; 39:575–577.
3. Levin J, Cluff LE: Endotoxemia and adrenal hemorrhage: A mechanism for Waterhouse-Friderichsen syndrome. *J Exp Med* 1965; 121:247–260.
4. Fitzpatrick PM, Swensen SJ: Report of an unusual case of postoperative adrenal hemorrhage in a young woman. *Am J Med* 1989; 86:487–489.
5. Findling JW, Korducki JM, Lahiri PK, et al: Bilateral adrenal hemorrhage associated with heparin-induced thrombocytopenia. *Wis Med J* 1987; 86: 27–29.
6. Shah HR, Love L, Williamson MR, et al: Hemorrhagic adrenal metastases: CT findings. *J Comput Assist Tomogr* 1989; 13:77–81.
7. Murphy BJ, Casillas J, Yrizarry JM: Traumatic adrenal hemorrhage: Radiologic findings. *Radiology* 1988; 169:701–703.
8. Schafer AI: The hypercoagulable states. *Ann Intern Med* 1985; 102:814–828.
9. Liu L, Haskin ME, Rose LI, et al: Diagnosis of bilateral adrenocortical hemorrhage by computed tomography. *Ann Intern Med* 1982; 97:720–721.
10. Thorn GW, Goldfien A, Nelson DH: The treatment of adrenal dysfunction. *Med Clin North Am* 1956; 40:1261–1279.
11. Hoffman R, Wasserman LR: Natural history and management of polycythemia vera. *Adv Intern Med* 1979; 24:255–285.
12. Rao RH, Vagnucci AH, Amico JA: Bilateral massive adrenal hemorrhage: Early recognition and treatment. *Ann Intern Med* 1989; 110:227–235.
13. Xarli VP, Steele AA, Davis PJ, et al: Adrenal hemorrhage in the adult. *Medicine* (Baltimore) 1978; 57:211–221.

Large endometrioma with intrauterine pregnancy

Jerry G. Ninia, MD

The incidence of true ovarian tumors in pregnancy is reported to be less than 0.1%.[1,2] The most common site of endometriosis is the ovary and two out of three women with endometriosis have ovarian involvement. However, only 5% of these women have ovarian enlargement detectable by pelvic examination.[3] Bilateral ovarian endometriomas are commonly multiloculated, hemorrhagic cysts of usually 5–10 cm in diameter. Additionally, it is not uncommon to find diffuse pelvic disease involving adhesion formation. The most characteristic features of endometriosis are cyclic premenstrual pelvic pain, dyspareunia, and infertility. Since the first description of two case of endometrioma in pregnancy by Scott in 1944,[4] other cases have been reported.[5] This case report describes, to our knowledge, the largest endometrioma in pregnancy reported to date.

Case Report

A 32-year-old woman, gravida 1, para 0, was first seen in the obstetrical triage room at 16 weeks gestational age (by dates) complaining of dull, non-radiating lower abdominal pain of three days duration. She had had no prenatal care. Except for a history of some increased urinary frequency, a review of systems was otherwise unremarkable for obstetric, gastrointestinal, or genitourinary complaints. Her medical and surgical histories were unremarkable. The patient reported no allergies and was currently taking no medications. Denying dysmenorrhea and dyspareunia, she reportedly was sexually active for over ten years without contraception and without conception.

On physical examination, she was afebrile and had a softly distended abdomen, and bowel sounds were present. There was slight abdominal tenderness to deep palpation. Bimanual pelvic examination revealed an anteverted 14–16 week size uterus with a posteriorly situated cystic, mobile, non-tender adnexal mass extending approximately five finger-breadths above the umbilicus. Laboratory values were entirely within normal limits, and a urine pregnancy test was positive. An ultrasound examination confirmed a 15.7 week intrauterine pregnancy, consistent with the timing of her last menstrual period. Additionally, a 20-cm complex cystic left adnexal mass was identified. The patient was admitted to the hospital and scheduled for exploratory laparotomy.

A left paramedian incision was made extending above the umbilicus. A 22 × 18 × 12 cm left ovarian, smooth cystic mass was found posterior to the uterus. Because of the cyst location, the uterus was exteriorized and was noted to be free of adhesions. The cyst was removed intact along with the remaining portion of ovarian tissue. The left fallopian tube was left in situ. Surprisingly, the remainder of the pelvis and bowel, including the contralateral ovary, was found to be free of disease. The ovarian and cyst capsules were smooth without excresences. The patient recovered well from the laparotomy, and she was discharged home after six days. Pathologic examination revealed that the mass was an endometriotic cyst weighing 2,200 g. Histopathologic analysis showed the characteristic features of endometrial glands and stroma with hemorrhage into adjacent tissue (Figs 1,2).

The patient continued to receive prenatal care. Serial abdominal examination and sonograms showed fetal growth appropriate for gestational age. She developed gestational diabetes mellitus in the third trimester, which was controlled by diet. At term, the patient went into spontaneous labor and vaginally delivered a female infant weighing 2,637 g with Apgar scores of 9/9.

Discussion

Although pelvic tumors in pregnancy are rare, an endometrioma in pregnancy is particularly unusual because of its association with infertility. Most ovarian tumors in pregnancy are either dermoid cysts or mucinous cystadenomas.[5] Only about 1% of all cases of endometriomas undergo malignant transformation.[6] A history of infertility should raise the possibility of an endometrioma in the differential diagnosis, although such an occurrence would be rare.

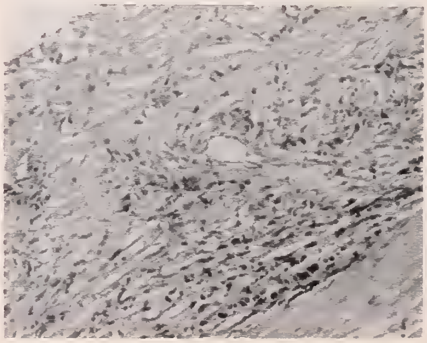

FIGURE 1. Hyperprogestational, pregnancy-induced changes in the endometrioma with a small atrophic endometrial gland lined by flattened epithelial cells. The cyst is lined by endometrial epithelium and a thick layer of smooth muscle. Note hemorrhage into endometrial stroma. (hematoxylin and eosin stain; original magnification × 100).

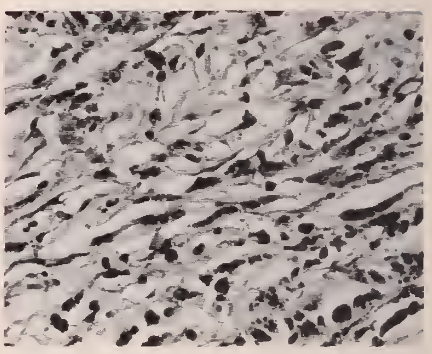

FIGURE 2. High power view of endometrial stroma showing metaplastic changes and hemorrhagic foci. (hematoxylin and eosin stain; original magnification × 450).

Ovarian tumors requiring exploratory laporotomy and pathologic examination include those that are enlarging, or are solid, complex, fixed, irregular, shotty, bilateral, hemorrhagic, twisted, or likely to interfere with labor. If surgery is required during early pregnancy, it should be done at 14–16 weeks. Delay until the early second trimester may allow the cyst to regress, avoids injury to a corpus luteum of pregnancy in the same ovary, and reduces the incidence of spontaneous abortion. If the need for surgery arises late in pregnancy, questions of fetal viability may influence timing. A short delay in surgical treatment to allow fetal maturation may be appropriate. Balancing the risk of delaying treatment to the mother with risks to the fetus of intervention can be a formidable task.[7]

References

1. White KC: Ovarian tumors in pregnancy: A private hospital ten year survey. *Am J Obstet Gynecol* 1973; 116:544–550.
2. Novak ER, Lambrou CD, Woodruff JD: Ovarian tumors in pregnancy: An ovarian tumor registry review. *Obstet Gynecol* 1975; 46:401–406.
3. Droegemueller W, Herbst AL, Mishell DR, et al: *Comprehensive Gynecology*, ed 1. St Louis, CV Mosby Co, 1987, p 479.
4. Scott R: Endometriosis and pregnancy. *Am J Obstet Gynecol* 1944; 47:608–632.
5. Birken RA, Stern SE, Halipolo MH: Case report: Large endometrioma in pregnancy. *Texas Med* 1982; 78:50.
6. Moll UM, Chumas JC, Chalas E, et al: Ovarian carcinoma arising in atypical endometriosis. *Obstet Gynecol* 1990; 75:537–539.
7. Graber EA, Feldman GB: What to do about ovarian tumors in pregnancy. *Contemp Ob Gyn* 1985; 26:129–138.

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Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

Appendiceal abscess presenting as an infected urachal cyst

TO THE EDITOR: The differential diagnosis of supravesical masses includes disorders of the urachus. Infected cysts result in symptomatology consistent with suprapubic pain, tenderness, and associated voiding dysfunction.[1] We present a case of a suspected infected urachal remnant, which on exploration was found to be an appendiceal abscess. The clinical course, radiographic findings, and pathology are presented to alert the clinician to the varied presentation of an appendiceal abscess.

Case Report. A 27-year-old male complained of suprapubic discomfort which caused a sensation to void. Upon voiding, the pain radiated to his right testicle. There was no relationship to sexual intercourse or other activities. Initial physical examination revealed a well-developed male with a BP of 160/60, pulse of 80, and a temperature of 98.6°F. His abdominal examination was unremarkable as was the rectal and genital examination. Hematologic and blood chemistries were normal, and urine culture was negative, and a urinalysis revealed 1–2 white blood cells. A neurologic evaluation, including cystometry, was normal.

The patient underwent cystoscopy, revealing bullous edema of the right lateral and anterior wall of the bladder. Bladder biopsies were obtained and a bimanual examination revealed a soft, movable supravesical mass. Urinary cytologies were negative, and the pathology revealed acute inflammation. A contrast computed tomography (CT) scan of the abdomen and pelvis revealed an irregular, thick-walled, bladder with a 3 × 3 × 4 cm fluid-filled structure anterior to the bladder (Fig 1). A cystogram did not show communication between the bladder and the mass. The radiologic impression was that the patient had a urachal cyst. He then presented with a temperature of 102°F and complained of dysuria. A repeat CT scan did not reveal changes of the mass. He was started on parenteral antibiotics and underwent surgical exploration for a suspected infected urachal cyst.

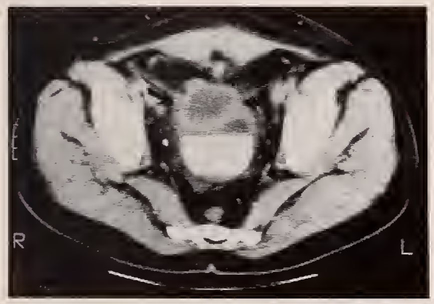

FIGURE 1. Pelvic CT scan demonstrating a 3 × 3× 4 cm cystic supravesical mass.

Laparotomy revealed an abscess cavity originating from the appendix draping the anterior wall of the bladder, and an appendectomy was performed. Pathologic sectioning revealed acute and chronic periappendicitis with abscess formation. Postoperatively, the patient recovered uneventfully.

Discussion. Appendicitis and appendiceal abscess may mimic urologic disorders including ureteral stones, ureteral obstruction, bladder masses, urinary retention, and urachal pathology.[2] Appendiceal abscess may also be difficult to determine radiographically.[3] In this case, the symptomatology and radiographic findings suggested an infected urachal remnant. Surgical exploration revealed the cause of the symptoms to be an appendiceal abscess. We present this case to remind physicians that an appendiceal abscess may mimic urachal pathology both on clinical and radiographic examination.

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2. Richie JP, Sacks SA, Rhodes D, et al: Urologic complications of appendicitis. *Urology* 1975; 6: 689–692.
3. Ngo C, Wong L, Yaghmai I: Unusual sonographic appearance of a perforated appendiceal abscess. *Pediatr Radiol* 1989; 19:335–336.

The importance of differentiating between anabolic steroids and glucocorticoids

TO THE EDITOR: Because of the widespread media exposure as well as physician education, there is increasing public awareness of the dangers of anabolic steroid use and abuse. Many are keenly aware of the acute and chronic risks of steroid use.

Unfortunately, physicians commonly use the word "steroids" when discussing glucocorticoid therapy with patients and families. Glucocorticoid therapy has been a mainstay of treatment for many common pediatric disorders and is being used with increasing frequency for acute inflammatory conditions of childhood. Recently, it has come to our attention that, in spite of extensive parental education concerning the desired actions and possible side effects of oral prednisone therapy, children seen in our clinics with vasculitis, nephrotic syndrome, and asthma were not receiving their prescribed prednisone therapy from their parents. In each instance, the reason the parental noncompliance was anxiety related to "steroid" use. One parent even stated that he did not wish his son to look like a body builder.

The clear distinction between anabolic steroids and glucocorticoid therapy should be explained to patients and to parents of pediatric patients. Physicians should be acutely aware of public sensitivity about the word "steroids" and be more specific when discussing anti-inflammatory prednisone therapy with patients and families. Furthermore, patients currently receiving glucocorticoid therapy should be counseled about the differences between anabolic and glucocorticoid therapy. Physician awareness of the public's fears of steroids may avoid therapeutic failures caused by non-compliance.

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The physician and the movies

TO THE EDITOR: Over the years physicians have been portrayed on movie screens as heroes, villains, or goats.

We are reminded of our image by two recent movies *The Doctor* and *Article 99*, a dedicated group of physicians in M.A.S.H-like fashion attack the bureaucracy in a Veteran's Administration hospital. Both movies capture our humanity and sense of caring.

They speak not so much to our status as icons but that we too get ill and have to confront the maze of the modern hospital. We too have to fight for our patients and their rights.

Our concern for the health of our communities was once again evident at the recent House of Delegates Convention in New York City.

An example of this, is the resolution pledging to work with the New York City Department of Health in combating the return of tuberculosis, a modern day scourge. The resolution articulated what has to be done and declares that we, as physicians, are eager to help in the battle.

Whether it be medicine or whatever fate

holds in store for us, we are there. The movies, which to some extent, reflect the perceptions of society, are acknowledging our worth. This is not to say that some future writer will not describe us in scathing terms. Next, some producer will be fascinated by the plot and will spatter mud all over us. It is the price we must pay for our favored niche. Admiration and awe mixed with envy and hostility makes for quite a brew.

Let us enjoy this positive portrait and hope that it continues.

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The Phi Lambda Kappa Medical Fraternity: Religion and medicine

TO THE EDITOR: The history of Phi Lambda Kappa (PLK) Medical Fraternity dates back to its inception at the University of Pennsylvania Medical School in 1907. Since that time, its members have been actively engaged in furthering medicine and encouraging activities compatible with humanitarism. A non-profit philanthropic arm of the fraternity was founded in 1949 known as the Medical Students' Aid Society (MSAS) for the purpose of advancing medical education and science. MSAS aims to provide interest free tuition loans to medical students, housing aid, and awards for professional achievement. The aims of the fraternity and the purposes of MSAS are carried out without regard to race, creed, or color.

Currently, there are chapters at the following medical schools: the Alpha Beta (AB) chapter at Hahnemann University School of Medicine in Philadelphia, the AM Chapter at New York Medical College in Valhalla, the AP chapter at Chicago Medical School in North Chicago and the K chapter at SUNY Buffalo School of Medicine, in Buffalo.

The AB chapter at Hahnemann has been in existence for seven years and has a growing membership of 25 men and women who are medical or other allied health professional students. Several basic science and clinical faculty as well as practicing physicians also hold membership in the organization. There are also quite a few retired physician members. Our chapter does a great deal to enrich the lives of our members. We have agreements with local synagogues for our members to observe the sabbath and Jewish holidays. We also schedule "breaking the fast" dinners and other events that coincide with the Jewish holidays. Other various social events are scheduled throughout the year with the Jewish Graduate and Professional Student Network of Philadelphia, providing a means of interaction for Jewish students in post-bachelor programs at the colleges and universities in the Philadelphia area.

Our chapter also invites guest lecturers to speak each semester on both medical and religious topics. A Big Brother and Big Sister mentoring system has upperclassman and faculty members serving as "counselors" to help individual members with the problems of medical education and career planning. The chapter maintains an extensive file of academic resources which are available to all members. Round table discussions with clinicians are scheduled throughout the year to aid students in choosing a medical specialty. We also maintain a file of elective clerkship opportunities in Israel as well as a vast amount of information about the country.

The chapter sends representatives to the annual PLK and MSAS business meeting and scientific sessions. Lectures are given by distinguished physicians and often times by PLK members. These meetings are both enjoyable and informative for the students and physicians. Physicians who attend the scientific sessions receive 25 Category I continuing medical education credits from the American Medical Association.

Through the MSAS, medical student members are eligible to apply for interest-free loans of up to $1,500.00 per year. MSAS also offers advise to residents and physicians about setting up a practice. The MSAS is supported by voluntary contributions from PLK members, clubs, pharmaceutical firms and friends both in and out of the profession.

To join PLK, each member pays national dues of $25.00 for the first year and $15.00 for each year thereafter. These dues entitle each member to receive a quarterly journal, PLK national literature, an invitation to our national convention, and a fraternity initiation pin.

We at the AB Chapter are proud to be members of PLK. It gives us the opportunity to further our knowledge of both medicine and Judaism. We feel that these interactions will ultimately make us more caring and informed physicians. If you are interested in information about the Phi Lambda Kappa Medical Fraternity, please write to Mrs Eleanor Halpern, National Executive Secretary of PLK, Bucks County Office Center, 1200 New Rodgers Road, Box 805, Bristol, Pennsylvania 19007 or call (215) 785-2325.

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THE HISTORY OF YELLOW FEVER

Edited by Francois Delaporte. 181 pp. Cambridge, Mass, The MIT Press, 1991. $22.50 (hardcover)

This book is almost poetic in its language and imagery but at the same time, confusing, verbose and sometimes incomprehensible. I found that frequently I had to return to the beginning of a sentence to find out what or who was the subject of the described activity. Truthfully, this is not the author's fault. The book was written in French and the translation of certain concepts owes its clumsiness to the inherent differences in the two languages. The French language can say more in fewer words owing to its nuances. English requires that these be written out. Certain themes and rhetorical questions seem to be repeated endlessly. Nevertheless, in reading this book one has a sense of the marvelous renaissance that was occurring in the fields of infectious diseases, entomology and most particularly tropical medicine. It is exciting to be brought into the milieu of Manson, Ross, Laveran, Finlay and Reed, to name only a few. The reader is transported back in time to a period when the concepts of vectors was first being elucidated. Scientists had to ask questions that produced original thinking, that rarest of all thought.

The book essentially describes the conflict between Carlos Finlay, who described the mosquito *Culex fatigans* as the vector of yellow fever, and Walter Reed who proved this theory. The author then explains the 18-year hiatus between elucidation of the theory and its proof. Conciliatory and antagonistic accounts of events are reviewed. As this volume shows, research on filariasis (Manson), malaria (Laveran), and yellow fever produced unique inquiries and theories.

The author sets out to describe how these resulting theories overlapped, how they were limited and finally to clarify the exchange of methods, techniques, and models among workers in the various areas.

It is fascinating to be transported back to a period of history when physicians considered noxious fumes, foul breezes, the atmosphere, and rotting garbage as the agents of disease. Physicians in Mexico, Brazil, and Cuba where yellow fever was endemic, proposed a bacterium and various fungi as the agent. Such diverse results produced violent polemics. Nationalism, politics and egos gave rise to charge and countercharge. Gradually, France and the United States became involved with equally caustic criticism of South American medical research. Again the names of such contemporaries as Koch, Pasteur, Gaffky, Metchnikoff, Roux, Yersin and others from the golden age of bacteriology come alive in this text, in spite of the often laborious sentences.

The author is a historian of 18th and 19th century medical investigations, having written two prior books. The first, is on the history of plant physiology in the 18th century, and the second deals with the cholera epidemic and its effects on Paris in 1832. This book, his third, addresses the conflict surrounding the elucidation of the agent of yellow fever and the disease's transmission. It is certainly a welcome and well researched addition to the literature of the history of medicine.

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THE CHANGING CONTEXT OF SOCIAL-HEALTH CARE: ITS IMPLICATIONS FOR PROVIDERS AND CONSUMERS

Edited by Helen Rehr and Gary Rosenberg. 143 pp. Bingamton, NY, Haworth Press, 1991. $14.00 (softcover)

"America needs a national health plan—a strategy that covers essential, universal coverage without financial barriers to access, is equitable and soundly financed and offers fair payment to providers," and which "encourages appropriate treatment and appropriate utilization by consumers."

With due credit for the quoted phrase attributed to a July, 1989, article in the American Public Health Association's newspaper, *The Nation's Health,* the above statement sums up the essence of this book. In addition, a constant theme of this volume deals with the future implementation of a new social-health policy orientation based on people-related needs. That orientation would be in contrast to the traditional dominating medical focus observed in the US health care delivery system. Social health is an expansive term that incorporates economic, political, and technological factors that are shaping the health care delivery system. Rehr and Rosenberg conclude that "For all Americans, a national health insurance coverage is socially and morally correct."

Essentially, this book reports on a colloquium that took place on October 20 and 21, 1988. Speakers included Dr Dennis O'Leary, President of the Joint Commission on Accreditation of Healthcare Organizations (JACHO), discussing health care today and tomorrow; Emily Friedman, a contributing editor for *Medical World News, Health Care Forum Journal, Hospitals, Health Business* and the *Journal of the American Medical Association,* talking about patients as partners; Bess Dana, Professor Emerita, Department of Medicine, Mount Sinai Medical School, reporting on five workshops and her deliberations about reaching for the social-health benefits of medical care; and finally the updated conclusions and recommendations by Helen Rehr and Gary Rosenberg, Professor Emerita and current Professor, Department of Community Medicine (Social Work), at Mount Sinai.

Much of the book reiterates the often mentioned difficulties in providing health care in the US such as access, commercialization, facilities, indigent care, patient education, professional education, quality of care, rationing, and reimbursement. Certainly, these issues are not new to practicing physicians and have appeared in numerous professional and popular mass media outlets ad nauseam.

Relative to reforming the health care system, Rehr and Rosenberg agree with Carol McCarthy, the past President of the American Hospital Association. In an August 1989 article in *Hospitals,* McCarthy posited that the tenets of health care service reform should be high quality; community focused; efficiently delivered; conducive to innovation; adequately and fairly financed; affordable; as far as essential services are concerned, available to all; and provide a sufficient supply of timely access.

Although these tenets and the problems with the US health care system are repeatedly enumerated, the solutions are not as obvious. Philosophical recommendations in this book will be taxing for physicians to comprehend if they are seeking easily identifiable and applicable resolutions. Many books already on the market come forward with more apparent rationalizations for changing the health care delivery system. In this book, the mixture of social and health care may be alien to physicians schooled to approach their patients with a narrowly focused concentration on strictly medical services.

In summary, this book will be of interest to the minority of providers who want to hear another visionary viewpoint. Most clinicians will not find this book to their liking. Are the authors seeking a golden grail? Perhaps. Friedman quotes management expert Peter Drucker who states that "Miracles are great, but they are so damned unpredictable."

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PRACTICAL HANDBOOK OF JOINT FLUID ANALYSIS

Edited by Robert A. Gatter, MD, and Ralph Schumacher, MD Second Edition. 122 pp, illustrated. Malvern, Pa, Lea & Febiger, 1991. $42.50 (hardcover)

This monograph on joint fluid analysis is now in its second edition and has acquired a new co-author, Dr Schumacher, and two new chapters. The authors, especially Schumacher, have published extensively in the area of synovial fluid research. Schumacher, in fact, maintains a reference laboratory for identification of unknown crystals. The book is a small, easily read "handbook" that takes the reader from joint aspiration to special stains for cellular and crystalline material.

A chapter on physiology and pathophysiology reviews characteristics of normal joint fluid and explains why normal joint fluid does not clot. The chapter on joint aspiration, indications and technique, has been expanded and there are very clear descriptions of where to place a needle for aspiration of most joints. The majority of other chapters are unchanged, including the chapter on compensated polarized light which continues to admonish against the use of the term negative or positive "birefringence" instead of negative or positive elongation.

This book is a must for the library of every rheumatologist (unless they already have the first edition), and anyone interested in tapping joints and appropriately examining joint fluid. It can be read in less than an hour and contains everything one needs to know about joint fluid.

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THE PEDIATRIC AIRWAY

Edited by H. Biemann Othersen, Jr., MD. 229 pp, illustrated. Philadelphia, Pa, WB Saunders, 1991. $75.00 (hardcover)

This book, as indicated by the author in the preface, is intended for the many specialists who deal with problems of the pediatric airway and presents treatment regimens that have been found to be successful in their management.

The editor of this volume has gathered a group of prestigious and experienced pediatricians, pediatric surgeons, otolaryngologists, radiologists and anesthesiologists to contribute chapters covering their field of expertise.

Although a small volume, the book covers general considerations such as embryology and development, pulmonary physiology, radiologic aspects and medical diseases of the airway and contains, as well, chapters that deal with congenital and acquired problems such as subglottic stenosis and tracheobronchial stricture, vascular malformations, tracheomalacia, and airway injuries. In another section, methods of laryngotracheoplasties are described. General techniques such as anesthesia for airway problems, endoscopy, management of foreign bodies in the airway, tracheostomy, and home care of a child with tracheostomy are described in depth. In the final page, listed as chapter 18, the editor discusses his view of future directions in dealing with pediatric airway problems and lists six important reminders for existing methods of management. These include using tubes of proper size; securing endotracheal tubes well to prevent shearing forces that may result from movements of tube or head and neck; not using cuffed tubes in children unless necessary; taking meticulous care with tracheostomy tubes, clean stoma and cauterize granulations; performing tracheostomy when the larynx or upper trachea are inflamed or edemetous or when endotracheal intubation is prolonged and advising early repair of blunt or penetrating airway injuries since this gives the best results.

Each of the chapters is well-organized. The material presented is current, clearly written, and accurate. The embryology, development, pulmonary physiology, radiologic aspects of airway obstruction, and the surgeon's role in medical diseases of the airway are succinctly covered in the first section.

The remaining sections similarly are well-organized and the material in them is discussed clearly and thoroughly. In the chapters dealing with specific problems, each specialist-author presents his or her approach to management by describing the problem, what is done to correct it, how it is done, and why that particular method of treatment was chosen. All of the sections are illustrated by appropriate roentgenograms or drawings. Unlike many multiauthored books, the overall style and presentation of the text is more like a book with a single author and this is a credit to editor.

This book should serve as both a basic and experienced-based comprehensive, reliable and outstanding reference for pediatricians, pediatric surgeons, anesthesiologists, and nursing and critical care personnel who deal with infants and children with acute or chronic airway problems.

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GROWING UP. PASTORAL NURTURE FOR THE LATER YEARS

By Thomas B. Robb, ThD. 148 pp. Binghamton, NY, Haworth Press, 1991. $14.95 (softcover)

Dr. Robb's book attempts to integrate contemporary theological, sociological and psychological research into a comprehensive analysis of the process our culture commonly calls "growing old." The works of scholars such as Abraham Maslow, Eric Erikson, and Elizabeth Kubler-Ross are woven throughout the text. These theories, coupled with interesting statistics related to post-retirement years, make for interesting and enjoyable reading. For the most part, however, the information contained in the book is somewhat elementary and, I believe, would be enjoyed most by readers not familiar with the classical theories related to the stages of human development or contemporary studies dealing with death and dying.

The author makes a valid and challenging contribution in his discussion of the failure of most organized religions to provide for the special needs of the elderly members of their congregations. In particular, the author points to the need for strategic planning in developing comprehensive programs to meet the complex social, medical and psychological needs of the elderly. His accusations are underscored by his reminder that the elderly represent the fastest growing segment of most religious congregations in the United States. The author, (himself an ordained minister), notes that most churches continue to focus their energies and resources on developing programs for the younger members of their congregations in spite of statistics that show that one fifth to one fourth of the total membership of most denominations are now over 65 years of age.

In particular, I found the author's discussion of "rites of passage" for the elderly interesting and insightful. He criticizes church leaders for their lack of initiative in developing rituals which signal passage into the later quarter of the life cycle. Robb clearly underscores the need for the "transition rituals" which are used by most religious traditions to mark significant points in the individual's passage through the life cycle (ie, "birth" is marked by baptism or circumcision; "puberty" by confirmation or "Bar/Bas Mitzvah"; "Marriage" by public exchange of wedding vows). These rituals are important because they reflect major transitions in the life of individuals. "Rites of passage," states Robb are essential for they "tell us who we are and what we are to do. They also tell others what to expect of us, and how to behave toward us." Yet, argues Robb, ironically there are no religious ceremonies to mark passage into retirement or infirmity; no religious ceremony to tell the community that an individual has moved to another stage of development and thus the person's obligations, needs, and expectations are different.

The book also touches on some of the major issues of concern to both the elderly as well as to family caregivers (ie, widowhood, illness and the long-term care decision, home care supports, the process of dying, Medicare and Medicaid). Unfortunately, the author's treatment of these topics is extremely brief, and thus the discussion lacks substance for the reader who needs specific information.

The book has a decidedly Christian perspective and the New Testament provides the philosophical underpinnings of the text. Unfortunately, there is nothing new, startling, or provocative in Robb's text. The book, however, clearly articulates the cultural forces at work that stereotype individuals who are "senior citizens." Thus, the author challenges all of us to rethink the cultural assumptions, which hold all of us hostage to a youth-oriented culture that look with disdain and fear on the later years of life that sooner or later we all must embrace.

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MALE AESTHETIC SURGERY

Edited by Eugene H. Courtiss, MD. 435 pp, illustrated. St Louis, Mo, Mosby Year Book Publishers, 1991. $125.00 (hardcover)

This is a beautifully crafted book, entirely devoted to aesthetic surgery in the male patient. In addition to surgical technique, it emphasizes the underlying anatomic and physiologic differences between the sexes. The editor, Eugene Courtiss, is a well known expert in the field of aesthetic plastic surgery. The contributors of the various sections are also acknowledged experts in their respective fields.

The book is an important adjunct to the practicing plastic surgeon because of the increasing numbers of male plastic surgical patients who are seeking surgery and, also because there are true differences in the approaches to surgery in men and women. The book is divided into various sections as diverse as medical liability, physiology, wound healing, anesthesia, minority patients, baldness, facial and neck contouring, surgery of the aging face, and body contouring. The book is well organized and clearly written and is fairly unique in its field. It continues the high quality of the first edition and with incorporation of many new concepts. This book fully covers the field of male aesthetic surgery and is a must for all plastic surgeons.

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BOOKS RECEIVED

CARDIOLOGY

5/1 Progress in Cardiology. Edited by Douglas P. Zipes and Derek J. Rowlands. 170 pp. illustrated. York, Pa, Williams & Wilkins, 1992. $45.00 (softcover)

Rapid Analysis of Electrocardiograms. A Self-Study Program. Second Edition. By Emanuel Stein, MD. 404 pp. York, Pa, Williams and Wilkins, 1992. $26.95 (softcover)

CLINICAL PATHOLOGY

Interpretation of Diagnostic Tests. A Synopsis of Laboratory Medicine. Fifth Edition. By Jacques Wallach, MD. Boston, Mass, Little, Brown and Company, 1992. $29.50 (softcover)

GERIATRICS

Handbook of Prescribing Medications for Geriatric Patients. By Judith C. Ahronheim, MD. 465 pp. Boston, Mass, Little, Brown & Co, 1992. $26.50 (softcover)

Vision and Aging. Issues in Social Work Practice. Edited by Nancy D. Weber, MSW. 196 pp. Binghamton, NY, Haworth Press, 1991. $14.95 (softcover)

Residential Care Services for the Elderly. Business Guide for Home-Based Eldercare. Edited by Doris K. Williams, PHD. 106 pp. Binghamton, NY, Haworth Press, 1991. $19.95 (hardcover)

Story Writing in a Nursing Home. A Patchwork of Memories. By Martha Tyler John, MD. 137 pp, illustrated. Binghamton, NY, Haworth Press, 1991. $19.95 (hardcover)

HEALTH SERVICES

Set No Limits. A Rebuttal to Daniel Callahan's Proposal to Limit Health Care for the Elderly. Edited by Robert L. Barry, OP, and Gerard V. Bradley. 134 pp. University of Illinois Press, 1991. $22.95 (hardcover)

Improving Access to Health Services for Children and Pregnant Women. By Joshua M. Wiener and Jeannie Engel. 90 pp. Washington, DC, Brookings Institution, 1991. $8.95 (softcover)

Strategic Health Planning. Methods & Techniques Applied to Marketing and Management. By Allen D. Spiegel and Herbert H. Hyman. 440 pp. Norwood, NJ, Ablex Publishing Corp, 1991. $45.00 (softcover)

Kidney Failure and the Federal Government. Edited by Richard A. Rettig and Norman G. Levinsky. 426 pp. Washington, DC, National Academy Press, 1991. $49.95 (hardcover)

HEMATOLOGY

Blood. Atlas and Sourcebook of Hematology. Second Edition. By Carola T. Kapff, SH, and James H. Jandl, MD. 158 pp, illustrated. Boston, Mass, Little, Brown & Co, 1991. $95.00 (hardcover)

Fibrinolysis, Thrombosis and Hemostasis. Concepts, Perspectives, and Clinical Applications. By Sol Sherry, MD. 318 pp, illustrated. Philadelphia, Lea & Febiger, 1992. $59.50 (hardcover)

Genetics of Hematological Disorders. Edited by Christos S. Bartsocas and Dimitris Loukopoulos. 306 pp, illustrated. Bristol, Pa, Hemisphere Publishing Corp, 1992. $95.00 (hardcover)

INFECTIOUS DISEASES

Manual of Antibiotics and Infectious Diseases. Seventh Edition. By John E. Conte, Jr, MD, and Steven L. Barriere, 419 pp. Philadelphia, Lea & Febiger, 1992. $29.95 (softcover)

MEDICAL EDUCATION

Beyond Flexner. Medical Education in the Twentieth Century. Edited by Barbara Barzansky and Norman Gevitz. 246 pp. Westport, CT, Greenwood Publishing Group, Inc, 1992. $49.95 (hardcover)

MEDICAL MALPRACTICE

The Guide to Medical Professional Liability Insurance. By Kirk B. Johnson, JD; Martin J. Hatlie, JD, Illene Davidson Johnson, JD. 146 pp. American Medical Association, 1991. $24.00 (softcover)

Grand Rounds on Medical Malpractice. By Francis X. Campion, MD. 372 pp. Chicago, American Medical Association, 1990. $40.00 (softcover)

MISCELLANEOUS

Family Systems Application to Social Work. Training and Clinical Practice. Edited by Karen Gail Lewis. 203 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

The Day America Told the Truth. What People Really Believe About Everything That Really Matters. By James Patterson and Peter Kim. 270 pp. New York, Prentice Hall Press, 1991. $19.95 (hardcover)

First Aid for the Boards. A Student-to-Student Guide. By Vikas Bhushan, MD, Edward Chu, Jeffrey Hansen, MD. 136 pp. Norwalk, Ct, Appleton & Lange, 1992. $9.95 (softcover)

Crusading Doctor. My Fight for Cooperative Medicine. By Michael A. Shadid, MD. 283 pp. Norman, Oklahoma. University of Oklahoma Press, 1992 (reprint). $18.95 (hardcover)

The American Dentist. A Pictorial History with a Presentation of Early Dental Photography in America. By Richard A. Glenner, DDS, Audrey B. Davis, PHD, Stanley B. Burns, MD. 194 pp, illustrated. Missoula, Mt, Pictorial Histories Publishing Co, 1990. $19.95 (hardcover)

Multigenerational Family Therapy. By David S. Freeman, DSW. 398 pp. Binghamton, NY, Haworth Press, 1992. $24.95 (softcover)

The Effect of Children on Parents. By Anne-Marie Ambert, PHD. 308 pp. Binghamton, NY, Haworth Press, 1992. $19.95 (softcover)

NEPHROLOGY

Renal and Electrolyte Disorders. Fourth Edition. Edited by Robert W. Schrier, MD. 846 pp. illustrated. Boston, Ma, Little, Brown & Co, 1992. $45.00 (hardcover)

OBSTETRICS AND GYNECOLOGY

Menstrual Health in Women's Lives. Edited by Alice J. Dan and Linda L. Lewis. 301 pp, illustrated. Urbana, Ill, University of Illinois Press, 1992. $16.95 (softcover)

PEDIATRICS

Adolescents and Their Families. By Mark Worden, PHD. 209 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

Adverse Effects of Pertussis and Rubella Vaccines. Edited by Christopher P. Howson, Cynthia J. Howe, and Harvey V. Fineberg. 367 pp. Washington, DC, National Academy Press, 1991. $39.95 (hardcover)

PSYCHIATRY

Disorders of Learning in Childhood. By Archie A. Silver, MD and Rosa A. Hagin, PHD. 683 pp, illustrated. New York, John Wiley & Sons, Inc, 1990. $69.96 (hardcover)

Women, Girls, and Psychotherapy. Reframing Resistance. Edited by Carol Gilligan, Annie G. Rogers, Deborah L. Tolman. 272 pp. Binghamton, NY, Harrington Park Press, 1991. $14.95 (softcover)

Drug-Induced Dysfunction in Psychiatry. Edited by Matcheri S. Keshavan and John S. Kennedy. 339 pp. Bristol, Pa, Hemisphere Publishing Corp, 1992. $59.50 (hardcover)

Integrating Individual and Family Therapy. By Larry B. Feldman, MD. 210 pp. New York, Brunner/Mazel Publishers, 1992. $25.95 (hardcover)

Family-of-Origin Therapy: An Intergenerational Approach. James L. Framo, PHD. 248 pp. New York, Brunner/Mazel Publishers, 1992. $29.95 (hardcover)

The Couple's Hypnotic Dance. Creating Ericksonian Strategies in Marital Therapy. By Carol J. Kershaw. 250 pp. New York, Brunner/Mazel, 1992. $29.50 (hardcover)

Treating Sex Offenders in Correctional Institutions and Outpatient Clinics. A Guide to Clinical Practice. By William E. Prendergast, PHD. 233 pp, illustrated. Binghamton, NY, Haworth Press, 1991. $14.95 (softcover)

SUBSTANCE ABUSE

Cocaine, AIDS, and Intravenous Drug Use. Edited by Barry Stimmel, MD. 124 pp. Binghamton, NY, Haworth Press, 1991. $8.95 (softcover)

What Works in Drug Abuse Epidemiology. Edited by Barry Stimmel, MD. 150 pp. Binghamton, NY, Haworth Press, 1991. $29.95 (hardcover)

Feminism and Addiction. Edited by Claudia Bepko, MSW. 224 pp. Binghamton, NY, Haworth Press, 1991. $17.95 (softcover)

Homelessness and Drinking. A Study of a Street Population. By Bernard Segal, MD. 150 pp. Binghamton, NY, Haworth Press, 1991. $24.95 (hardcover)

VASCULAR SURGERY

Laser Disobstruction and Laser-Assisted Balloon Angioplasty. A Color Atlas. By Teruo Matsumoto. 97 pp, illustrated. St Louis, Mo, Ishiyaku EuroAmerica, Inc, 1991. $75.00 (hardcover).

Harold A. Bergner, MD, Roslyn. Died April 4, 1992; age 74. Dr Bergner was a 1945 graduate of the Middlesex University School of Medicine, Waltham. His memberships included the American Society of Clinical Hypnosis, the American Geriatrics Society, the Academy of Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Alexander M. De La Garza, MD, Flushing. Died April 25, 1992; age 63. Dr De La Garza was a 1953 graduate of the Univ. Nacional Autonoma De Mexico Facultad De Medicina, Mexico, D.F. He was a Diplomate of the American Board of Surgery. He was a Fellow of the American College of Surgeons. His memberships included the Queens County Medical Society and the Medical Society of the State of New York.

Wilfred Dorfman, MD, Brooklyn. Died April 12, 1992; age 83. Dr Dorfman was a 1934 graduate of New York University College of Medicine, New York. He was a Diplomate of the American Board of Internal Medicine and the American Board of Psychiatry and Neurology. He was a Fellow of the American College of Physicians. His memberships included the American Association Psychoanalytic Physicians, the American Psychiatric Association, the Academy of Psychosomatic Medicine, the Pan American Medical Association, the Kings County Medical Society, and the Medical Society of the State of New York.

Emerick Friedman, MD, Pompano Beach, Florida. Died December 16, 1991; age 82. Dr Friedman was a 1934 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Psychiatry and Neurology. His memberships included the American Geriatrics Society, the American Psychiatric Association, the American Academy of Neurology, the Albany County Medical Society, and the Medical Society of the State of New York.

Joshua L. Galiani, MD, Pelham Manor. Died April 2, 1992; age 84. Dr Galiani was a 1934 graduate of the State University of New York Downstate Medical Center, New York. He was a Fellow of the International College of Surgeons. His memberships included the Bronx County Medical Society and the Medical Society of the State of New York.

William August Griesau, MD, Troy. Died February 10, 1992; age 77. Dr Griesau was a 1941 graduate of Albany Medical College of Union University. He was a Diplomate of the American Board of Urology. He was a Fellow of the American College of Surgeons. His memberships included the Rensselaer County Medical Society and the Medical Society of the State of New York.

Albert W. Grokoest, MD, New York. Died September 22, 1991; age 75. Dr Grokoest was a 1943 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the American Rheumatism Association, the New York County Medical Society, and the Medical Society of the State of New York.

David Gurin, MD, Great Neck. Died April 25, 1992; age 88. Dr Gurin was a 1929 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the Nassau County Medical Society and the Medical Society of the State of New York.

Salvatore Gusmano, MD, Brooklyn. Died March 1, 1992; age 62. Dr Gusmano was a 1958 graduate of the Universita Degli Studi Di Roma, Facolta Di Medicina E. Chirurgia. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Surgeons and the American College of Obstetricians and Gynecologists. His memberships included the Pan American Medical Association, the American Fertility Society, the Kings County Medical Society, and the Medical Society of the State of New York.

Paul C. Jenks, MD, Elbridge. Died January 14, 1992; age 73. Dr Jenks was a 1943 graduate of the University of Vermont College of Medicine, Burlington. His memberships included the Seneca County Medical Society and the Medical Society of the State of New York.

Melbourne H. Lent, MD, Buffalo. Died April 11, 1992; age 72. Dr Lent was a 1943 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Urology. His memberships included the American Urological Association, the New York State Urological Society, the Erie County Medical Society, and the Medical Society of the State of New York.

Lillian E. McGowan, MD, Boynton Beach, Florida. Died September 8, 1991; age 83. Dr McGowan was a 1933 graduate of the New York University College of Medicine, New York. She was a Diplomate of the American Board of Psychiatry and Neurology. Her memberships included the American Psychiatric Association, the New York County Medical Society, and the Medical Society of the State of New York.

John H. Reid, MD, Troy. Died July 7, 1991; age 71. Dr Reid was a 1951 graduate of Albany Medical College of Union University. His memberships included the American Academy Family Practice, the Rensselaer County Medical Society, and the Medical Society of the State of New York.

Anthony C. Reiger, MD, North Palm Beach, Florida. Died March 14, 1992; age 83. Dr Reiger was a 1931 graduate of the State University of New York Health Science Center at Brooklyn, New York. His memberships included the Queens County Medical Society and the Medical Society of the State of New York.

Leon Roizin, MD, New York. Died March 6, 1991; age 80. Dr Roizin was a 1936 graduate of the Universita Degli Studi Di Milano, Facolta Di Medicina E Chirurgia. He was a Diplomate of the American Board of Pathology and the American Board of Psychiatry and Neurology. He was a Fellow of the American College of Pathologists. His memberships included the American Society of Clinical Pathologists, the American Society for Experimental Pathology, the American Association of Neuro- Pathologists, the American Psychiatric Association, the American Academy of Neurology, the American Neurological Association, the New York County Medical Society, and the Medical Society of the State of New York.

Edward W. Rosner, MD, Tonawanda. Died September 10, 1991; age 69. Dr Rosner was a 1949 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Surgery. His memberships included the Niagara County Medical Society and the Medical Society of the State of New York.

Edward George Sass, MD, Clearwater, Florida. Died April 9, 1992; age 84. Dr Sass was a 1935 graduate of Medizinische Fakultat Der Universitat, Wien. His memberships included the American Academy of Family Practice, the Queens County Medical Society, and the Medical Society of the State of New York.

Alvin Schuman, MD, Virginia Beach, Virginia. Died April 24, 1992; age 94. Dr Schuman was a 1923 graduate of the State University of New York Health Science Center at Brooklyn, New York. His memberships included the Kings County Medical Society and the Medical Society of the State of New York.

Michael Sierp, MD, Wayne, Pennsylvania. Died April 6, 1992; age 72. Dr Sierp was a

1949 graduate of the Friedrich Alexanders University Medical Fakultat, Erlangen, Bavaria. He was a Diplomate of the American Board of Urology. He was a Fellow of the American College of Surgeons. His memberships included the American Geriatrics Society, the American Urological Association, the Academy of Medicine, the Westchester County Medical Society, and the Medical Society of the State of New York.

I. Jackson Tartakow, MD, Syosset. Died August 25, 1991; age 94. Dr Tartakow was a 1926 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Preventive Medicine, Inc. He was a Fellow of the American College of Preventive Medicine. His memberships included the American Public Health Association, the New York State Academy of Preventive Medicine, the Nassau County Medical Society, and the Medical Society of the State of New York.

Philip Benjamin Wahrsinger, MD, North Miami, Florida. Died April 24, 1992; age 86. Dr Wahrsinger was a 1930 graduate of the State University of New York Health Science Center at Brooklyn, New York. His memberships included the Bronx County Medical Society and the Medical Society of the State of New York.

Harold Weinstein, MD, East Meadow. Died February 9, 1992; age 85. Dr Weinstein was a 1932 graduate of the New York Medical College, Flower and Fifth Avenue Hospitals, New York. His memberships included the New York Cardiological Society, the Kings County Medical Society, and the Medical Society of the State of New York.

Bernard Jacobs, MD, 1924–1992

Dr Bernard Jacobs, an internationally respected orthopedic surgeon, died at home on April 19, 1992 at the age of 68. He served as an orthopedic surgeon on the staff of New York's Hospital for Special Surgery for over thirty years, and was also Clinical Professor of Orthopedic Surgery at Cornell University Medical College and Chief of Orthopedic Surgery at the Bronx Veterans Administration Hospital. Dr Jacobs, who specialized in spinal surgery for much of his career, graduated from the University College Hospital, London, England in 1948. After serving as a house surgeon in Great Britain for two years he spent six months as a fellow at the Montreal Neurological Institute in Canada. In 1950, he came to the United States where he served as a resident in surgery for three years at Adelphi Hospital. As a Captain in the US Army Medical Corps, he was stationed in Trieste in 1953 and 1954 and later in Frankfurt, Germany for two years. From 1956 to 1959, he was a resident in orthopedic surgery at the Hospital for Special Surgery after which he was an Orthopedic Research Fellow for a period of three years at the same institution. At the Hospital for Special Surgery, Dr Jacobs occupied a number of important positions including Chief of the Foot Disorder Clinic, Chief Consultant to the Neck Clinic, Chief of Spinal Surgery, and Chief of Orthopedic Surgery at the Veterans Administration. He was a founding member of the Cervical Spine Research Society, President of the New York State Society of Orthopedic Surgeons, Chairman of the Orthopedic Section of the Medical Society of the State of New York, Chairman of the Orthopedic Section of the New York Academy of Medicine, Chairman of the Board of Councillors of the American Academy of Orthopedic Surgeons and a member of the Board of Governors of the American College of Surgeons. In addition, he was a member of many other professional societies and a Diplomate of the American Board of Orthopedic Surgery.

Bernard, or "Bernie" as some affectionately called him, was a prolific scholar who published numerous articles in peer-reviewed scientific medical journals. Residents and students were not only inspired by his vast knowledge of orthopedic surgery, but also by his enthusiasm. He carried this enthusiasm into everything he did, including what otherwise might have been very dull committee meetings. This writer served as a member of the Committee on Legislation of the New York County Medical Society in the late 1970s when Bernard was the Chair. His more frequent and agenda-packed meetings were initially of concern to some of those accustomed to a slower approach to problems. However, this concern was soon dispelled by the fact that he produced dramatic results. Bernard felt no hesitancy in approaching state legislators and members of Congress with regard to regulatory matters. His presentations were always concise, to the point, and above all, convincing. He brought to this effort the special perspective of a physician who had trained in Great Britain under a national health service. In later years, he devoted much time and energy to the medical malpractice crisis where his efforts were much appreciated by his colleagues.

Bernard viewed the Medical Society of the State of New York as having a crucial role in preventing the establishment of a system of government controlled medical care similar to the one he had experienced first-hand in Great Britain. He was also a strong supporter of the *Journal.* His opinions were regularly sought not only on scientific topics related to his specialty, but also on socio-economic ones for which he had great expertise. He was always generous with his time as a peer reviewer, and we greatly valued his wise counsel and advice.

The last scientific article he wrote, "Alcoholism-Induced Bone Necrosis," appears in this issue of the *Journal.* He submitted it in December 1990, and it was peer reviewed and recommended for publication in early 1991. However, his last illness prevented him from revising it until January of this year.

Dr Jacobs is survived by his wife, Ingrid, a son Mark Jacobs of Washington, DC, a daughter, Karen Lauder of New York, a son-in-law, William Lauder and a granddaughter, Rachel Jennifer Lauder. A memorial service was held on Friday, May 8, 1992, at the Hospital for Special Surgery, and donations are being accepted to the Bernard Jacobs Fund and Fellowship in Spinal Research at the Hospital for Special Surgery.

Pascal James Imperato, MD
Editor

Children of Hippocrates 1992

There is the
struggle to establish
with moonlighting of all types
then being too busy and
the difficulty of getting away
the piles of paperwork to do
waiting rooms with crowds of people
or sometimes not enough
people

We hear about
Gate Keeping
DRGSs
new technologies
defensive medicine
disgruntled colleagues.
And don't forget
the undergrad years with so much study
the hope
of medical school
the long courses, long hours, long labs
and it all cost so much money.

Don't forget residency
being on call
studying
certification exams
and re-certification
and CME

It is very easy to lose sight of what it's all about,
what it means to be a physician. We probably don't
appreciate the profound effect we have on people.
We save lives and take it for granted.

We relieve pain and suffering
and go on
to the
next
case.

Doctors are so busy . . .
rushing to rounds
conferring with house-staff
consulting with colleagues
doing admissions
looking at films
on the phone with results
examining patients
doing procedures
then speaking to patients
and speaking to families
and managing the office
and signing the checks
and paying the bills.

Doctors are so barraged by
malpractice insurance
accepting assignment
HMOs, PPOs, PROs, IPAs,
answering service troubles,
missed appointments, "no shows,"
carve out days,
unpaid bills
the Medicare freeze
and R-B-R-V-S!

There are
exhausted medical supplies to reorder
there are meetings with office staff
accountants
and salesmen
and drug reps,
and new tax codes to contend with
and don't forget
the wife
and
kids.

We comfort the sick and those who feel hopeless
and just assume it's all part of a day's work.
We listen.
We heal.
We help.
Give hope
make a difference . . .

What a blessed privilege, still, to be a physician
and what greater joy
than to make this profession
our
life's
work!

MARTIN EDELSTEIN, MD

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1992; 92:122–125). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

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Samples references:

1. Bowen J: Autoimmune paraneoplastic diseases of the nervous system. *NY State J Med* 1991; 91:2–3.
2. Cotran RS, Kumar V, Robbins SL: *Robbins Pathologic Basis of Disease,* ed 4. Philadelphia, WB Saunders Co, 1989, pp 842–845.

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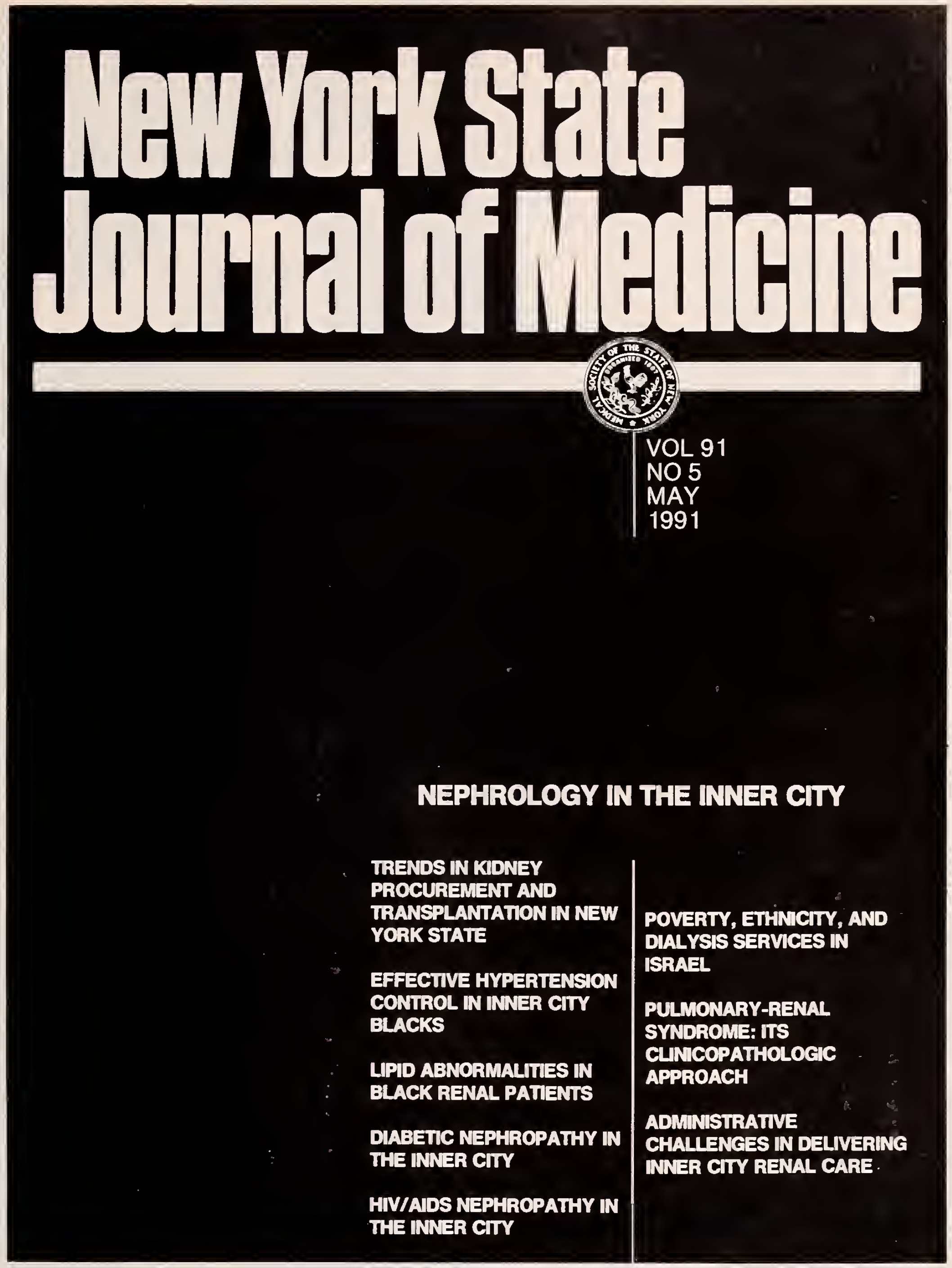

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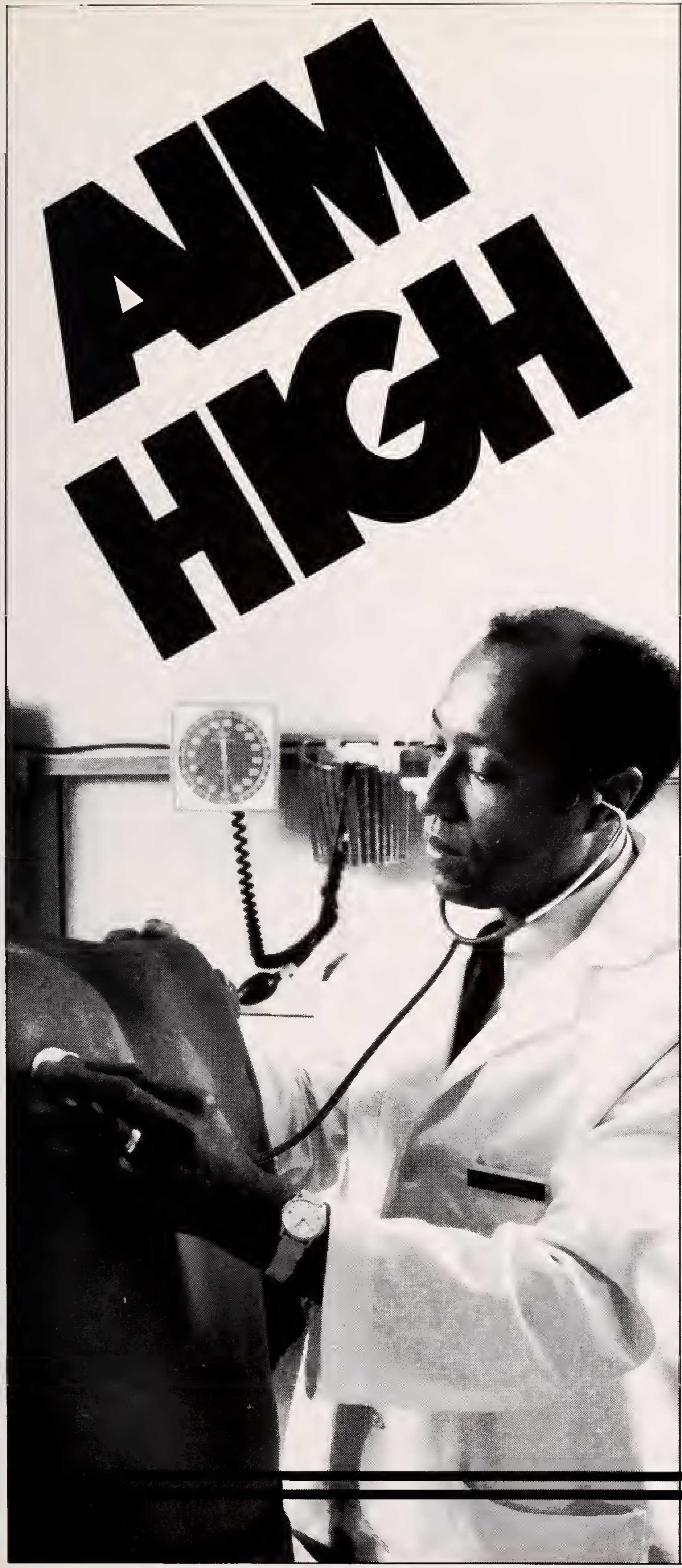

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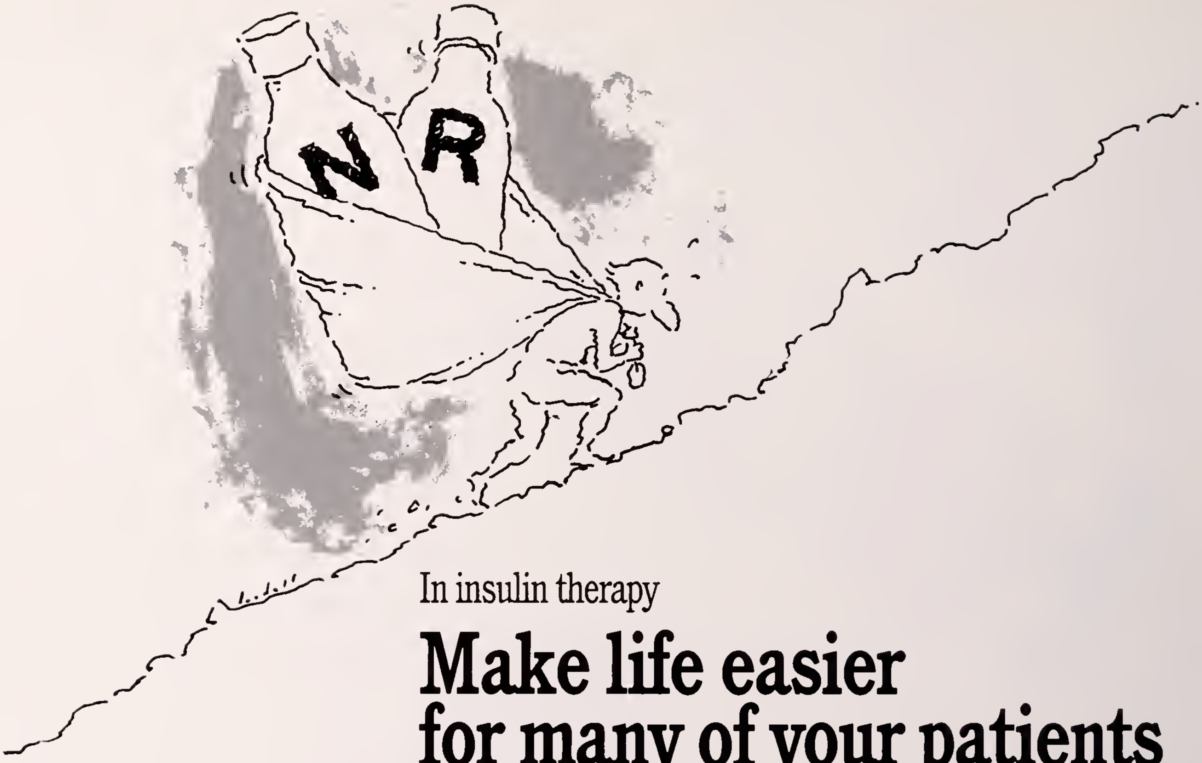

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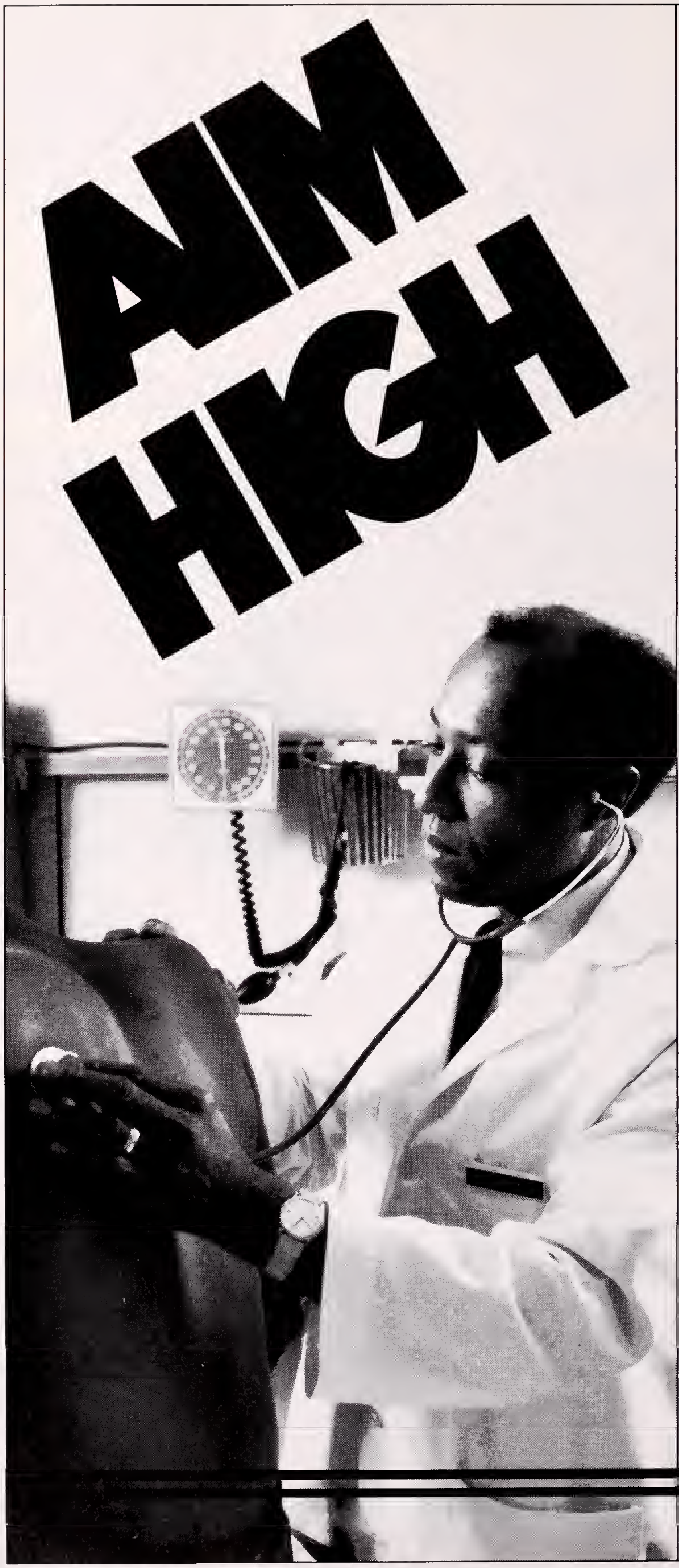

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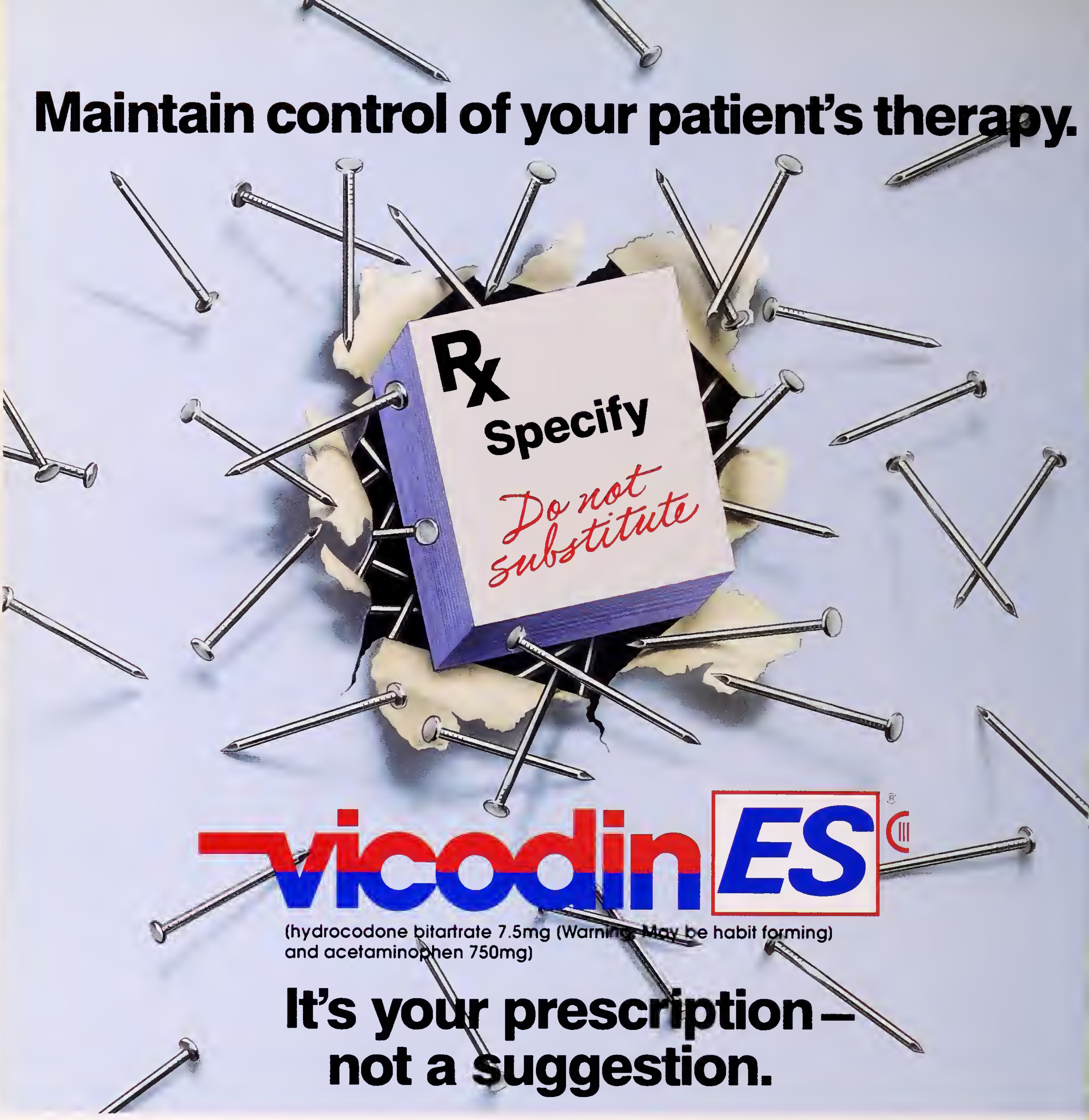

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The New York State Journal of Medicine (ISSN 0028-7628) is published monthly by the Medical Society of the State of New York.

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SUBSCRIPTION INFORMATION. The annual subscription rate for members is included in the annual membership dues. The subscription rate to nonmembers in the United States and Canada is $40.00 ($45.00 for all other countries). The single copy rate is $6.00 Domestic/Canada, and $7.00 Foreign, except for special issues. This includes postage. Change of address requests should give both the old and new address and allow six weeks for changeover.

The address for EDITORIAL, CIRCULATION, and ADVERTISING OFFICES is PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York, and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, PO Box 5404, Lake Success, New York 11042.

New York State Journal of Medicine

SEPTEMBER 1992 / Volume 92, Number 9

Contents

COMMENTARIES

375 **The physician and the courts**
RANDALL D. BLOOMFIELD, MD

376 **Television physicians**
JONATHAN D. MORENO, PHD, ALLEN D. SPIEGEL, PHD, MPH

RESEARCH PAPERS

378 **Cardiovascular risk factors in Native American children**
ANN S. BOTASH, MD; RAEELLEN W. KAVEY, MD; NANCY EMM, LPN; DIANE JONES

381 **The persistent vegetative state: An analysis of clinical correlates and costs**
DAVID MYLAND KAUFMAN, MD, RICHARD B. LIPTON, MD

SPECIAL ARTICLES

385 **Catfight in the labs**
NAOMI R. BLUESTONE, MD

386 **Not a valentine**
LOIS GREENE STONE

COMMITTEE REPORT

388 **Physician-assisted suicide**
FRED ROSNER, MD; PETER ROGATZ, MD; ROBERT LOWENSTEIN, MD, PHD; HERMAN M. RISEMBERG, MD; ALLEN J. BENNETT, MD; ANTHONY BUSCAGLIA, MD; ERIC J. CASSELL, MD; PETER B. FARNSWORTH, MD; ABRAHAM L. HALPERN, MD; JOHN B. HENRY, MD; BERNARD KABAKOW, MD; PIETER R. KARK, MD; ALLISON B. LANDOLT, MD; LAURENCE LOEB, MD; PATRICIA J. NUMANN, MD; FERNANDO V. ONA, MD; PHILIP H. SECHZER, MD; PETER P. SORDILLO, MD, PHD; JONATHAN R. WOLPAW, MD

(continued on p 6A)

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

OCTOBER 1992

AROUND THE STATE

ALBANY

Oct 16–17. **Prostate Ultrasound—Applications, Techniques and Advances in Prostate Ultrasound and Prostate Imaging.** Quality Inn. Contact: Office of Continuing Medical Education, Albany Medical College, 47 New Scotland Ave, Albany, NY 12208. Tel: (518) 445-5828.

BRONX

Oct 23. **Arthroscopic Surgery: Issues and Controversies.** 7 Cat I Credits. Montefiore Medical Center Cherkasky Auditorium. Contact: Lois Grossman, Program Director, Office of Continuing Medical Education, Albert Einstein College of Medicine, 3301 Bainbridge Ave, Bronx, NY 10467. Tel: (212)920-6674.

GARDEN CITY

Oct 14. **A Clinical Day in Ophthalmology. Selected Retinal Topics.** 7 Cat I Credits. Garden City Hotel, Garden City. Contact: June Lewis, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

MANHATTAN

Oct 10. **Alumni Day 1992: Laser Surgery in Ophthalmology.** 5.75 Cat I Credits. NYU Medical Center. *Oct 22–24.* **Advances in Dermatology.** 17.75 Cat I Credits. NYU Medical Center. *Oct 26–29.* **The Fourth Conference on DNA Topoisomerases in Therapy.** 25 AMA/PRA Cat I Credits. NYU Medical Center Alumni Hall. *Oct 20, 22, 27, & 29.* **Review of Medical Mycology #156.** 12 Cat I Credits. NYU Medical Center. *Oct 30.* **Diagnosis and Management of Ear, Nose and Throat.** 6 Cat I Credits. NYU Medical Center. Contact: NYU Medical Center, Post-Graduate Medical School, 550 First Ave, New York, NY 10016. Tel: (212) 263-5295.

Oct 14–17. **Renal Biopsy in Medical Diseases of the Kidneys.** 24.5 Cat I

(*continued on p9A*)

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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CONTENTS, CONT'D

New York State
Journal of Medicine

HISTORY

392 **History—The great reminder: The relationship between the Long Island College Hospital and the State University of New York Health Science Center at Brooklyn**
JOHN N. EDSON, MD

394 **Health care for seamen in the Port of New York—Part II: The rise of the Seaman's Retreat**
FLORENCE KAVALER, MD, MPH; SHIRLEY A. ZAVIN, PhD

CASE REPORTS

400 **Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder?**
STEVEN J. TURCHAN, MD; VALERIE F. HOLMES, MD; CHARLES S. WASSERMAN, MD

403 **Progressive pulmonary thromboembolism in association with HIV disease**
ROY MALIAKKAL, MD; SANDOR A. FRIEDMAN, MD; SUNDARA SRIDHAR, MD

404 **Unexpected reversal of an idiopathic dilated cardiomyopathy and the urgent need for a re-evaluation of the indicators of patient survival**
MICHAEL L. SACHER, DO; HOWARD L. SACHER, DO; STUART W. LANDAU, MD; ALLEN CHIEN, MS; FRANCES DOOLEY, RN; KATHLEEN A. DIETRICH, DO

406 **Acute emphysematous cholecystitis: A case report and review of the literature**
BOK Y. LEE, MD, CARLITO V. MORILLA, MD

LETTERS TO THE EDITOR

408 **The *New York State Journal of Medicine***
FRED B. CHARATAN, MD

408 **The future of the *New York State Journal of Medicine***
RICHARD H. LANGE, MD

408 **The physically handicapped children's program: A helping hand for children with serious health problems**
BERNARD N. MILLNER, MD

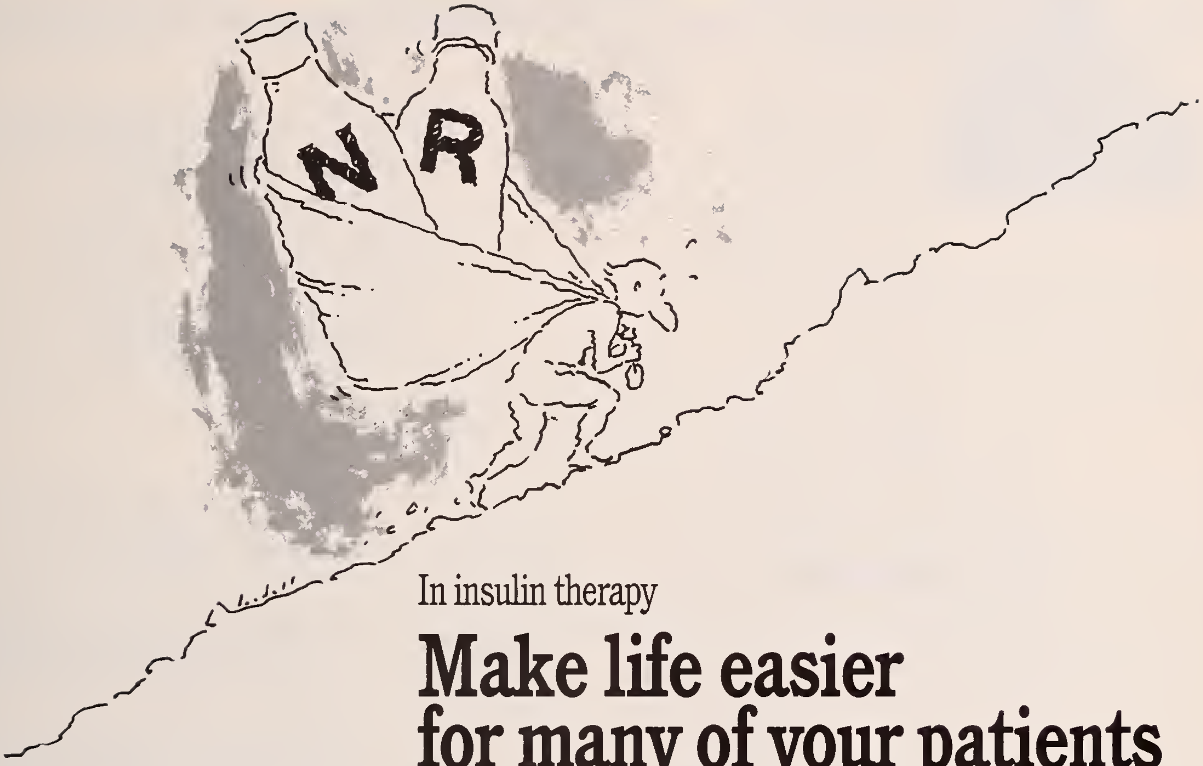
408 **Choice of surgical procedure for prostatic surgery**
REUBEN TIZES, MD

408 **Leiomyoma of the nipple**
YUSUF N. SILK, MD; MUHAMMAD A. HENA, MD; VERNE PILON, MD

410 BOOK REVIEWS

414 AUTHOR GUIDELINES

5A MEDICAL MEETINGS AND LECTURES



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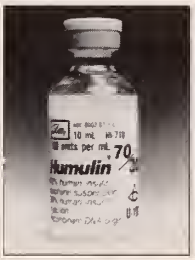

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COMMENTARIES

The physician and the courts

Much attention has been centered on the arcane world of physician reimbursement and Medicare regulations. In the midst of this preoccupation, medical liability is reasserting itself. The number and settlement costs of medical liability suits are once again increasing. Even while we seek federal relief and a substitute for the tort system, the ground is shifting beneath our feet. I would like to review some of the major factors affecting the course and outcome of malpractice litigation.

Site of court. Certain communities have achieved prominence because of their seeming sensitivity to the claims of the plaintiff. In Illinois, it is Cook County and in New York State it is Nassau County. According to informal queries, the Bronx and Kings are proving to be unfavorable areas for physician defendants.

The case. Is the case defensible? Has the defendant exercised the requisite problem solving, decision-making, management, and treatment skills? "*Res Ipsa Loquitur*," the thing speaks for itself, still defines the arena. An adverse outcome dominates the proceedings. Except in those states in which the standard of care applied becomes the arbiter, "outcome is everything." In obstetrics, hypoxia as a cause of cerebral palsy is successfully used by the attorney for the plaintiff, in spite of the evidence that shows the importance of genetic etiologies.

Attorneys. Some attorneys for the plaintiff have achieved considerable fame and fortune with their ability to sway juries. Attorneys for the defense are at a disadvantage since they try to present the potential consequences of surgery. A neurosurgical, orthopedic, or obstetric patient who is not made completely "whole" again is compelling evidence. Patient expectations for perfect results may be greater than the surgical limitations.

Principals. Court rooms fill many a doctor with dread. The attorney for the plaintiff is determined to paint the physician in the worst possible light. The plaintiff often gains the sympathy of the jury; for example, the little girl in pink with cerebral palsy who wears braces. Jurors feel that someone should pay for this. It seemingly makes no difference to them that the mother may have presented for the first time at a municipal hospital, ready to deliver.

Co-defendants. Often, everyone whose name is on the chart becomes involved in the case. The hospital is a prime target.

Exposure. The limits of the defendants' insurance coverage is an important factor, with the focus on the deepest pockets that can be tapped.

Negotiation. At some point, before or during the trial, a settlement may be offered by the defendant's insurance company. This is a two-edged sword. If the company for the insured settles too often, it can become known as a cash cow, thus initiating many suits. If the insurer defends a case that could have been settled for an acceptable figure, and the jury comes in with a large sum, recriminations and the search for a scapegoat can become the order of the day.

Physicians, for their part, must recognize that as unpleasant as the courtroom may be, this is now the price they have to pay as professionals. Accepting this, what can physicians do to stay afloat in a litigious environment?

Physician-patient relationship. A word of advice to physicians: Be nice! Hospitals must minimize the system failures that plague them, whether it be personnel behavior or lax reporting systems. Physicians must support their quality initiatives.

Medical students must be made aware of the litigious climate they face. They must realize that a knowledge of molecular biology may not convince a jury of their caring skills.

We as physicians tend to keep information about patients and their care in our heads. Unfortunately, failure to document these thoughts leaves us vulnerable when we have to defend our actions.

When there are several parties to the suit, finger pointing does not help. Plaintiffs' attorneys like nothing better than to divide and conquer. Physicians should prepare for the examination before trial and for the actual trial with the same intensity as they prepare for their licensing examination.

As physicians, we must seek to achieve order in the chaos that confronts us. We should continue to pursue alternatives to the present system, which is a prolonged, costly, demoralizing process that claims as its victims both the plaintiff and the defendant.

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Television physicians

A new television doctor show, *The Human Factor*, has recently appeared on the Columbia Broadcasting System (CBS). This TV entertainment deals with the emotional factors involved in medical care. In it, Dr Alec McMurty is featured as the latest TV physician, evoking the specters of Dr James Kildare, Dr Ben Casey, Dr Marcus Welby, St Elsewhere's healers, and assorted TV doctors and health care personnel. *The Human Factor* features people struggling with and suffering from illness and disease. Familiar elements include doctor and nurses, patients and relatives, professors and students, hospitals and ambulances and, inevitably, the life-saving, dramatic diagnosis and resolution. The new twist is that the patient requires attention as a person, not merely as someone who is ill. Health care workers are portrayed as being immersed in a saintly halo of caring, curing, compassion, and good will. The medical champions of this TV drama are portrayed as dedicated, overworked, overextended, and humanistic.

Thematically, *The Human Factor* asserts that the days of medical paternalism, as portrayed by TV, Drs Gillespie and Welby, are moribund. Physicians must now find a new way to express their humanity to their patients from a position of equality. In real life, both doctors and doctors-in-training sometimes stumble in this effort. What TV doctors and students end up doing is frequently indistinguishable from old-fashioned paternalism. When medical students behave paternalistically in *The Human Factor*, they look rather silly. When the humble-but-wise senior professor of medicine does these things, he looks, well, humble-but-wise. In fact, he looks a lot like a 1990s version of his erstwhile TV colleagues, Drs Gillespie and Welby, only more human and humble.

How should the TV audience react to these incongruities? Matters worsen when *The Human Factor's* Dr McMurty's nonpaternalistic paternalism leads him into the relentless and nonreimbursable pursuit of Christian Science parents who neglect their son's need for surgery. In a climactic scene, the mother says, "I took an oath." McMurty responds, "So did I," even though his oath is not relevant in this situation. In another disparity, McMurty inexplicably violates a patient's confidentiality after a medical student is nearly expelled for doing the same thing.

Long before the television doctor acquired the glamour of high technology, the human factor, also known as caring, was a fundamental principle. Hippocrates and other healers throughout the centuries conveyed the same or similar caring phrases in messages to their peers and to their students. At the Harvard University Medical School graduation ceremony of 1927, the eminent physician, Francis W. Peabody,[1] told the audience that, "The secret of the care of the patient is in caring for the patient." Research indicates that patients want two things from their physicians: technical competence and empathy.[2] Not surprisingly, patients will sacrifice empathy in favor of competence, if a choice has to be made.

No one can argue with the idealistic intent of the writers and producers of *The Human Factor*. Would that the behavior portrayed were so in real life. In spite of the procedures, good intentions, the lack of reality—in integrating what actually happens today in the health care system—is disappointing.

Every practitioner in the health care field is aware that cost containment is the critical element fueling the current crisis in the health care system.[3(pp43–48)] Even politicians have advanced health care to a top priority, as they see elections being won on this issue. Every medical educator is aware that students need to be prepared to enter the existing medical-industrial complex. Everyone watching *The Human Factor* is aware that altruistic medical care, as seen on this newest drama, may be rare.

In spite of all of the above, those of us who teach medical students, interns, and residents smile as we watch *The Human Factor*. It is heartwarming to see the dramatic blending of the art of care with the science of medicine. Therefore, we do not despair. We still strive to inculcate a caring attitude among our students. In fact, Spiegel has been teaching a seminar, "Curing and Caring: Complementary or Competitive Causeways?" for more than 20 years. Moreno teaches bioethics to medical students, interns, residents, and faculty.

Nevertheless, our pedagogical efforts would not be relevant if we somehow did not integrate prevailing health care economic concepts into our teaching. There is no way to escape the harsh economic realities in health care. Even the TV doctor must realize this and make the adjustment. In real life, physicians must also find an accommodation that allows them to combine curing with caring.

TV doctors, however, may create difficulties for genuine health care workers endeavoring to do the best that they can under trying situations. TV medical dramas may not engender authentic expectations among viewers. Historically, there were tearful laments from faithful viewers who yearned for a Dr Welby to cure and care for them at the same time. We know that parents were so impressed that they named their children "Kildare," hoping they would grow up to be compassionate doctors. Pointedly, if *The Human Factor* aims to depict contemporary doctors and patients, both the network and society may be better off with yet another documentary covering 24, 48, or 72 hours in an emergency room.

TV doctor shows may generate unrealistic desires in the viewing audience. As a result, physicians may bear the brunt of those cravings when patients demand the same services that *The Human Factor's* Dr McMurty gives. Are TV doctors setting standards to remind and stimulate physicians to emulate them? As the King of Siam was wont to say: "It is a puzzlement."

Data to help resolve these issues vary due to individual subjectivity. Information about patient satisfaction with medical care provides clues. Although generally satisfied, people complain that they wait too long to see the doctor, that the doctor does not take the time to explain everything, and that their medical bills are too high and incomprehensible. Where is the happy medium? Can a physician bill for time spent talking to a patient? Management experts offer methods for meeting the therapeutic obligations of practicing physicians. Allied health care personnel can handle some patient education. If so, can those services be charged to the patient?

TV producers employ physician experts to ensure accuracy regarding equipment, drugs, terminology, and diagnostic procedures. Those experts generally know what is happening in the health care system. Why, then, does the TV doctor drama emerge from the cocoon of improbability to flit about in the fairyland of a desirable health care environment? We can only assume that dramatic considerations are given precedence over reality. TV ratings would most likely support producers' claims that the TV doctor show is pure drama, not real life. Perhaps producers should present a disclaimer before each show announcing that the viewers' doctors may not be like the ones they see on TV.

Efforts to combine curing with caring in the US health care system should definitely continue. At times, caring may be a powerful curative placebo. Certainly, the practice of medicine, notwithstanding the miracle drugs and the sophisticated technology, is still an art as well as a science.

Hippocrates[4(p192)] aptly identified the human factor demands of medical education and medical practice in his *Aphorisms:*

> Life is short, and the Art long; the occasion fleeting; experience fallacious, and judgment difficult. The physician must not only be prepared to do what is right himself, but also make the patient, the attendants, and externals co-operate.

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1. Peabody FW: The care of the patient. *JAMA* 1927; 88;877–882.
2. Spiegel AD, Backhaut BH: *Curing and Caring: A Review of the Factors Affecting the Quality and Acceptability of Health Care.* New York, SP Medical and Scientific Books, 1980.
3. Spiegel AD, Kavaler F: *Cost Containment and DRGs: A Guide to Prospective Payment.* Baltimore, National Health Publications, 1986.
4. Adams F: *The Genuine Works of Hippocrates, Vol 2.* New York, William Wood & Company, 1886.

Cardiovascular risk factors in Native American children

Ann S. Botash, md; RaeEllen W. Kavey, md; Nancy Emm, lpn; Diane Jones

ABSTRACT. Information concerning cardiovascular disease risk factors in Native American children is limited. In adult Native American populations, cardiovascular disease risk factors of non-insulin dependent diabetes mellitus, obesity, and cigarette smoking are prevalent, and recent reports indicate mortality caused by cardiovascular disease has dramatically increased. In a screening program at the Onondaga Nation School, six cardiovascular disease risk factors were evaluated. Of 95 school children, 55 representing 39 interrelated families, participated. Family histories were positive for diabetes mellitus in 72%, for cardiovascular disease in 54%, and for passive smoking in 90% of families. Physical examinations of the children revealed obesity (weight/height > 90th percentile) in 42% and hypertension (systolic or diastolic blood pressure/height > 95th percentile) in 22%. Fingerstick cholesterol levels (Reflotron system) were greater than 170 mg/dL in 25%. Overall, 85% of participants had three or more risk factors for cardiovascular disease. The study results indicate that Onondaga Native American children are at significant risk for cardiovascular disease; programs for identification and modification of cardiovascular disease risk factors are urgently needed.

(NY State J Med 1992; 92:378–381)

Cardiovascular disease (CVD) is the leading cause of disability and death in the United States. Native Americans (NAs) have long been believed to be at low risk for CVD.[1–4] However, recent studies report a significant increase in NA mortality rates from CVD.[5–7] Few studies have examined specific CVD risk factors in NA children,[8–10] and none have described the prevalence of these risk factors in NA children. There are no published studies of CVD risk factors among NAs in Onondaga County, NY. As a primary care provider at the Onondaga Nation Pediatric Clinic, one of the authors (ASB) was impressed by the apparent high prevalence of CVD risk factors in this community. Review of the mortality data of NAs in Onondaga County (1981–1986) revealed that, in this NA population, CVD is the leading cause of death. This study was designed to assess the prevalence of CVD risk factors among the Onondaga Nation NA school children.

Patients and Methods

All 95 children who attended the Onondaga Nation Elementary School (Nedrow, NY) in the spring of 1989 were invited to participate in a cardiovascular disease risk screening program. The Onondaga Nation Territory, located south of Syracuse, NY, had an estimated population of approximately 880 in 1989, consisting primarily of Onondaga and Mohawk NA. These are two nations of the Six Nation Iroquois Confederacy. The project was approved by the Onondaga Nation Council of Chiefs, and parental consent was obtained.

Six CVD risk factors were assessed: obesity, hypertension, hypercholesterolemia, passive smoking, family history of non-insulin-dependent diabetes mellitus (DM), and family history of CVD.

Heights to the nearest one-half inch and weights to the nearest one-half pound were measured using a standard scale (Health-O-Meter Continental). Weight for height percentiles were determined using National Health Survey Data Vital and Health Statistics.[11,12] Obesity was defined as weight for height greater than or equal to the 90th percentile.

Using the standard method, each child seated with his or her right arm antecubital fossa raised to heart level, manual sphygmomanometry was performed. With deflation of the cuff, the first and fourth Karotkoff sounds were noted, and readings were recorded to the nearest 2 mm Hg. Systolic and diastolic blood pressure percentiles for height were determined using established grids developed from pediatric population surveys (5–18 yrs).[13] Children with systolic or diastolic blood pressures greater than the 95th percentile had repeat measurements obtained at school or at the Onondaga Nation Pediatric Clinic by one of the authors (ASB). Their medical records were reviewed to ascertain previous or subsequent high blood pressure readings.

Using a Reflotron (Boehringer Mannheim Diagnostics, Indianapolis, IN), nonfasting serum cholesterol levels were obtained. Using manufacturer supplied calibration materials, the instrument was calibrated and operated according to the manufacturer's recommendations. Control values were consistently obtained. All testing was done by one of the authors (NE) who had greater than 100 hours of previous experience with this method. Cholesterol percentiles for age were determined using a pediatric reference grid generated by the Bogalusa Heart Study.[14] High values were reported as those greater than the 75th percentile.

By telephone interviews with parents, one author (ASB) obtained family histories concerning the health status of first and second degree relatives (siblings, parents, aunts, uncles, grandparents). Positive responses to questions regarding histories of stroke, acute myocardial infarction, or sudden death before 60 years of age, bypass surgery, hypertension, medications for blood pressure, heart disease, or angina were considered positive histories for CVD. In a similar manner, histories of diabetes mellitus (DM) and smoking were obtained. If a parent could not remember or did not know if a family member had CVD or DM, this was recorded as negative. When any household member smoked any number of cigarettes, this was reported as a positive history of passive tobacco smoking. The smoking habits of the children were also addressed.

Results of the physical examination and cholesterol test were distributed to the children to take home. All high risk factor findings were recorded in the child's medical record at the Onondaga Nation Pediatric Clinic. Mothers or fathers

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were subsequently contacted and the results and need for follow-up studies were discussed. When appropriate, referrals to the Onondaga Nation Pediatric Clinic Nutritionist (registered dietician) were made and the American Heart Association prudent diet was recommended.

RESULTS

A total of 55 of the 95 children (58%) who were enrolled at the Onondaga Nation Elementary School were evaluated. Thirty-nine nuclear families were represented. These families were often related as first or more distant cousins. There were 22 males (40%) and 33 females (60%) with an age range of 5–16 years. The mean age was 10 ± 3 with 44 (80%) children who were 12 years old or younger. All of these children lived on Onondaga Nation territory and received health care at the Onondaga Nation Pediatric Clinic.

Results are shown in Table I. Obesity was evident in 23 (42%) children. The mean weight/height ratio percentile was 73 ± 22%. Hypertension was evident in 12 (22%) children (Fig 1). Ten of these 12 hypertensive children had weight/height ratios greater than the 95th percentile. Chart reviews and repeat blood pressure measurements of the 12 hypertensive children revealed that six children had at least three blood pressure measurements greater than the 95th percentile for height.

Cholesterol observations are illustrated in Figure 2. Fourteen children (25%) had cholesterol levels greater than the 75th percentile for Caucasians. The mean serum cholesterol was 150 ± 36 mg/dL. More than half (65%) had a serum cholesterol below the 50th percentile. All 14 children with high cholesterol levels were referred to the Onondaga Nation Clinic for nutritional counselling and determination of central fasting total cholesterol, triglyceride, high-density lipoprotein, and low-density lipoprotein levels. To date, six of the 14 children with high cholesterol levels have had their cholesterol tests repeated and, in five of them, the total serum cholesterol remained greater than the 75th percentile. Repeat cholesterol counts were obtained in a total of ten children; all were within 20% of the first determination.

Family histories of DM and CVD in first and second degree relatives, as well as household tobacco use are shown in Table II. There was a history of passive smoking in 35 families (90%). All of the children denied tobacco use. Twenty-eight families (72%) had a positive history for DM, and 21 families (54%) had a positive history for CVD. In individual children, 40 (72%) had a family history of DM, and 30 (55%) had a family history of CVD. Twenty families (51%) had positive family histories for both CVD and DM. Twenty-nine families (74%) had a positive history for either DM or CVD. Only three children (6%), from two families, had a positive family history for CVD but not for DM.

Excluding male gender as a risk factor, 85% of subjects had three or more risk factors present (Fig 3).

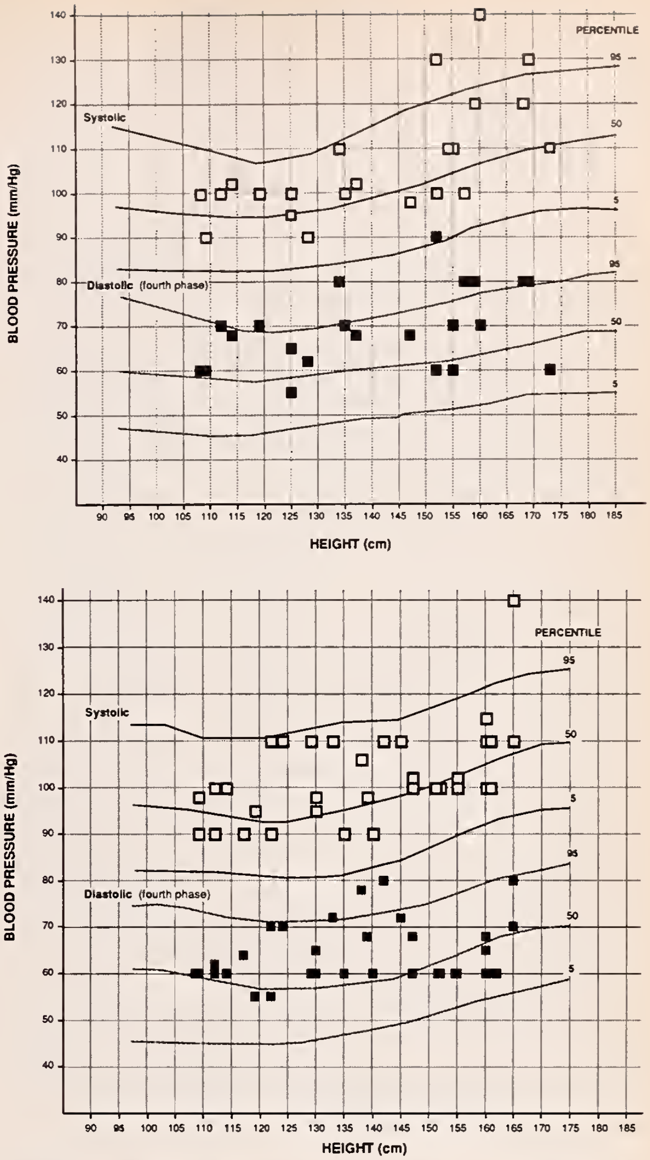

FIGURE 1. Male (top) and female (bottom) systolic and diastolic blood pressure percentiles of NA subjects. (Percentile references developed by Berenson GS, et al.[16])

DISCUSSION

To our knowledge, this study is the first to examine the prevalence of CVD risk factors in a population of NA children. Preliminary results of this survey have been previously published.[15] The need for CVD risk factor identification in childhood has been addressed by others.[16–18] In children, studies have shown an association of postmortem atherosclerotic plaques with antemortem hypercholesterolemia and hypertension.[16] Since childhood percentiles for hypertension, hypercholesterolemia, and obesity are known to track to the adult years,[17,18] early identification of CVD risk in Onondaga NAs, development and implementation of intervention measures and studies to determine the impact of these measures are needed. Since CVD is the leading cause of death in this NA population, our finding of a high prevalence of multiple CVD risk factors in the Onondaga NA school children is very significant.

Among NA populations there are many genetic, cultural, geographic, and environmental variables affecting CVD risk and limiting the development and interpretation of broad epidemiologic studies. Ethnicity and a family history of CVD are significant elements in the assessment of CVD

TABLE I. Individual Risk Factors

Risk Factor	Male $n = 22$	%	Female $n = 33$	%	Total $N = 55$	%
Cholesterol ≥ 170 mg/dL	4	18	10	30	14	25
Wt/Ht ≥ 90th %	10	45	13	39	23	42
Bp/Ht > 95th %	8	36	4	12	12	22

TABLE II. Family History of Risk Factors

Risk Factor	Families N = 39	%
Diabetes Mellitus	28	72
Cardiovascular Disease	21	54
Smoking	35	90

risk,[19-22] and a genetic factor may be important in the Onondaga Nation. Some study participants have Mohawk ancestry, a group with a known high prevalence of DM and CVD.[8] The prevalence of NA CVD mortality has gradually increased, and specific NA communities with high prevalences of CVD have reported high prevalences of DM, hypertension, obesity, and cigarette smoking.[6-8,23] Early studies of the Pima NAs of southern Arizona report a high prevalence of obesity and DM, yet a low prevalence of coronary heart disease.[2,5] A study of Indian Health Service data[3] (1981) suggested that NAs had a decreased risk for CVD mortality. However, recent studies of the Navajo[7] and of NAs in Western South Dakota[6] (Sioux) report higher rates of morbidity and mortality from acute myocardial infarction; the leading causes of death for Chippewa NAs (Minneapolis, MN) are heart disease and stroke.[4]

The actual prevalence of CVD and CVD risk factors within the Onondaga community are impossible to assess. The census of the Onondaga Nation contains approximate information, and health care is delivered sporadically by multiple providers. Onondaga NA mortality data indicating that CVD is the leading cause of death, and our finding of a high prevalence of a family history of DM and CVD, suggest that DM and CVD are highly prevalent within the community. Because of familial interrelationships, this implied prevalence may be overestimated.

The study population screened here includes fewer adolescents than younger children. The high prevalences of family histories for CVD and DM does not seem to be due to excessive numbers of older children with older parents or relatives having higher age associated risks.

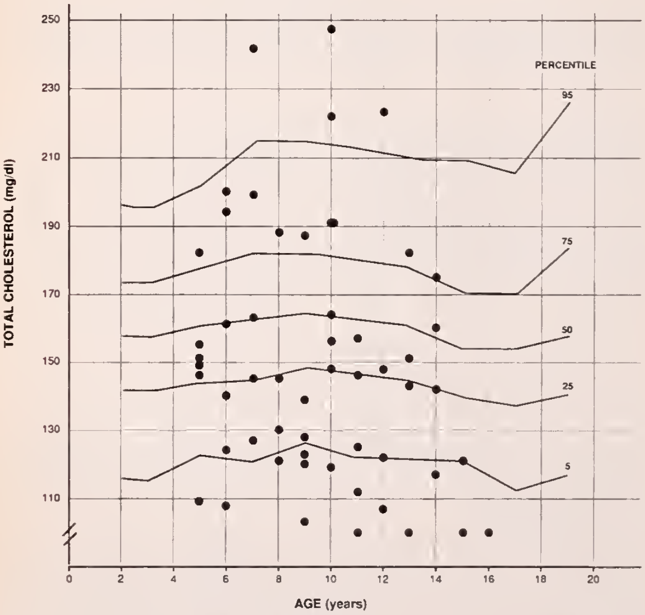

FIGURE 2. Serum cholesterol percentiles of NA subjects. Increased risk for CVD is defined as total serum cholesterol above the 75th percentile (14 children). (Percentile references developed by Cresanta JL, et al.[17])

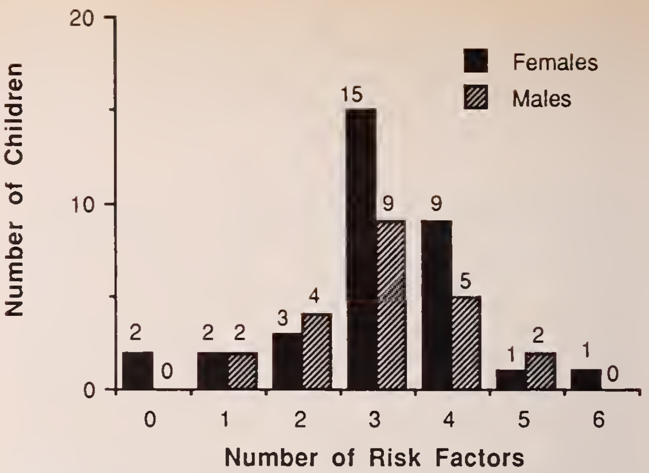

FIGURE 3. CVD risk factors: family history of DM, family history of CVD, passive smoking, high cholesterol, hypertension, and obesity.

Differences in the methods for obtaining family histories and in population demographics cause difficulty in comparisons of studies describing the prevalence of positive family histories for CVD, DM, or household smoking. In spite of these differences, some comparisons can be made. In a recent study, approximately 65% of Caucasian children had a family history of CVD or DM.[20] In comparison, approximately three fourths of the Onondaga NA children (78%) have either a family history of CVD or DM. In a population of black children, 36% of the children had a family history of DM;[21] the frequency of a family histories of DM for Onondaga NA children is much higher (72%).

Our finding that 42% of the children have weight/height ratios greater than the 95th percentile for Caucasian standards is significant. Obesity is not only a risk factor for CVD, but is also implicated as a cause of non-insulin-dependent diabetes mellitus. Other studies of NA children indicate a propensity for childhood weight/height percentile ratios greater than the 50th percentile.[9] The known high prevalence of obesity in other adult NA populations suggests that an inherent risk for obesity is present in NAs. In Caucasian children, obesity may be controlled, and there may be a significant reduction in CVD risk with combined diet and exercise programs. NA children could also certainly benefit from appropriate nutritional counselling and exercise programs.

Similar to the findings of early Pima studies, in which Pima children had cholesterol levels 20–30 mg/dL lower than Caucasian populations,[10] our study reports the interesting finding of a mean serum cholesterol below the Caucasian reference for the 50th percentile. Although 25% of the children have serum cholesterol levels greater than the 75th percentile, more than half of the children have a total cholesterol below the 50th percentile. Diet and heredity are thought to play a role in cholesterol levels. The Pima diet was considered to be a low cholesterol diet; the Onondaga NA diet is similar to the usual US diet. Our data suggest that, although lower serum cholesterol levels may be inherent, some NA children may be hypercholesterolemic. Longitudinal studies of serum cholesterol levels in large NA populations are indicated.

Our observation of hypertension in 22% of the Onondaga children is consistent with other reports. Studies of NA adults report a high prevalence of hypertension, often as high as 13%.[4,7,23] In Mohawk NAs in Quebec, 71% with diabetes had hypertension and 41% without diabetes had hypertension.[8] Since 10 of the 12 hypertensive Onondaga children were obese, the need for weight control intervention measures is again evident.

Passive smoking has recently become of interest as a risk factor for CVD.[24] Boys from smoking families have been shown to have thicker heart walls, stiffer aortas, and lower serum high-density lipoprotein levels.[24] Surveys have approximated that 70% of children in the United States live in homes where there is at least one adult smoker.[25] Many factors may influence smoking habits in NA populations, including cultural acceptance, association with alcohol use, and the tax-free status of tobacco on NA territory. Exposure to smoking by family members is believed to be the most likely factor influencing later adolescent or adult behavior. Approximately half of the Canadian Mohawk population smoke cigarettes.[8] In the present study, all but four of the households have at least one family member who smokes cigarettes. The frequency of exposure to cigarette smoking in the Onondaga NA household is extremely high.

In summary, Onondaga Native American school children have many risk factors for cardiovascular disease. The high prevalence of family histories of DM and CVD imply that this population may have an inherited predisposition to DM and CVD, and that routine risk factor surveillance during childhood is necessary. The high prevalence of modifiable risk factors, including obesity and passive smoking, indicate the need for improved intervention programs. This school-based screening serves as a foundation for future education and prevention curricula. Physicians who provide primary care to NA children should be aware of the possible high risk for CVD and should include an evaluation for CVD risk factors and appropriate parental and child education.

Acknowledgments. The authors wish to thank the Onondaga Nation Council of Chiefs for their support, and the people of the Onondaga Nation for their interest and participation.

REFERENCES

1. Ingelfinger JA, Bennett PH, Liebow IM, et al: Coronary heart disease in the Pima Indians: Electrocardiographic findings and postmortem evidence of myocardial infarction in a population with a high prevalence of diabetes mellitus. *Diabetes* 1976; 25:561–565.
2. Gillum RF: Ischemic heart disease mortality in American Indians, United States, 1969–1971 and 1979–1981. *Am Heart J* 1988; 115(5):1141–1144.
3. Gillum RF, Gillum BS, Smith N: Cardiovascular risk factors among urban American Indians: Blood pressure, serum lipids, smoking, diabetes, health knowledge and behavior. *Am Heart J* 1984; 107(4):765–776.
4. Howard BV, Davis MP, Pettitt DJ, et al: Plasma and lipoprotein cholesterol and triglyceride concentrations in the Pima Indians: Distributions differing from those of Caucasians. *Circulation* 1983; 68:714–724.
5. Hrabovsky SL, Welty TK, Coulehan JL: Acute myocardial infarction and sudden death in Sioux Indians. *West J Med* 1989: 150:420–422.
6. Klain M, Coulehan JL, Arena VC, et al: More frequent diagnosis of acute myocardial infarction among Navajo Indians. *Am J Public Health* 1988; 78: 1351–1352.
7. Macaulay AC, Montour LT, Adelson N: Prevalence of diabetic and atherosclerotic complications among Mohawk Indians of Kahnawake, PQ. *Can Med Assoc J* 1988; 139:221–224.
8. Indians and Inuit Health Committee, Canadian Pediatric Society. Growth charts for Indian and Inuit children. *Can Med Assoc J* 1987; 136:118–119.
9. Savage PJ, Hamman RF, Bartha G, et al: Serum cholesterol levels in American (Pima) Indian children and adolescents. *Pediatrics* 1976; 58:274–282.
10. Gillum RF, Prineas RJ, Palta M, et al: Blood pressure of urban Native American school children. *Hypertension* 1980; 2:744–749.
11. Hamill PV, Drizd TA, Johnson CL, et al: Physical growth: National Center for Health Statistics Percentiles. *Am J Clin Nutr* 1979; 32:607–629.
12. National Center for Health Statistics. Data from the National Health Survey, 1973. Vital and Health Statistics. Height and weight of youths 12–17 years, United States. Series 11. No. 124. (HSM)73-1606. Rockville, MD, Health Services and Mental Health Administration, 1973.
13. Berenson GS, Cresanta JL, Webber LS: High blood pressure in the young. *Ann Rev Med* 1984; 35:535–560.
14. Cresanta JL, Srinivasan SR, Webber LS, et al: Serum lipid and lipoprotein cholesterol grids for cardiovascular risk screening of children. *Amer J Dis Child* 1984; 138:379–387.
15. Botash AS, Kavey RE, Emm N, et al: Hyperlipidemia in childhood and the development of atherosclerosis: Cardiovascular risk factors in Native American children. *Ann NY Acad Sci* 1991; 623(poster):416–418.
16. Newman WP, Freedman DS, Voor AW, et al: Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis. *N Engl J Med* 1986; 314:138–144.
17. Berenson GS, Srinivason SR, Hunter SM, et al: Risk factors in early life as predictors of adult heart disease: The Bogalusa heart study. *Am J Med Sci* 1989; 298(3):141–151.
18. Lauer RM, Lee J, Clarke WR: Factors affecting the relationship between childhood and adult cholesterol levels: The Muscatine Study. *Pediatrics* 1988; 82:309–318.
19. Blonde CV, Webber LS, Foster TA, et al: Parental history and cardiovascular disease risk factor variables in children. *Prev Med* 1981; 10:25–37.
20. Griffin TC, Christoffel KK, Binns HJ, et al: Family history evaluation as a predictive screen for childhood hypercholesterolemia. *Pediatric* 1989; 84:365–373.
21. Durant RH, Kinder CW, Jay S, et al: The influence of a family history of CHD risk factors on serum lipoprotein levels in black children and adolescents. *J Adol Health Care* 1982; 3(2):75–81.
22. Srinivasan SR, Freedman DS, Webber LS, et al: Black-white differences in cholesterol levels of serum high-density lipoprotein subclasses among children: The Bogalusa Heart Study. *Circulation* 1987: 76:272–279.
23. McIntyre L, Shah CP: Prevalence of hypertension, obesity and smoking in three Indian communities in Northwestern Ontario. *Can Med Assoc J* 1986; 134:345–349.
24. Land D: Smoking by parents increases risk of heart disease in boys. *Cardiovasc Res Rep* 1988; (Spring):5
25. Fielding JE, Phenow KJ: Health effects of involuntary smoking. *N Engl J Med* 1988; 319:1452–1460.

The persistent vegetative state: An analysis of clinical correlates and costs

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ABSTRACT. A review was compiled of 23 patients in the persistent vegetative state; a condition that developed while the patients were in an acute care hospital. Before the onset of the persistent vegetative state, eight patients had had dementia, 11 were functionally dependent, and seven had neurologic disorders that gradually led to the persistent vegetative state. For patients in the persistent vegetative state, three types of mechanical support—respirators, nasogastric or gastrotomy tubes, and intravenous lines—were utilized. All three were necessary in 43% of patients, two types in 52%, and one type in 4%. All patients in the persistent vegetative state required at least one type of mechanical assistance. Their course was complicated in all cases by incontinence, and in the majority, by decubiti,

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pneumonia, and urinary tract infection. Only one patient improved enough to be able to say a few words. These patients required active medical care and invasive procedures that were costly but futile. The hospital bills obtained for 13 patients averaged $170,000, and the length of stay for all patients averaged 197 days, the equivalent to a total number of bed-days of 12.5 bed-years. The poor outcomes, requirements for mechanical support, and frequency of complications—especially when neurologic impairments were present before the onset of the persistent vegetative state—should be considered when evaluating the cost in hospital bills and bed occupancy.

(NY State J Med 1992; 92:381–387)

The persistent vegetative state (PVS), a term introduced by Jennett and Plum[1] in 1972, describes a state of severe brain damage, present for longer than three months, that is characterized by lack of recognizable mental function, mutism, and responses limited to primitive postural and reflex limb movements. In 1989, the American Academy of Neurology (AAN) described the PVS as a state in which patients have "eyes-open," permanent unconsciousness with periods of wakefulness, but no awareness of themselves or their environment.[2] They also have no voluntary actions or behavioral response over an extended period of time; however, they may have persistent primitive reflexes and vegetative functions, spontaneous breathing, chewing, and swallowing. Other organizations have adopted similar definitions.[3] The implication of the diagnosis is that patients have little or no likelihood of recovery of mentation or independent living.

Most patients in the PVS have had preceding brain injury, such as cardiac arrest or trauma associated with anoxia, that caused coma.[1,4] Functional recovery, so rarely documented that it is virtually unique,[5] has been estimated to occur in fewer than one in 1,000 patients in whom the PVS has been present for at least 12 months after head injury, or three months after cardiac arrest and hypoxic-ischemic cerebral injury.[3] For example, in one study, only two of 36 PVS patients "regained any psychologic self-awareness," and that limited degree of recovery was evident within three months of the neurologic insult.[6] Rather, patients will linger for months or even years without cognitive recovery.[7,8]

The dismal prognosis has prompted an evaluation of the medical care and routine sustenance of people in the PVS. The AAN has indicated that if it can be determined with a high level of certainty that the patient in the PVS will never improve, the artificial provision of nutrition and hydration may be discontinued.[2] Discontinuing mechanical feeding of individual patients in the PVS has also been permitted by the courts in certain circumstances.[9]

It would be helpful if the quality of life and cost to the public of the PVS could be clarified before decisions regarding maintenance of mechanical support are made by the medical profession, relatives, and individuals. We studied the antecedents of the condition, the support required by patients, complications during hospitalization, and outcomes in patients with the PVS in an urban, acute care hospital. The study also uses bed-days and hospital bills to estimate the cost of PVS.

Patients and Methods

Charts of comatose, brain-injured patients hospitalized for at least one month at Montefiore Medical Center (MMC) from January 1, 1978, to January 1, 1991, were identified by computer review. This span was chosen because an improvement in the computerized record system was installed that permitted the identification of such patients. The charts of patients who fulfilled criteria of the AAN for the PVS were analyzed.[2] All medical charts were reviewed by a neurologist. The review included a detailed assessment of the patients' pre-hospital social and demographic profile, medical conditions, living arrangements, and functional status. The etiology of the PVS and the hospital course were reviewed. Nutritional and respiratory support as well as medical complications were noted. Finally, status at discharge, length of stay, and if possible, the hospital bill and ultimate outcome were noted.

Results

We identified 23 patients in whom the PVS had developed. Patients ranged in age from 40 to 97 years; 19 patients were 65 years or older, of whom eight were 80 years of age or older. Ten patients were males, and ten had cardiovascular disease.

The patients' pre-admission status was assessed in three areas: presence of pre-existing medical conditions (particularly dementia), living arrangements, and eating ability (Table I). Before admission, eight patients had dementia from Alzheimer's disease or multiple cerebral infarctions. Although 12 patients had been living independently, the other 11 either needed help at home or were nursing home residents. Regardless of their residence, ten patients required assistance in eating (Table I). The cause of the PVS was determined to be intracranial hemorrhage in nine patients, anoxia in five, and cerebrovascular accidents in four (Table II). Degenerative neurologic illnesses, neoplasms, and metabolic insults also caused the PVS.

Although cases of PVS typically evolved from coma, seven patients gradually slipped into a PVS over a period of weeks without passing through coma. The causes for this were intracerebral hemorrhage (two patients); ischemic cerebrovascular accidents (two patients); and multiple sclerosis, cerebral metastases, and ALS-dementia complex (one patient each) (Table II). Before admission, one of the patients had mild

TABLE I. Pre-admission Status of Twenty-Three Patients in the Persistent Vegetative State

	No.	(%)
Pre-admission medical conditions		
Cardiovascular disease	10	(43)
Hypertension	9	(39)
Diabetes	4	(17)
Cancer	4	(17)
Alcohol abuse	1	(4)
Pre-admission dementia		
Severity		
Mild	2	(9)
Moderate	2	(9)
Severe	4	(17)
Total	8	(35)
Cause of Dementia		
Alzheimer's disease	3	(13)
Cerebrovascular accidents	5	(22)
Pre-admission living situation		
Home independently	12	(52)
Home with help	7	(30)
Health related facility	0	(0)
Nursing home	4	(17)
Pre-admission eating status		
Ate independently	13	(57)
Could not eat independently	5	(22)
Needed help	4	(17)
Gastrotomy	1	(4)

TABLE II. Etiology and Onset of the Persistent Vegetative State

	No. Patients (%)	Abrupt Onset (%)*	Gradual Onset (%)*
Part A			
Etiology			
Intracerebral hemorrhage	6 (26)	4 (67)	2 (33)
Subdural hematoma	3 (13)	3 (100)	0 (0)
Anoxia	5 (22)	5 (100)	0 (0)
Ischemic cerebrovascular accidents	4 (17)	2 (50)	2 (50)
Multiple sclerosis	1 (4)	0 (0)	1 (100)
Cerebral metastases	1 (4)	0 (0)	1 (100)
Sepsis	1 (4)	1 (100)	0 (0)
Heat stroke	1 (4)	1 (100)	0 (0)
ALS-dementia complex	1 (4)	0 (0)	1 (100)
Total	23 (100)	16 (70)	7 (30)
Part B			
Seven patients who slipped into the PVS (average age: 72 years)			
Etiologies			
Intracerebral hemorrhage	2		
Cerebral infarctions	2		
Multiple sclerosis	1		
Cerebral metastases	1		
ALS-dementia complex	1		
Preexisting conditions			
Dementia severe	2		
Dementia mild	1		
Cerebrovascular accidents	2		
Needed help at home	3		
Number of mechanical supports (nutritional, respiratory, intravenous)			
Three required	2		
Two required	4		
One required	1		
Outcome			
Died in hospital	4		
Transferred to nursing home	3		
Bed-days			
Range, 28 to 458			
Average, 150			

*Percentage of cases of that etiology.

dementia, and two had severe dementia. Those two, and another patient, had to have help at home. The number of mechanical supports, complications, and outcome of this group were similar to the group as a whole.

Whether or not patients entered the PVS from coma or slipped into it, once in the PVS, they all required the mechanical provision of nutrition, respiration, or intravenously administered substances (Table III). For nutrition, all 23 patients required either a nasogastric tube or a surgically placed gastrotomy tube (five patients). Seventeen patients (73%) required mechanical respiratory support with either a respirator, a tracheostomy, or both. Prolonged dependence on a respirator or tracheostomy tube was necessary for pulmonary toilet, maintaince of a patent airway, or proper ventilation more than for breathing. An intravenous line (IV) was required in 15 patients for the administration of fluids, nutrition, antibiotics, or other medications. All but one PVS patient who required an IV, needed mechanical support for feeding, respiration, or both.

All patients in the PVS were incontinent, and most of them also suffered from decubiti (78%), pneumonia (61%), and urinary tract infections (57%). In addition, numerous other complications occurred as a consequence of the bed-bound, debilitated state, or the multiple medical procedures and interventions required to support these patients; these complications included sepsis (22%), seizures (17%), gastrointestinal bleeding (13%), and renal failures (9%).

TABLE III. Mechanical Support of Vital Functions During the Persistent Vegetative State

Patients	No.	(%)
Nutritional support		
Nasogastric tube	18	(78)
Gastrotomy tube	5	(22)
Intravenous lines	15	(65)
Intravenous lines only	0	(0)
Oral intake	0	(0)
Respiratory support	No.	(%)
Respiratory*	10	(43)
Tracheostomy†	7	(30)
Total tracheostomy	12	(52)
No support	6	(26)
How many patients needed multiple mechanical types of support: respirator, nasogastric or gastrotomy tube, or IV line?		
Three types	10	(43)
Two types	12	(52)
One type	1	(4)

*With endotracheal tube or tracheostomy.

†But no respirator (tracheostomy had been performed for initial illness, for pulmonary toilet, or to maintain patent airway).

Although survival and placement varied, the patients' outcomes were uniformly poor. Sixteen patients died in the hospital. Of the other seven patients, six remained in the PVS in a nursing home and one improved to have discernable mental function. However, this patient, who survived a massive intracerebral hemorrhage complicated by hydrocephalus, remained confined to a nursing home where he had a moderate to severe neurologic deficit that left him bedridden. Although able to comprehend simple requests and say a few words, he was disoriented, unaware of his situation, and unable to converse. No patient recovered sufficiently to live independently. Five of the surviving patients were among the seven youngest. Of the seven patients transferred to nursing homes, two died six months and three years later. All deaths resulted from systemic problems such as infection.

Treatment of the PVS was costly for the hospital and for the health care system. The average stay in this hospital was 197 days, the median 110 days. Although the majority of patients remained hospitalized between 50 and 200 days, five were hospitalized longer than one year, and one for over three years. Hospital bills obtained for 13 patients ranged from $30,000 to $427,000, and averaged $170,000, with a median of $98,000. Four patients' bills were each greater than $300,000.

DISCUSSION

Many previous reports describe the etiology and predictive factors of the PVS.[3,4,6,7,10–12] Almost all reported cases of the PVS followed massive anoxic, traumatic, or vascular brain injuries. It is not generally recognized that patients may slowly develop the PVS as a result of progressive structural or degenerative disorders, such as Alzheimer's disease.[3,7,13,14] Although Higashi et al[7] and Walshe and Leonard[13] included cases of degenerative illnesses, such as Creutzfeldt-Jacob syndrome, subacute sclerosing panencephalitis, and Pick's disease, the PVS patients with these illnesses usually have not been discussed because the onset has not been dramatic and the continued deterioration is considered part of the underlying, progressive illness.

Tresch et al[14] described 51 PVS patients who comprised 3.2% of the residents of four nursing homes.[14] The cause of the PVS in these patients was said to be dementia in 25%. In our series, a large proportion of patients (30%) had gradually slipped into the PVS because of structural lesions, such as cerebral infarctions and metastatic tumors; or degenerative illnesses, such as multiple sclerosis and ALS-dementia complex. In these patients, where there was no single neurologic insult, the subsequent course, complications, outcome, and cost were similar to the cases that followed a dramatic cerebral insult.

Many of the patients presented in this report had substantial impairments or limitations before the neurologic injury that caused the PVS. Before their admission to the hospital, these patients had not been independent: 35% had some degree of dementia, 48% required partial or complete help, and 43% needed help with eating. In addition, 35% of the patients were 80 years of age or older. These patients, and perhaps equal proportions in other medical centers, have a limited prognosis. Whatever their prehospital status, all of the patients in the PVS required intravenous lines or other mechanical assistance of vital functions: nutrition, respiration, and intravenous fluids, or medications. In addition, surgery was required to install a gastrostomy tube in 22% and a tracheostomy in 30%. Incontinence was present in all cases, and decubiti, pneumonia (61%), and urinary tract infection (57%) complicated the course of the majority of cases and gastrointestinal bleeding occurred in many (13%).

Other series report similar requirements for mechanical devices and the treatment of medical complications. Higashi et al[7] reported that 15% of PVS patients needed a gastrotomy and 30% a tracheostomy; however, they found slightly lower but still substantial rates for pneumonia (44%) and urinary tract infections (42%) and also for gastrointestinal bleeding (3%). Moreover, Tresch et al[14] reported a uniform need for mechanical devices for feeding: 66% of the patients described required a nasogastric tube and 33% required gastrotomy or jejunostomy tube.[14] In addition, they reported that 75% of the patients had indwelling urinary catheters, 65% had decubiti that required specific treatment, and most illustrative of the need for active medical care, 61% had to be transferred to an acute care hospital.

The outcome in many cases in our series seems to be greatly influenced by the patient's age, preadmission mental and functional impairments, and the cerebral injury that caused the PVS. Although the PVS patients presented here survived for months or years, only one, who still remains in a nursing home, recovered a modicum of mental function. Patients in the PVS rarely achieve a functional recovery.[5] Walshe and Leonard,[13] who reported a series that was comparable to ours, found that of 29 PVS patients, none improved and ten died.[13] One case of recovery from PVS has been challenged because no neurologist examined the patient and no electroencephalogram was ever obtained.[15] Groswasser and Sazbon reported that 50 of 72 patients (69%) with post-traumatic unawareness—not exactly the PVS—became socially integrated.[8,12] Higashi et al[7] suggested that the possibility for recovery exists for at least two months but probably not much longer. They found that 7% of their PVS patients recovered but that only 3% had become "comprehending and communicative." Those truly in a PVS longer than three months have less than one in 1,000 chance of a functional recovery, and that chance may be restricted to patients younger than 40 years.[3]

Caring for PVS patients entails continual, active, and invasive treatment and the use of mechanical devices. Surgical interventions are often required to feed, ventilate, and otherwise sustain these patients. At the same time, the chance of recovering self-awareness, much less an independent life, is miniscule. Being in the PVS does not entail, as some of the public may fantasize, living in either a state of suspended animation or being a "sleeping beauty."

For the hospital, patients in the PVS consume numerous bed-days as well as nursing and physician services. Five patients remained in our acute care hospital for more than a year. In the series of Higashi et al,[7] 25% of 110 patients had a vegetative existence for more than three years, and four lived more than ten years. Walshe and Leonard[13] found that seven patients with the PVS from head trauma survived for an average of 5.7 years. For our series of 23 patients, the average length of acute care hospitalization was 197 days (median, 110 days), yielding a total of 12.5 bed-years. The patient's average bill in this hospital was $170,000 (median, $98,000). The costs must be viewed against the limited quality of life, numerous medical complications, and almost uniformly poor outcome.

REFERENCES

1. Jennett B, Plum F: Persistent vegetative state after brain damage: A syndrome in search of a name. *Lancet* 1972; 1:734–737.
2. Executive Board, American Academy of Neurology. Position of the American Academy of Neurology on certain aspects of the care and management of the persistent vegetative state patient. *Neurology* 1989; 39:125–126.
3. Council on Scientific Affairs and Council on Ethical and Judicial Affairs. Persistent vegetative state and the decision to withdraw or withhold life support. *JAMA* 1990; 263:426–430.
4. Levy DE, Sidtis JJ, Rottenberg DA, et al: Differences in cerebral blood flow and glucose utilization in vegetative versus locked-in patients. *Ann Neurol* 1987; 22:673–682.
5. Rosenberg GA, Johnson SF, Brenner RP: Recovery of cognition after prolonged vegetative state. *Ann Neurol* 1977; 2:167–168.
6. Levy DE, Knill-Jones RP, Plum F: The vegetative state and its prognosis following nontraumatic coma. *Ann NY Acad Sci* 1978; 315:293–306.
7. Higashi K, Sakata Y, Hatano M, et al: Epidemiological studies on patients with a persistent vegetative state. *J Neurol Neurosurg Psychiatry* 1977; 40:876–885.
8. Groswasser Z, Sazbon L: Outcome in 134 patients with prolonged posttraumatic unawareness. Part 2: Functional outcome of 72 patients recovering consciousness. *J Neurosurg* 1990; 72:81–84.
9. Cruzan v. Director, Missouri Dept. of Health, 110 S. Ct. 2841 (1990).
10. Ingvar DH, Brun A, Johansson L, et al: Survival after severe cerebral anoxia with destruction of the cerebral cortex: the apallic syndrome. *Ann NY Acad Sci* 1978; 315:184–214.
11. Relkin NR, Petito CK, Plum F: Coma and the vegetative state associated with thalamic injury after cardiac arrest. *Ann Neurol* 1990; 28:221–222.
12. Sazbon L, Groswasser Z: Outcome in 134 patients with prolonged posttraumatic unawareness. Part 1: Parameters determining late recovery of consciousness. *J Neurosurg* 1990; 72:75–80.
13. Walshe TM, Leonard C: Persistent vegetative state: Extension of the syndrome to include chronic disorders. *Arch Neurol* 1985; 42:1045–1047.
14. Tresch DD, Sims FH, Duthie EH, et al: Clinical characteristics of patients in the persistent vegetative state. *Arch Intern Med* 1991; 151:930–932.
15. Steinbock B: Recovery from persistent vegetative state?: The case of Carrie Coons. Hastings Center, Report July/August 1989, pp 14–15.

Catfight in the labs

NAOMI R. BLUESTONE, MD

The political leadership of my specialty organization has become so shrill in its attack on the opponents of the use of animal subjects for medical experimentation, it has sparked the oppositional streak in me. I am now ready to spring to the defense of the underdog antivivisectionists, who advocate controls on unlimited research on the lower orders. This country's psychiatric leadership is so intent on uncovering the mysteries of the locus ceruleus and putamen, it is not about to let a bunch of perverted animal lovers do them in. That is consistent with all we have been taught. However, furry creatures, possibly excepting scruffy ones with hideously long tails and absolutely no redeeming social value whatsoever, should be protected from the carelessness and inadvertent cruelty of the increasingly dehumanized world of medicine. When the animal lovers of this world are deemed irrelevant, obstructive, and expendable, we will have finalized our sorry state indeed.

I have become sensitized to this interesting social issue by virtue of an experience with the thoroughly unplanned but clinically useful intrusion of an animal into my private practice. I tell the story the better to cast my vote that animals be used properly, both in basic laboratory research, and in the essentially unexploited area of clinical therapy.

A few years ago, I adopted a two-year old cat as a present for my husband who was still mourning the loss of Maxi, his recently departed Siamese. (Maxi, a petite lilac-point with the personality of a junkyard dog, was a 22-year-old who had gone to meet her maker in a far more merciful way than that accorded most super-annuated bipeds). Having left her under the butternut tree in New Hampshire with as much relief as pain, we were ready to welcome a newcomer. The new cat's previous owner, an elderly snowbird who was off to Florida for the season, brought her to us in a state of high dudgeon, having but recently discovered that all the silk wallpaper in her guest bedroom had been silently and systematically stripped by the forepaws of this feline menace.

Mini was a real beauty, with unusual mask and points. (We later discovered that she was a valuable blue-point Siamese.) We made it our business to rehabilitate her, no mean feat, for her basic temperament (until the neurological ravages of her final illness made her unusually docile) could have appealed only to a skilled therapist with a lot of free hours.

One day the bedroom window was accidentally left open, and Mini jumped out onto the roof over the den below. Locating the dogwood tree at its corner, she scooted down fearlessly and began to make herself at home in the garden. This became her habit, unknown to me, until one day I happened to look through the slats of the vertical blinds and saw her "talking" to a patient who was leaving her psychotherapy session (the office being at the rear of my home). The patient, who was soon fondling and playing with this basically suspicious and unrelated cat, was a schizoid type herself, the one person some might think least likely to approach a feline at all.

That, as the saying goes, was the beginning. Mini would greet and escort patients to the door, reverse the process on their departure, and pout when patients would have to leave her behind. Requests began that we admit her to our sessions because it seemed so mean to leave her alone outside. This became an exercise in setting limits and dealing with anger at me for not permitting the cat to join us as we did our work. Mostly, she was an instant activator of parental transferences, a potent trigger for early childhood memories. One women, for example, spent considerable time remembering her mother's sadistic habit of bringing home pets for her, only to spirit them away soon after during the night, after attachments had been made. (The patient presumed that they had been sold to those who figure in the animal research activities now under protest.)

Much of my direct out-patient/out-cat observation occurred from behind the almost-closed ribs of the vertical blinds, as each patient came and went. It became almost a game with me, to track the cat and its impact on my case load. I had some concerns, at first, that the orthodox establishment would come and get me if I persisted in my little deviance, but I took heart from the example of a much-revered senior analyst who persisted in admitting a yapping and sincerely hostile mongrel to her patient sessions, only to croon periodically, "Don't worry, Schatzie won't hurt you!"

It was strikingly obvious that each patient related to Mini in a way that reflected his or her own personality and problems. One woman, severely arthritic and unable to rise from a straight-backed chair, bent down to the ground to pick Mini up. Another, who went through a tortured pregnancy with a leukemic cat of her own and finally had to destroy it out of a desire to protect her own fetus, used Mini as a surrogate to work through her own grief, guilt, and despair. For another, the idea that a therapist would keep a cat was a revelation and a sign that I could be trusted. On the other hand, an obsessive expressed considerable loathing that I might be letting filthy paws walk on my food preparation surfaces, and so how could I be expected to understand her anxiety when dishes were left in the sink. Some patients, of course, never even became aware of her presence at all, these patients generally being classic "Type A" males. Mini became a therapeutic fixture and provided many insights for me. Like all illegally obtained evidence,

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however, I could not make use of it directly. After all, how could one say with a straight face, "I was spying on you a minute ago when you thought I was doing my notes, and I noticed that you were very nasty to my animal!"?

Within three years Mini was exposed to the dread feline leukemia virus, became emaciated and cachectic, and started hallucinating. For some inexplicable reason, we had assumed that the shots she had been given before adoption included this particular series, when in fact she had been left totally unprotected. It was a measure of our familial pathology that we did not recognize the signs of advanced illness until our by now much loved pussy cat was ready for passage to cat heaven. After a grim and expensive encounter with the vet, which we found exploitative, enraging and unnecessarily cruel, we cried like babies and sent her away to that great bourne from which no traveller returneth.

The day Mini was destroyed was the only day in six years of private practice that I was so grief stricken as to be unable to do my work properly. No stranger to death, I sent my husband off on the grim errand with selfish disregard for his suffering. We buried her next to Maxi up at the country place and still water up when we think of her. It was some time before we were able to start again with new seal-point, litter-mate kittens, whom we named for our two favorite bacteria, *Shigella* and *Salmonella* (Shag and Sally).

To tell or not to tell patients? I raised her absence as an issue only for those who had made a therapeutic alliance with Mini. One of them raged, accused me of neglect (not realizing how right she was!), and screamed, "That cat was easier to talk to than you were!" The schizoid woman cried for 45 minutes straight, schizoid no longer. With only one patient was it necessary to invent the therapeutic fiction of Mini being invited to a farm in the country where the cream was rich and the mice nimble. One patient took four months to say, "Didn't you used to keep a cat?" Those who never noted her presence never noted her disappearance. Narcissists all.

Mini did her work well. She had been useful as simile and metaphor, as object lesson and object relation. In time, I began to wonder how anyone could run a psychiatric office without a resident feline as reality tester, foil, and play object. She never inspired the dread that dogs sometimes do, except for one phobic patient who was so troubled by neighborhood felines and racoons that she came to her evening sessions with a torch to keep the nasty beasties away.

It is certain that Mini was by no means unique in her capacity to comfort and heal the troubled in spirit. I remember, for example, a "domestic longhair," resident illegally at a state hospital in New York City at which I was employed. Fed by a kind-hearted aide, she produced litter after litter of kittens for the delight of paranoid schizophrenic patients. None of these innocents succumbed to the atrocities of psychosis, as responsible staff feared. The literature is full of such stories, nor have they been given the attention they deserve.

One cannot have friends such as Mini without gaining a new perspective on the inherent worth of research animals. It is not fake sentiment to blur the boundaries between our own at-home pets and the creatures on whom we work. I would respectfully request our research colleagues not to attack our humanity and humiliate us for refusing to make that distinction. We are not all character disorders who indulge phallic monuments in granite at the pet cemetary, nor are we hysteroid euphorics who leave pets our fortune in perpetual trusts. It is sad that if one is opposed to all but the most ascetic and carefully monitored animal experimentation, one must feel like a closet religious in an analytic institute. We can do with less use of animals whose soulful eyes and wagging tails rightly give us pause. Let psychological science and human feelings no longer be at war! To reconciliation, not catfights!

Not a valentine

LOIS GREENE STONE

Ever hear a doctor ask, "How about heart disease in your family?" As a blood pressure cuff was being caught between velcro fasteners, I thought about my parents and how pleased they both were about their low blood pressure, as if it were a gold star awarded for good performance. What difference did it make for either of them! "How about heart disease in your family?" My answer is like a proclamation of doom when I have to describe my maternal/paternal aunts and uncles who died from defective hearts.

My father had been buried before jowls, loose flesh, or grey hair appeared. His light blue eyes once looked into a bathroom mirror daily, watching his hand smooth shaving cream on his overnight stubble; the heavy, whipped, white cream was stroked with a fat sable brush. After the pine box containing his body was placed into Long Island soil, I fondled the idle silky brush that stuck out of a mug, and I remembered the fragrance of his after-shave lotion. He had been afraid he would die during the fourth decade of his life as had his own father. His fear was well founded; he was 45. He made it four years past his father, as if it were a contest with a prize. He was lean, ate a good diet—my mother saw to that—and had risen from stock boy to one of the board of directors in a major Manhattan firm. Was it stress, genes, or fate? Why did his heart stop beating while he lay on the living room couch, clutching his chest as if he could squeeze out his pain with a clenched palm?

How could my mother have had a first heart attack in her early 50s! The second one was more intense, with massive, permanent damage. Blood clots raced from her pelvis to her lungs, threatening her already fragile intensive-care existence. Stubbornly she fought to survive and, to each attending physician's amazement, she did.

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Her third attack brought emergency open-heart surgery. Because of extensive injury to the heart muscle, survival was considered impossible. Not for my spunky mother, who carried her grief and misfortunes within and shared only her philosophy and smiles. A silent killer, contaminated blood received during transfusion, was the ultimate laugh that fate forced out. "Ha," it mused, "I'll let her live through this heart thing but I'll eradicate her by a means few would even consider." As contaminated blood moved through her system, it very slowly shut down all vital organs with its incurable and untreatable viruses.

I popped into my physician-husband's office before appointment hours had officially begun. He first put his stethoscope to my chest. A heart murmur had been with me since childhood, and a clicking noise passing up rubber stethoscope tubing is just a mitral valve that flops around but isn't anything to worry about.

My words, "humor me," as my spouse moved his trained eyes over the strips of paper being spewed from the electrocardiogram machine, changed his twinkle into terror.

I'm skinny, active, don't smoke, don't drink, eat balanced meals, and work out stress rather than let it overwhelm me. Obese people with boxes of fatty doughnuts beside their lit cigarettes were sitting in the visitors' corridors. I was placed in a hospital bed. I had inherited the best of my family's talents and abilities, but had I already inherited death by heart disease?

I imagined a "heaven" where one is rejoined with loved ones who have passed on. However, could my father find me since I'm no longer a 20-year-old girl? I am already beyond his living age and, since time stopped for him, how would I feel finding a parent younger than myself? How complicated a hereafter could be. I consciously tried not to think about this as I was wheeled into a room with words "cardiac catherization" printed on its door.

A cut was made in my groin so special tubes could be passed upward and finally into my heart. I knew the risks of injury to arteries, allergic reactions to dyes that would show up internal vessels, on-the-table sudden heart attacks, and blood clots pulling away and soaring to the lungs.

A foreign object was invading my body and traveling from my groin upward. I felt like the people on the doomed DC-10 airplane who watched the take-off and their crash on the plane's television monitor, since a viewing screen was put directly in front of my turned head. My eyes could not avert this object inching toward my beating pump. Might I watch it accidentally nick a vessel and see myself bleed to extinction? Perhaps non-medically informed patients find as much wonder in this image as seeing men on the moon. It is exciting yet unreal. I couldn't separate my personal medical knowledge from my body and look outside myself.

Dyes infused me, and my body felt fire within. The chambers and vessels were not being lit up, I cooly reasoned. Since I was still "feeling," I was still alive. My mother survived this technique in spite of a severely damaged heart from past heart attacks. I would be all right. I followed instructions, then focused my eyes on the tubing seen onscreen. If it nicked anything and I saw death coming, I would have time to utter last words. Why is horror so fascinating! Maybe this was not me anyway, but some acting lessons for an English course.

After the angiogram, a sandbag sat shapelessly on my groin, compressing the site of entry. I had come out of the cardiac catheterization alive and did not have a stroke or any other problems afterward. I briefly thought of local bleeding that would require surgical intervention, and I appreciated the adage "ignorance is bliss," although it could not apply to me. Along with my physician-mate, my older son is a doctor, and my daughter is a registered nurse. They, too, knew fluids had to be forced into my prone body to get rid of the dye that was used to light up my blood vessels. However, elimination of fluids had to be done without the sandbag moving out of position. My younger son, a certified public accountant, brought me violets.

The heart is sung about in songs, poetically praised in valentines, and broken by disappointments. Mine, for the moment, was spared a surgeon's knife.

An angiogram reminded me of my mortality; the time allotted to us is seldom long enough. I have accepted the gifts and burdens passed to me by genes. I sometimes think that an awareness that life is precious and briefer than one's hopes has made me extra productive.

Physician-assisted suicide

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The debate about the role of physicians in active euthanasia and assisted suicide is longstanding, ethically complex, and unlikely to be resolved soon.[1] Some physicians consider assisted suicide to be an act of murder, whereas others consider it to be an act of mercy. Can both be right? Must it be one or the other?

The debate was catapulted to center stage because of several recent developments, including the famous "It's over, Debbie" case,[2] the endorsement of physician-assisted suicide by a panel of distinguished physicians,[3] and the widely publicized Dr Jack Kevorkian[4] and Dr Timothy Quill[5] cases. Initiatives in several western states including California, Washington, and Oregon to legalize active euthanasia have so far failed but will be proposed again. The Hemlock Society's "how to" book entitled *Final Exit* was on the best seller list for many weeks and contributed to several widely publicized suicides.[6] These pivotal developments are briefly summarized below.

It's Over, Debbie

An anonymous first person account of active euthanasia by a house officer and a terminally ill, suffering cancer patient called Debbie whom he met for the first time that evening on call, was published by the prestigious *Journal of the American Medical Association (JAMA)*.[2] In a separate editorial, *JAMA's* editor defended his decision to publish the controversial, anonymous, and possibly fictitious essay. He stressed that the only reason for publishing it was to stimulate debate over euthanasia. An enormous outcry resulted both in the medical and lay press and in the mass media. Most were critical of the resident physician who administered the lethal dose of morphine to Debbie and also of the editor of *JAMA* for publishing it, although some supported the concept of "good death."[7] The editors of the *New York State Journal of Medicine* called the conduct of the house officer "an affront to human decency, a violation of medical ethics, and a crime. . . ."[8] An Illinois judge quashed a subpoena that would have forced *JAMA* to release the name of the anonymous resident physician. *JAMA* attorneys argued that *JAMA* was protected by law from disclosing the name of the essay's author. Officials of the American Medical Association and the editor of *JAMA* emphasized their strong opposition to mercy killing and said that they published the essay to stimulate debate and discussion.

Dr Jack Kevorkian's "Suicide Machine"

In a van in a suburb of Detroit on June 4, 1990, Dr Jack Kevorkian, a retired pathologist, connected Mrs Janet Adkins, said to be suffering from the early stages of Alzheimer's disease, to a homemade device which some people have called a "suicide machine" or a "self-execution machine." Mrs Adkins opened the intravenous tubing containing lethal potassium chloride and ended her life. Few actions in recent years received more public attention. *The New York Times* carried the story as a front page, three column article including a picture of Dr Kevorkian and his homemade intravenous device.[9]

Physicians, lawyers, and ethicists expressed their abhorrence of Dr Kevorkian and his suicide machine. A rare voice defended him.[10] In spite of the storm of criticism from the medical community and from the public, a discernible vote of sympathy for the patient was articulated by many people. Some physicians decried the absence of a thoughtful public debate regarding human suffering, assisted suicide, and euthanasia.[11] Charges of first degree murder against Dr Kevorkian were dismissed, but the judge issued a permanent injunction against using the suicide machine or other devices that aid people in committing suicide.

Defying the court order, Dr Kevorkian arranged for a dramatic double suicide in late October 1991 in a rustic woodland cabin north of Detroit. The public outcry was as vehement as the one after the June 1990 death of Janet Adkins. The two women who committed suicide using Dr Kevorkian's suicide machine released a videotape explaining why they wanted to die.[12] Since Michigan has no laws against assisted suicide, Dr Kevorkian was not prosecuted. However, his license to practice medicine was revoked. Bills have since been introduced in Michigan to make it a felony for intentionally helping someone commit suicide. The main reason for the ongoing debate of the patient's right to die and the physician's role in that right is the fact that physicians have previously hastened patients' deaths by removing or withholding medical devices or treatments. However, Dr Kevorkian used a "killing machine" to produce death.[4] Hence, the moral outcry.

Dr Timothy Quill and his Patient Diane

In an emotional, sensitive, and highly articulate article in the prestigious *New England Journal of Medicine,*[5] Dr

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Timothy Quill of the Genesee Hospital in Rochester, NY, described the case of his patient Diane. Diane was 45 years old when she developed acute myelomonocytic leukemia. Dr Quill explained all the options and their consequences. After careful reflection and consideration, Diane rejected chemotherapy and its one in four chance of long-term remission. After months of agonizing over Diane's decision, Dr Quill sadly accepted it. He wrote a prescription for enough barbiturates for Diane to commit suicide and made sure she knew how many pills she had to ingest to cause death.

Although the public media coverage of this case was as great as that after the Kevorkian suicides, Dr Quill's article was met with considerable understanding and even praise for his courage and forthrightness. His case reopened the debate of physician assisted suicide. Dr Quill was characterized as a sensitive, caring health professional who provided comprehensive medical care with deep concern for the patient's well-being and respect for her choices.[13] Not all physicians agree with the method used by Dr Quill, even if his motivations were noble. Certainly, assisted suicide is not part of hospice care.[14] The slippery-slope argument of "what comes next is euthanasia for the chronically ill patient without a terminal illness"[15] can also be made. One can also question the patient's decision, since Diane was only 45-years old and had a 25% chance of long-term cure.

A grand jury in Rochester declined to indict Dr Quill, and the New York State Health Department decided not to bring charges of professional misconduct against him for his admitted role in Diane's suicide.

The Dutch Experience with Active Euthanasia

The Netherlands allows physician participation in active euthanasia, although euthanasia is still a criminal offense. The legal community will not prosecute physicians who end the lives of patients provided a series of conditions are met: that there be an explicit and repeated request by the patient that leaves no reason for doubt concerning his or her desire to die; that the mental or physical suffering of the patient be very severe with no prospect of relief; that the patient's decision be well informed, free, and enduring; that all options for other care have been exhausted or refused by the patient; and that the doctor consult another physician.[16]

In 1990, in preparation for a discussion about legislation on euthanasia, the Dutch government appointed a commission to study physicians' practices regarding medical decisions and patient care at the end of life. The first results of the nationwide study were published in English by van der Maas et al shortly after the commission's final report was presented on September 10, 1991.[17] The results show that 49,000 deaths annually involve explicit end-of-life decisions such as foregoing treatment, using painkillers that hasten death, assisted suicide, and active euthanasia. About 9,000 patients annually request active euthanasia, and 2,300 receive it. In about 400 cases, physicians assist patients to commit suicide, and in more than 1,000 cases, euthanasia is administered without a specific request from the patient. The conclusion of the report is that euthanasia without a free and informed request is widely practiced in the Netherlands. Thus, such end-of-life decisions should get more attention in research, teaching, and public debate.[17] The Dutch experience remains a topic for both professional and public debate.

Legal Initiatives in the United States

In 1988, an attempt to place a Humane and Dignified Death initiative on the ballot in California was unsuccessful.[18] A similar initiative in Washington State called the "Death with Dignity Act," strongly opposed by the Washington State Medical Association, was defeated by a 56%–44% vote in 1991. The relatively narrow margin allows for a broad range of interpretations of the election results, with both sides in the debate claiming victory.[19]

Legislation was also introduced in Oregon that would allow physicians to give lethal injections to certain patients on request. The driving force behind these legal initiatives in California, Washington, and Oregon is the Oregon-based proeuthanasia Hemlock society, headed by Derek Humphrey, author of the "how to commit suicide" best-selling book entitled *Final Exit*. In New York State, Penal Law 120-30 states that a person is guilty of promoting a suicide attempt when he intentionally causes or aids another person to attempt suicide.

Physician Surveys

The Committee on Bioethical Issues of the Medical Society of the State of New York conducted a survey of 500 internists and 500 surgeons practicing in New York State (data unpublished). Responses were received from 541 (54%) physicians. Fifty-seven percent of the respondents were opposed to the legalization of euthanasia. Fifty-three percent felt that it is unethical for a physician to participate in active euthanasia, and 63% said they would not administer active euthanasia if it were legalized.

Obviously, a substantial minority of physicians favored the legalization of euthanasia and felt that it would not be unethical for a physician to participate in or administer euthanasia if it were legalized. In a national survey of 2,000 primary care physicians, 29.9% of respondents said that there are circumstances in which a physician is justified in causing a patient's death. Nearly 45% and 9.4% of respondents, replied that they had deliberately taken clinical actions that would indirectly or directly, respectively, cause a patient's death. Religious and legal repercussions were identified as the strongest outside influences in end-of-life decisions.[20]

Philosophical Issues

Advocates of physician-assisted suicide offer a number of emotional arguments in favor of their position. Questions of patient autonomy, death with dignity, or of a patient's right to determine his own fate are very different than the question of the physician's role in the care of the terminally ill patient. Many physicians acknowledge a patient's right to do what he wishes with his own life, but are strongly opposed to physician assisted suicide. They would argue that even if one accepts the notion that suicide in certain instances may be a rational or moral choice for the patient, it is the obligation of the physician not to take part in or assist the suicide. The most relevant question is not "Whose life is it anyway?" but "What does it mean to be a physician?"

Physicians, like the public at large, are clearly divided over the question of physician-assisted suicide. Much of the conflict between the opposing views derives from the fact that to be a physician is to have a dual obligation—a physician must both preserve life and minimize suffering. Those who advocate physician-assisted suicide argue, in effect, that at times the two obligations conflict, and specifically that physicians must sometimes abandon their obligation to preserve life to fulfill their obligation to minimize suffering.

The Committee on Bioethical Issues does not agree with this view. We believe there to be no fundamental conflict between these two obligations. A physician must not abandon either the obligation to preserve life or to relieve suffering. When it is not in the physician's power to preserve life, all his efforts should rightly be directed at relieving the patient's suffering, by administering, for example, whatever amount of pain medication is necessary. This is done with the recognition that intense administration of pain medication in a terminal patient may unintentionally help to precipitate his death. However, the physician must never intentionally cause or assist in causing the patient's death.

Pellegrino succinctly summarizes the various arguments for and against active euthanasia and assisted suicide:

> Those who favor voluntary euthanasia argue that it is a beneficent and compassionate act; it respects autonomy by preserving the patient's control of the manner, method, and timing of death; it takes the matter outside the reach of "medical power" and scrupulosity; and it prevents the injustice that allows some patients to choose death by refusal of life support measures, while denying others the right to do so by active euthanasia.[21]

Proponents of involuntary euthanasia argue that beneficence to patient, family, and health professionals, as well as the conservation of society's limited resources, makes involuntary euthanasia a positive moral duty of physicians.

Pellegrino presents a dissenting view:

> The opponents of active euthanasia argue that it undermines the value of, and respect for, all human life. They fear that guidelines cannot avoid sliding down the "slippery slope" to involuntary euthanasia and selective devaluation of the lives of the most vulnerable among us. Diagnosis, prognosis, intolerable suffering, and freedom of consent are all difficult to define. Moreover, euthanasia should be unnecessary, since the major reasons for requesting it—intolerable pain and fear of overtreatment—can now be handled by better palliative care, analgesia, and living wills . . . . Euthanasia, its opponents further argue, is socially destructive: it undermines trust in physicians, desensitizes society to killing, and imperils the ground already gained in legitimating passive euthanasia. Finally, and most powerful of all for many Americans, is the religious belief that human life is the gift of the Creator and that humans are its stewards but not its absolute masters.[21]

The Council on Ethical and Judicial Affairs of the American Medical Association has repeatedly expressed its opposition to a physician intentionally causing the death of a patient.[22] The World Medical Association issued the following declaration on euthanasia in October 1987:

> Euthanasia, that is the act of deliberately ending the life of a patient, even at his own request or at the request of his close relatives, is unethical. This does not prevent the physician from respecting the will of a patient to allow the natural process of death to follow its course in the terminal phase of sickness.[23]

Yet physician-assisted suicide received a tremendous boost when a panel of distinguished physicians, assembled by the Society for the Right to Die, concluded that if hopelessly ill patients believe their condition is intolerable, a physician should provide such patients with the medical means and the medical knowledge to commit suicide.[3] These physicians recommend aggressive use of pain-relieving substances, even when such use may result in shortened life.

The enormous correspondence in response to this article was published in a subsequent issue of the *New England Journal of Medicine*, much of it critical.[24] The authors responded by saying that they do not endorse the direct killing of suffering people but emphasize the creation of a peaceful environment for dying.[25] The American Medical Association's Ethics and Health Policy Counsel reacted to this article by saying that "much more thought needs to be given before involving physicians in the process and compromising their essential role as healers."[26]

One author traces the substantial changes that have occurred in medical practices and patient attitudes concerning life sustaining treatments during the past three decades.[27] Until the Quinlan case in 1976, it was considered unethical and unacceptable medical practice to remove the respirator or hydration or nutrition from a patient who was not brain dead. In the 1980s, courts have equated hydration and artificial feeding with other forms of futile medical therapy and have allowed their withdrawal in patients who were not terminally ill. This author concludes that, "Active euthanasia programs in the United States are likely in the near future."[27]

Our society has revised the definition of death and related standards for the maintenance of life.[28] We seem to be observing the slippery slope in these changing attitudes and practices concerning the treatment or non-treatment of the terminally ill. Dr Quill said that he was greatly influenced and encouraged to help his patient, Diane, commit suicide as a result of the famous article by prominent physicians in the *New England Journal of Medicine* sanctioning assisted suicide (personal communication). He is an experienced hospice medical director who claims that 5%–10% of patients who are dying continue to suffer in spite of adequate hospice comfort care or control of pain. He defines suffering as physical or emotional pain which includes "disillusionment of what it is that makes one a person, or loss of dignity" (personal communication). A person may believe that he has lost his dignity by being bedbound, incontinent, delirious, or dependent on others for care. Many hospice workers are outraged at such an approach, claiming that people who are comfortable, secure, and lovingly cared for do not want to commit suicide. One writer asserts that history has taught us that "it is but a short step that leads from assisted suicide to homicide, to genocide and ultimate moral abyss."[29]

Some writers applaud the growing attention to the right to die, rational suicide, and assisted suicide.[1] Stripped of its euphemistic overtones, however, physician-assisted suicide is viewed by other writers as the direct or indirect killing of patients by physicians.[30] One physician questions "Mercy-killing: mercy for whom?"[31]

Legalization of euthanasia is emerging as an important issue and physicians should be prepared to debate it.[32] In fact, the debate is already well under way. If physicians paid more attention to the relief of pain and suffering, including psychosocial, emotional, and spiritual support of the dying, there may be fewer requests for active euthanasia.[33] Most patients who exercise their right of autonomy say "please let me die" and not "please make me die."[34] Many reasons are offered for not legalizing euthanasia.[35] Two prominent bioethicists wrote the following:

> Pressure to legalize euthanasia will surely increase in an era of spiraling health care costs, but it must be resisted. Euthanasia represents a development that is dangerous for many vulnerable

> patients and that threatens the moral integrity of the medical profession. Physicians must become more responsive to the concerns of patients that underlie the movement for euthanasia and must provide better pain management, more compassionate terminal care, and more appropriate use of life-sustaining treatments. However, physicians need to draw the line at euthanasia. They and their professional associations should defend the integrity of medicine by speaking out against the practice. Finally, even if euthanasia is legalized in some jurisdictions, physicians should refuse to participate in it, and professional organizations should censure any of their members who perform euthanasia.[36]

On the other hand, two other prominent bioethicists argue that the debate over assisted suicide and euthanasia should shift to an examination of the needs and values of patients in a context that recognizes the limits of modern medicine and the inevitability of death.[8] They further assert that "the rigid view that physicians should never assist in suicide denies the complexity of the personal meanings life can have in favor of a single-minded devotion to its maximal duration." Although these authors do not support an indiscriminate easing of sanctions on physician assisted suicide, they suggest that the issues deserve a thorough and thoughtful analysis and "should not be obscured by inflexible rules."

CONCLUSIONS

Two diametrically opposing views on active euthanasia and physician-assisted suicide have split both the medical community and lay public approximately equally. One thesis is that a person who is suffering severe pain or distress that cannot be satisfactorily relieved by available medical/surgical techniques, and who is terminally ill or suffering from an incurable disease or disability, should have the right to elect to terminate his or her life and to obtain assistance in achieving an effective, painless, rapid death. Of course no physician should be obliged to provide such assistance, but a physician who is willing to should be permitted to do so without fear of criminal or civil liability.

The other point of view is that recent technological and therapeutic advances in medicine do not justify the alteration of the time-honored ethical canon prohibiting physician-assisted suicide and active euthanasia. The motto of physicians was, is, and always should be to affirm life and not to hasten or postpone death. This view allows and even mandates, under certain circumstances, the foregoing or withholding of futile or useless medical therapy and attention to pain relief, but not the active killing of a patient.

POSITION STATEMENT OF THE COMMITTEE ON BIOETHICAL ISSUES

Consonant with the recommendation of the Council on Ethical and Judicial Affairs of the American Medical Association, the Committee on Bioethical Issues of the Medical Society of the State of New York offers the following position statement.

The principle of patient autonomy requires that physicians respect the decision of a patient who possesses decision-making capacity to forego life-sustaining treatment. Life-sustaining treatment is defined as any medical treatment that serves to prolong life without reversing the underlying medical condition. Life-sustaining treatment includes, but is not limited to, mechanical ventilation, renal dialysis, blood transfusions, chemotherapy, antibiotics, and artificial nutrition and hydration.

Physicians are obligated to relieve pain and suffering and to promote the dignity and autonomy of dying patients in their care. This obligation includes providing effective palliative treatment even though it may occasionally hasten death. However, physicians should not perform euthanasia or participate in assisted suicide. Support, comfort, respect for patient autonomy, good communication, and adequate pain control may dramatically decrease the demand for euthanasia and assisted suicide. In certain carefully defined circumstances, it is humane to recognize that death is certain and suffering is great. However, the societal risks of involving physicians in medical interventions to cause patients' deaths is too great to condone active euthanasia or physician-assisted suicide.

REFERENCES

1. Conwell Y, Caine ED: Rational suicide and the right to die. Reality and myth. *N Engl J Med* 1991; 325:1100–1102.
2. Anonymous: It's over, Debbie. *JAMA* 1988; 259:272.
3. Wanzer SH, Federman DD, Adelstein SJ, et al: The physician's responsibility toward hopelessly ill patients. A second look. *N Engl J Med* 1989; 320: 844–849.
4. Annas GJ: Killing machines. *Hastings Center Rep* 1991; 21:33–35
5. Quill TE: Death and dignity: A case of individualized decision making. *N Engl J Med* 1991; 324:691–694.
6. Humphrey, D: *Final Exit: The practicalities of Self- Deliverance and Assisted Suicide for the Dying*, Eugene, OR, Hemlock Society, 1991.
7. It's over, Debbie. *JAMA* [letter] 1988; 259: 2094–2098.
8. Imperato PJ, Bluestone NR: It isn't over, Doctor. *NY State J Med* 1988; 88:171–172
9. Belkin L: Doctor tells of first death using his suicide device. *N Y Times* June 6, 1990, pp A1.B6
10. Angell M: Don't criticize Doctor Death. *NY Times* June 14, 1990; p A14.
11. Cassel CK, Meier DE: Morals and moralism in the debate over euthanasia and assisted suicide. *N Engl J of Med* 1990; 323:750–752.
12. Wantz M: Why we went to Dr Death. *NY Post* Oct 29, 1991; p 16.
13. King SA, Freer JP: Death and dignity: the case of Diane. *N Engl J Med* 1991; 325:658.
14. Standards of a hospice program of care. Arlington, VA, National Hospice Organization, 1987.
15. O'Connor NK: Death and dignity: the case of Diane. *N Engl J Med* 1991; 325:659.
16. deWachter MM: Active euthanasia in the Netherlands. *JAMA* 1989; 262: 3316–3319.
17. Van der Maas PJ, van Dellen JJM, Pijnenborg L, Looman CWN: Euthanasia and other medical decisions concerning the end of life. *Lancet* 1991; 338:669–674.
18. Parachini A: The California humane and dignified death initiative. *Hastings Cent Rep* 1989; 1:(suppl)10–12.
19. Merz B: Despite defeat of state's suicide initiative, issue still unsettled. *American Medical News* Nov 18, 1991;pp1,23.
20. Kanoti GA, Orlowski JP: National survey: Physicians' views on the right to die. *Physician's Management* 1991; 31:40–76.
21. Pellegrino ED: Ethics *JAMA* 1991; 265:3118–3119.
22. Council on Ethical and Judicial Affairs of the American Medical Association. "Current Opinions," American Medical Association, Chicago, 1992.
23. World Medical Association: World Medical Association declaration on euthanasia. Document 17.P, presented at the 39th Assembly, Madrid, October 1987.
24. The physician's responsibility toward hopelessly ill patients: A second look. [letter] *N Engl J Med* 1989; 321:975–978.
25. Wanzer SH, Federman DD, Adelstein J, et al: The physician's responsibility toward hopelessly ill patients. *N Engl J Med* 1989; 321:978.
26. Orentlicher D: Physician participation in assisted suicide. *JAMA* 1989; 262:1844–1845.
27. Sprung CL: Changing attitudes and practices in foregoing life-sustaining treatments. *JAMA* 1990; 263:2211–2215.
28. Carton RW: The road to euthanasia. *JAMA* 1990; 263:2221.
29. Stoddard S: Terminal but not hopeless. *NY Times* August 31, 1991, p A19.
30. Spritz N: Speeding the dying. *Hastings Cent Rep* 1991; 21:4.
31. Goodwin JS: Mercy killing: mercy for whom? *JAMA* 1991; 265:326.
32. Angell M: Euthanasia. *N Engl J Med* 1988; 319:1348–1350.
33. Lynn J: Euthanasia. *N Engl J Med* 1989; 321:119.
34. Scofield GR: Euthanasia. *N Engl J Med* 1989: 321:120.
35. Conolly ME: Euthanasia. *N Engl J Med* 1989: 321:119–120.
36. Singer PA, Siegler M: Euthanasia–A critique. *N Engl J Med* 1990; 322: 1881–1883.

History—The great reminder

The relationship between the Long Island College Hospital and the State University of New York Health Science Center at Brooklyn

JOHN N. EDSON, MD

Friday, May 1, 1992, marked the signing of an agreement of mutual assistance between the State University of New York Health Science Center at Brooklyn and the Long Island College Hospital. The Hospital became the primary voluntary affiliate of the Medical School for the clinical teaching of medical students. Although each remains autonomous, the bond is intended to strengthen the stature of both.

Dr Duncan Clark, Professor Emeritus of Preventive Medicine and Community Health at the Health Science Center and former Dean of the Long Island College of Medicine, was chosen to introduce the speakers, for he had witnessed the separation of the Medical School from the Hospital in 1930 and the evolution of the School as it became a part of the State University of New York in 1950.

The ceremony took place in the beautiful Court Room of Brooklyn's Borough Hall. Borough President Howard Golden was on hand to give his blessings to this latest venture in medical education. Dr Donald J. Scherl, President of the Health Science Center at Brooklyn, and Mr Joseph Broadwin, Chair of the Board of Regents of the Hospital, spoke briefly about the advantages of this closer union between the Medical School and the Hospital. Mr Harold L. Light, President and Chief Executive Officer of the Hospital, described the long-involved process taken to arrive at the final document, and thanked members of the Medical School and the Hospital for their cooperation. After these brief remarks, the Affiliation Agreement was signed by Dr Scherl and Dr Irwin M. Weiner, Dean of the College of Medicine of the Health Science Center, and by Mr Broadwin and Mr Light. Although this was an auspicious occasion, a more auspicious one took place back in 1858, when the original Long Island College Hospital charter for a hospital-medical school was received from the State of New York.

In 1856, two physicians of German extraction envisioned a need for a free dispensary for the poor German population of downtown Brooklyn. These two physicians, Dr Louis Bauer and Dr Gustavus Braeunlich, purchased a building on Court Street and opened its doors to needy patients. According to the Dispensary records, the first patient was Louis Prell who was diagnosed as having "splenitis chronica."

Soon thereafter, the need for a hospital became evident. In fact, a number of operations were performed in the Dispensary, with some patients being held there overnight. Drs Bauer and Braeunlich interested Dr Daniel Ayres and Dr John Byrne, prominent physicians practicing on Brooklyn Heights, in the need for a hospital to care for these indigent patients. They, in turn, convinced several distinguished lay persons living in the community to assist in this endeavor.

Dr Ayres, Bauer and Byrne, and a little later Dr William H. Dudley, had observed training programs for physicians in Europe, where they had studied. Consequently, when a need for a hospital arose in downtown Brooklyn, they envisioned it as a teaching hospital where, in addition to caring for needy patients, a school could be established for training students to become physicians. Thus, when an application for a charter went to the State of New York in early 1858, the application included the request to grant properly trained students the degree of Doctor of Medicine.

Section 9 of that original Charter, issued on March 6, 1858, stated[2] "The Regents for the time being shall have the power to grant and confer the degree of doctor of medicine upon the recommendation of the Faculty and Council of said institution; but no person shall receive a diploma conferring such a degree unless he shall be 21 years of age, and shall have received, at least, a good education, and have pursued the study of medical science at least three years after the age of sixteen, with some physician or surgeon duly licensed by law to practice his profession, and shall also after that age have attended two complete courses of lectures delivered by the professors of this college."

Section 11 goes on to state: "There shall be a Medical Council, and a Faculty of Physicians, Surgeons, and Professors, who, together with all medical and surgical assistants that may be required, shall be appointed by the Regents, as hereinafter provided."

The Board of Regents and their medical advisors very carefully developed an outstanding faculty, including Austin Flint, Sr, MD, LLD, Professor of the Theory and Practice of Medicine, Frank H. Hamilton, AB, AM, MD, LLD, Professor of the Principles and Practice of Surgery, John C. Dalton, AB, MD, Professor of Physiology and Microscopic Anatomy, and Robert O. Doremus, AM, MD, LLD, Professor of Chemistry and Toxicology.

Unfortunately, in 1859, before the medical school could start, the Hospital lost what little money it had. The Perry Mansion on Henry Street, acquired in 1858 and already used for patients, had to be sold at public auction. Dr William H. Dudley, realizing the urgency of the situation, bought the Hospital and loaned it to the Regents until they could pay off his mortgage on the property. Thus, the first medical school term was presented in the spring of 1860.[1(p31)]

The privilege of granting degrees in Medicine given "for the time being" to the Board of Regents of the Long Island College Hospital lasted until 1930, when the State gave the

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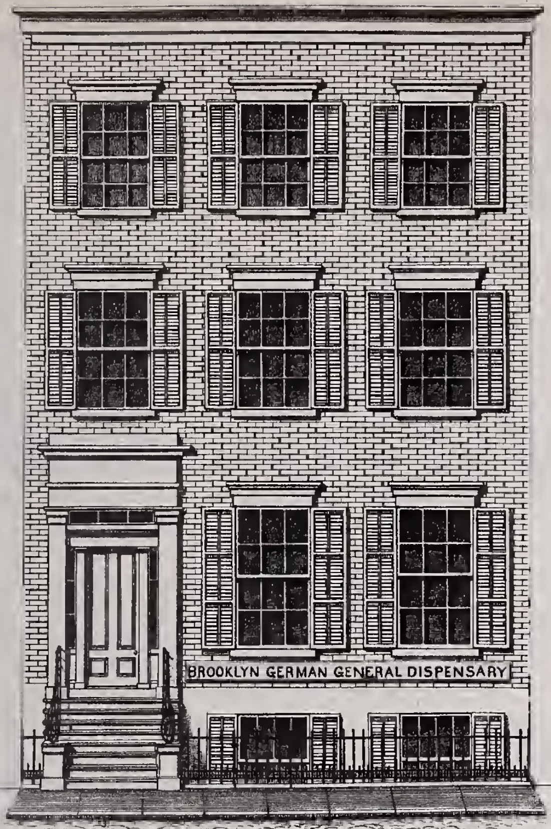

FIGURE 1. The Brooklyn German Dispensary on Court Street, 1856 (reproduced from *History of The Long Island College Hospital and Its Graduates*, Brooklyn, Association of the Alumni, 1899).

degree granting privilege to the Long Island College of Medicine, and in 1950, to the State University of New York.

Every since the Long Island College Hospital's medical school began, teaching medical students has been a major concern of the hospital. True to its reputation of being the first hospital in the country to have a medical school develop within it, there has been a continuum of teaching medical students right up to the present. Actually, this latest agreement with the Health Science Center is the third document of its kind joining both institutions in a

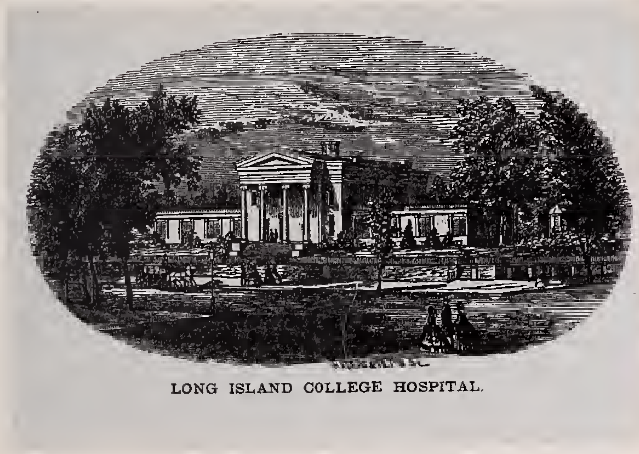
LONG ISLAND COLLEGE HOSPITAL.

FIGURE 2. The Perry Mansion on Henry Street, purchased in 1858 as The Long Island College Hospital (reproduced from History of *The Long Island College Hospital and Its Graduates*, Brooklyn, Association of the Alumni, 1899).

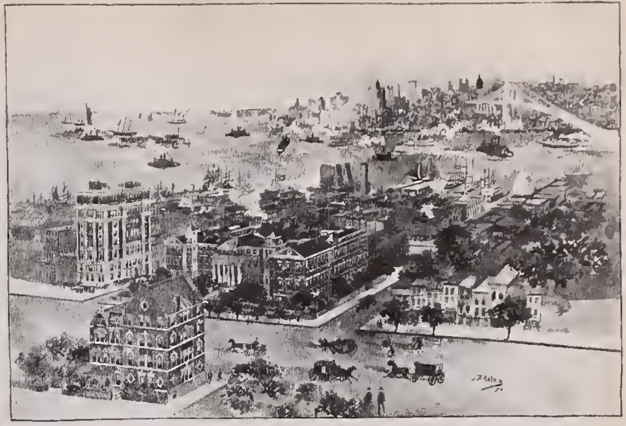

FIGURE 3. The Long Island College Hospital, the Hoagland Laboratory, the Polhemus Memorial Clinic, with New York harbor and the Statue of Liberty in the background, as envisioned by J. F. Nolan in 1898 (Courtesy of the library, State University of New York, Health Science Center at Brooklyn).

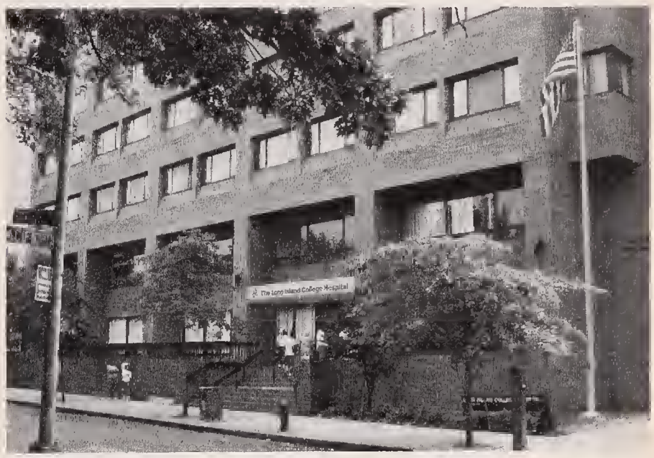

FIGURE 4. The new entrance to The Long Island College Hospital, through the in-patient Fuller Pavilion on Hicks Street (Courtesy of The Long Island College Hospital).

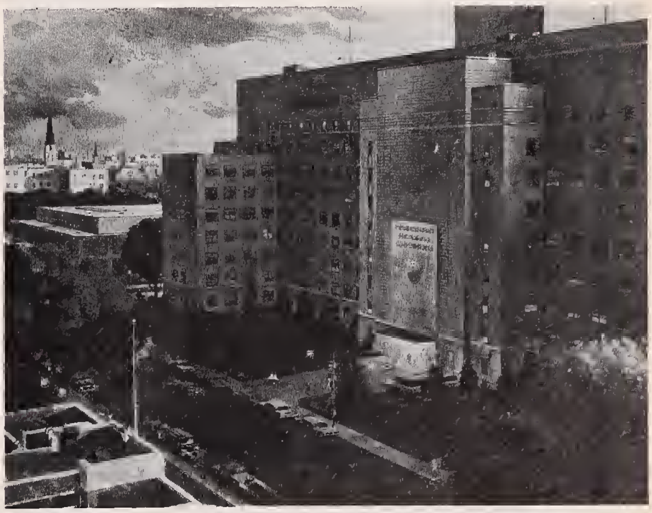

FIGURE 5. State University of New York Health Science Center at Brooklyn (Original color printing by Robert McCall, 1966. Collegia Media Squibb Collection. Courtesy of E. R. Squibb and Sons, Inc).

teaching arrangement. The first occurred in 1963 when Dr Robert A. Moore was President of the Medical School, and the second in the 1970s when Dr Calvin Plimpton was President.

REFERENCES

1. Raymond JH: *History of The Long Island College Hospital and its Graduates* Brooklyn, NY, Association of the Alumni, 1899.
2. Charter granted to the Long Island College Hospital by the State of New York, 1858 (Archives, The Long Island College Hospital).

Health care for seamen in the Port of New York

Part II: The rise of the Seaman's Retreat

FLORENCE KAVALER, MD, MPH; SHIRLEY A. ZAVIN, PHD

Preparation of the hospital grounds and buildings had gotten underway soon after the Corson farm was acquired. Two wells were sunk and advertisements placed for the construction of a wharf, 130-feet long and 20-feet wide, a hospital structure, barns, fences, and other outbuildings.[1] Shown on the surveyor's map which accompanied the sale is the former Corson homestead (Fig 1);[2] to the Corson farmhouse was added a mirror image of the original building, with a front porch and matching doors at each end (Fig 2). The capacity of this, the hospital's first building was considered to be approximately 90 patients (Fig 3).

The inadequacy of the original building to fulfill the hospital's anticipated mission—the reception of all sick and disabled seamen in the port of New York—had certainly been evident to the trustees even before the addition to the Corson farmhouse, intended surely as an interim facility, had been completed. By early November 1831, the Executive Committee had contracted for the construction of a hospital with an anticipated capacity of 150; when completed, by January 1832, the hospital's total capacity would reach 240.[1]

Construction probably began soon thereafter, and the exterior walls had been completed by January 1832, when a fierce winter storm struck Staten Island and demolished most of what had been built. Later commentators address the folly of building during that time of year and attribute the weakness of the walls to the fact that the mortar had frozen rather than set. The debris was collected, and construction plans were postponed until the spring.

By June 1832, the new hospital had been rebuilt and was opened the following month. It is this building that appears in the background of a charming *naive* watercolor drawing of the Retreat grounds done by a Norwegian seaman, Lars Christensen, the 30th patient listed in the book of admissions, who stayed at the hospital between March 1–20, 1833 (Fig 4).[3]

Completion of this building did not terminate the hospital's building program. In 1833, a proposal was made to construct a "main building" that would be attached to the recently finished structure, but this plan had been discarded in favor of an even more elaborate and grandiose building plan. With construction of the Retreat's third new building, which exists today and is actively occupied, the hospital's total capacity reached 300.

The planner's goal was to construct a building that would be even more monumental and impressive than the previous brick structure. The building materials chosen for the Retreat's newest hospital, hammered "blue stone," granite trim, and an abundant use of copper, were closely modeled after the hospital's at Quarantine with their Greek Revival style or, as contemporary documents refer to it, "Grecian" and more sophisticated and more modern than that of the hospital on the hill.

On July 4, 1834, the cornerstone of the new building was formally laid by the president of the trustees assisted by the builder. In a leaden box were deposited the newspapers of the day, a *Sailors' Magazine* for the month of June 1834, and a plate engraved with a lengthy inscription that has been preserved in Dr Moffatt's 1862 history.[1]

After completion of the building in 1837, no further expansion of the hospital was required for another decade. An epidemic of ship fever in 1847 and the resulting increase in admissions placed such a strain on the existing facilities that the Board voted in July to enlarge the hospital by adding new wings.

During this period of expansion and growth, from 1831 to 1852, many other changes to the hospital's physical plant also occurred. Of the various buildings recorded in the documents as having been constructed on the grounds (wash house, gate house, dead house, barns), the only one besides the main hospital that remains today is the Chief Physician's house on the north edge of the Retreat grounds.

References are also made to an ice house, carpenter's shop, and carriage house. Although the hill hospital continued to be used as temporary ward space when the "lower hospital" was filled, it was adapted for other functions as well. In 1841, part of it was converted to a "lunatic asylum," a use it retained over a long period of time. An oven was constructed there the same year, and a portion of the building was put into use as the hospital's bakery. Here too, at least in the 1840s, were the segregated wards for the "colored" patients.[1]

From the beginning there was a cemetery on the Retreat grounds. The original burying ground was located a relatively short distance west of the hill hospital. By 1837, it was described as "nearly filled up,"[1] and the trustees ordered it enlarged by moving the boundary fence some 60–70 feet farther west. In 1847, it again required expansion, but by 1849 the trustees had decided to establish the cemetery in

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Part I appeared in the August issue of the *New York State Journal of Medicine*.

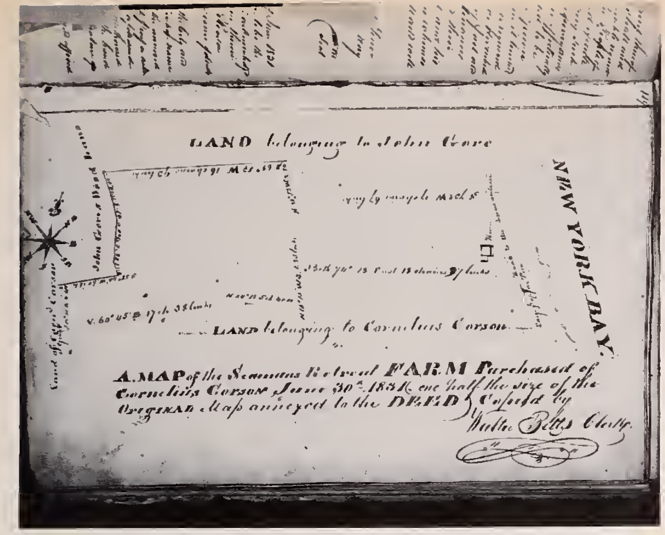

LAND belonging to John Gore
NEW YORK BAY.
LAND belonging to Cornelius Corson
A MAP of the Seamans Retreat FARM Purchased of Cornelius Corson June 30th 1831 one half the size of the Original Map annexed to the DEED. Copied by Walter Betts Clerk.

FIGURE 1. Surveyors maps of the Corson Farm, Staten Island, 1831 (Credit, Office of the Richmond County Clerk, *Liber of Deeds T*, p 165).

an entirely new location. The earlier burials were disinterred and the land was incorporated as part of the Retreat's farm. Later excavations on hospital property, most recently in the 1960s, have unearthed remains from the Retreat's first cemetery.

The Retreat grounds also provided ample room for the farm and gardens, supplying much of the hospital's meat and produce. Included among the early records are reports on the purchase of farming equipment and construction of woodsheds and barns. The annual harvest for 1849 is cited here as an example to suggest the extent of this activity:

> Superintendent's Report, Dec. 1, 1849 . . . the amounts of produce raised on the farm this year is as follows: Potatoes 523 bushels, Turnips 111 bushels, Carrots 30 bushels, Beets 30 bushels, Cabbages 1163 heads, Celery 522 heads, Leeks 3300 heads, Corn 50 bushels, Hay 10 tons, Peas, Beans, Pumpkins, Squashes, etc. all sufficient for use . . . Dec. 8, 1849 . . . I have killed the eighteen hogs, sold five and gave Dr. Boardman one and salted the rest. The whole weighed 3,586 lbs . . . made 100 lbs. of sausage, cut out the hams and shoulders and salted the rest.[3]

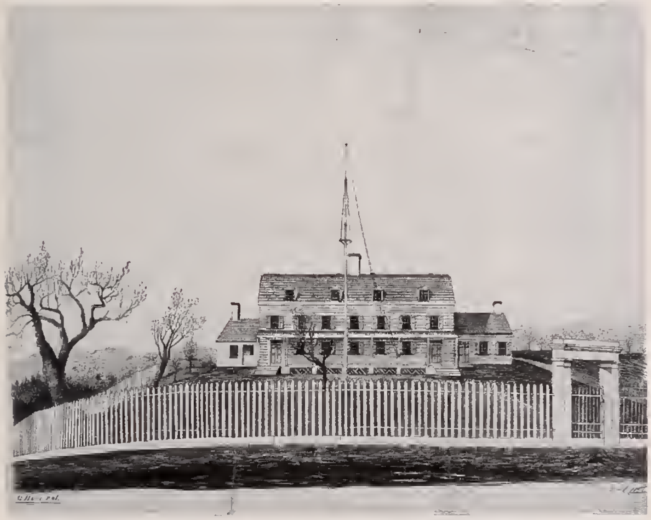


FIGURE 2. Captain Gamaliel Hart, The Seaman's Retreat, Staten Island, 1832. Watercolor drawn during hospital stay at the Retreat, showing the extension added to the Corson farmhouse to accommodate 90 patients (Credit, The New-York Historical Society).

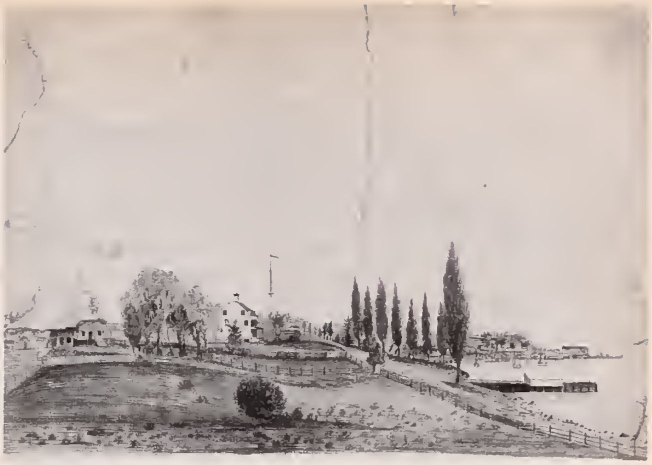


FIGURE 3. Captain Gamaliel Hart, The Seaman's Retreat, Staten Island, 1832. Watercolor showing the hospital from a vantage point near today's Vanderbilt Avenue, with the dock at the right; in the distance is the Village of Stapleton (Credit, The New-York Historical Society).

In addition, the need for milk, considered particularly important for the treatment of "fever," was usually met by the hospital's own dairy cows. Although the yields varied from year to year according to the weather and the hospital's needs, the farm remained an active one throughout the period of the institutions's existence as the Seaman's Retreat; its last annual report, that of 1881, still includes a record of the year's crops.

BOARD OF TRUSTEES AND ADMINISTRATION

Just as the hospital's physical plant had evolved during this period, so too was there a parallel evolution of its administrative organization and operating policies. A basic organizational framework for the new hospital had been established by the provisions of the 1831 Act. Under its terms, ultimate responsibility was vested in the Board of Trustees. It was the Board that had the authority to collect and disburse the funds allocated by the Act; it had the power also to appoint "a physician, assistants, nurses and other attendants," to determine their salary, and to make the necessary rules and regulations for the governance of the hospital. A more specific statement of policies for the hospital's daily operations was required, and in October 1832, the Board adopted a "carefully prepared set of

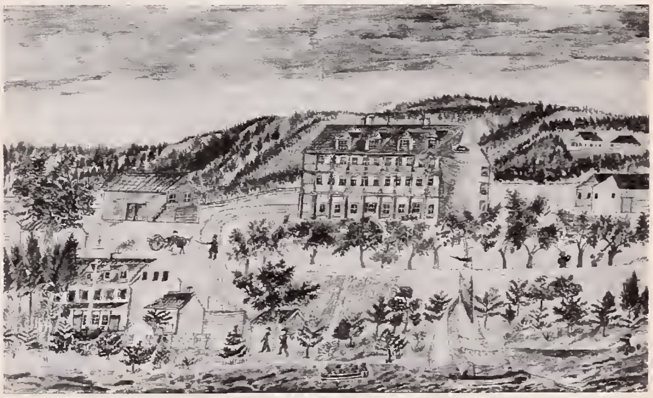


FIGURE 4. Seaman's Retreat, 1853, The North wing was added in 1848 and the South wing in 1853. Shown in this print, but never built, was a house for the Superintendent on the south edge of the property identical to the Chief Physician's residence on the right (Credit, Staten Island Institute of Arts and Sciences).

bylaws" in which "the respective duties of the Chief Physician and Superintendent were explicitly stated."[1]

The bylaws did not, however, immediately succeed in resolving the hospital's principal organizational problem during its earliest years: the division of administrative authority between the offices of Chief Physician and Superintendent. Differences were intensified by the evidently strong and forceful personalities of several early holders of these positions, personal rivalries, and perhaps, most basically some degree of incompatibility between the medical and the nautical aspects of the new institution. As legislated, the Board of Trustees was dominated by marine interests; its five appointed members were to be ship captains, and three of the five ex-officio Trustees had similar backgrounds. The policy adopted by the Board of having a former sea captain fill the position of Superintendent would seem to be, in part, an expression of the nautical community's continuing proprietary interest in an institution that it had struggled so long to bring into existence. In contrast, there was only one representative from the medical profession on the Board, the Health Officer (Health Commissioner) of the City of New York.

The duties of the Chief Physician, as outlined in the bylaws specify the following:

> He shall have the immediate charge of the treatment and classification of the sick . . . He shall visit each patient in the Retreat at least one every day . . . He shall direct the quantity and quality of food for each patient, give such directions to the Superintendent relative to the neatness and ventilation of the wards, cleanliness of the beds, bedding, etc . . . and report to the Superintendent all such patients as are in proper condition to leave the Retreat . . . He shall have charge and direction of all medicines . . . and present a written order to the Superintendent for such as are wanted who . . . shall procure them.[4]

The Superintendent was responsible for the following:

> . . . (he) shall act as Steward . . . and see that the regulations of the Trustees and the directions of the Visiting Committee are complied with . . . He shall have charge and custody of the Farm, Stock, Buildings Gardens . . . He shall purchase . . . fuel, provisions, and all other stores and necessaries . . . except medicines, of which he shall procure only as the Resident Physician shall . . . direct . . . He shall have power to hire and dismiss all laborers and servants (except such as are in immediate attendance on the sick, or whom he shall hire in such number, and such description of persons as the Resident Physician shall direct) and he shall cause all such as are guilty of profane swearing, drunkenness . . . or other gross misconduct to be dismissed . . . the admission and discharge of all patients shall be entered in a register . . . by the Superintendent . . . he shall state to the Visiting Committee . . . such improvements and alterations as have suggested themselves to his mind . . . by which the usefulness of the Institution may be extended.[4]

The hospital was, as described in a nautical metaphor by one of the early trustees, like a ship with two captains. The bylaws also outlined the role of the Assistant Physician, who was to be under the direction of the Chief Physician and would "assist in the performance of all his duties" and "in the temporary absence of the Chief Physician . . . shall resume his place."[4] A Visiting Committee was constituted to serve as a liaison between the Board of Trustees and the two hospital executives and was composed of two Board members serving one-month terms at a time.

It is evident that the roles of the Chief Physician and Superintendent, as defined by the bylaws, retained areas of responsibility in which administrative authority overlapped. This potential source of conflict was further exacerbated by the Board's early policy demanding that the Chief Physician and Superintendent work together as a team; if irreconcilable differences between them existed, both were to be dismissed, regardless of who bore major responsibility for the discord. Thus, the hospital's earliest holders of these offices served relatively brief terms.

Dr Peter Townsend, the first Chief Physician, developed severe disagreements with each of three successive Superintendents (all former seafaring man) culminating in their dismissals as well as his own (Table 1). These conflicts were described by Dr Townsend at an 1840 legislative investigation of the Retreat (recorded in documents of the New York State Assembly).

> A superintendent, with equal powers with the physician will of necessity clash and produce discord; and when witness was in the Retreat, they did lead to collision and quarrels . . . The Superintendent's duties were pointed out by the rules of the house, and also the Physician's. Sometimes the supplies were not approved of by myself, as Physician, as to quality, and was one of the causes of difficulty between him and myself; sometimes too, as to quantity of food I differed with him. He, the Superintendent, assumed the right to dismiss the nurses and men, which I disapproved of. This coolness led to many unpleasant feelings between us, and I was not permitted to live out which might have obviated some of them.

The hospital's additional staff had greatly expanded beyond the two nurses, cook, washerwoman, and farmer who served the hospital when it opened in October 1831. An 1845 Annual Report describes the personnel and their jobs, revealing a considerable amount about the hospital's day-to-day operation:

> The apothecary at $95 per month . . . prepare all medicines . . . to care of the medicine room, surgical instruments . . . The carpenter, at $20 per month . . . to do everything . . . appertaining to his trade . . . keeping the different houses in repair . . . making coffins, building sheds.
>
> The washerwoman and two assistants at $10 per month each . . . to wash for the whole establishment . . . about 800 pieces per week, then to be ironed and folded and distributed to each nurse to whose wards they belong.
>
> Two orderly men, at each $10 per month each . . . to keep the establishment clean, sweeping the halls and piazzas daily . . . attending to remove the dead from the house to the examining room, and from thence to the grave.
>
> The barber, at $10 per month . . . four days in the week shaving patients, and the rest of the time in assisting two orderlies.
>
> The cook and his mate at $10 each . . . to cook for the whole number of patients and hired people, an average of 140 persons a day, the year round.
>
> The scullion at $10 per month . . . to take care of the close-stools and chamber-pots, in every ward, emptying them and keeping them clean.

TABLE I. Early Executive Officers

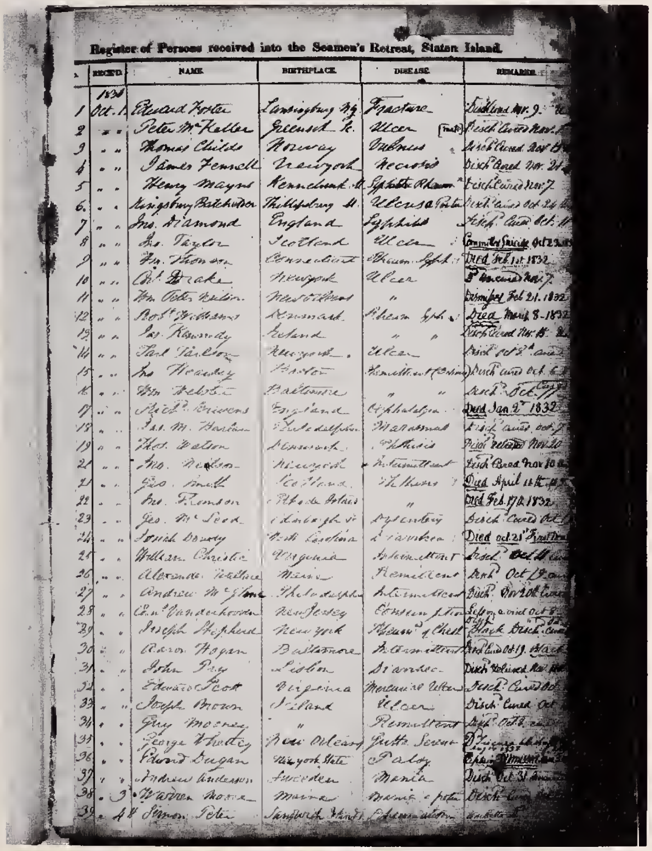
	Chief Physicians	Superintendents
1831	Dr. Peter Townsend	Captain David Bray Captain James Morgan Captain David Russell
1833	Dr. Robert Hathaway	Captain Thomas Brownell
1836	Dr. James Boardman Dr. John S. Westervelt (Principal Physician)	Captain Oliver O'Bear
1843		Captain Thomas Vermilye
1844	Dr. Samuel Boyd	
1845	Dr. William C. Anderson	Captain James Hart
1849	Dr. James Boardman (reappointment)	
1854	Dr. Thomas Moffatt	

> The coachman at $15 . . . to take care of three horses, also, the carriages, carts and wagons . . . to attend the steamboat with the carriage, for the transportation of patients, each time she arrives; do all errands, attend for the marketing; and lend a hand upon all occasions.
>
> The nurses, of whom there are nine, at $10 each; seven of these are attached to the main hospital and are under the supervision of the physician as to their attendance upon the sick . . . two are stationed at the insane wards and have the care, at present, of eight men; their duty is quite arduous as they, also, keep watch during the night over the whole place, assisted by one of the laboring men.
>
> The steward at $25 per month . . . in receiving all stores purchased for the hospitals by the Superintendent, and the serving out the same as per order of the physician and superintendent . . . has the oversight of all the hired people, seeing that they do their duty in their various stations, preventing all waste or abuse of the substance of the institution.

At the close of this period in the hospital's history, a similar enumeration of staff (prepared for the 1853 Annual Report) reveals that although admissions were 1,158 more than they had been in 1845, there was no substantial increase in nurses and support staff.[5] It is difficult to evaluate the health care implications of such statistics as these, except to note the paucity of health care staff for the number of patients served.

UTILIZATION REVIEW

The first patients, their names, places of birth, and diagnosed diseases transcribed in the book of admissions, were 39 seamen transferred to the Retreat from the Quarantine on October 1, 1931 (Fig 5).[3] There were both American and foreign born; they came from Maine, New York, Baltimore, Virginia, New Orleans, Norway, Scotland, England, Ireland, Lisbon, and the Sandwich Islands. Their diseases, many endemic to their vocation—fracture, ulcer, rheumatism, syphilitic rheumatism syphilis, intermittent and remittent fevers, diarrhea, phthisis—were predictive of the illnesses that would be treated at the hospital over the ensuing years.



Register of Persons received into the Seamen's Retreat, Staten Island.
RECD.
NAME.
BIRTHPLACE.
DISEASE.
REMARKS.

FIGURE 5. Register of persons received into the Seaman's Retreat, 1831, The Retreat's first patient-39 seamen transferred from the hospital's at Quarantine were admitted on October 1, 1831 (Credit, US Public Health Service Hospital Archives, Staten Island, NY).

In the hospital's first official report made to the New York State Legislature in December 1831, Captain James Morgan, as Secretary of the Board of Trustees, summarized the hospital's first three months of operation.[5] Between October 1 and December 31, 1831, 167 patients had been admitted. The hospital's first statistical tabulation continues as follows:

> There has been discharged, cured—78; at their own request discharged, relieve—8; at their own request discharged, uncured—4; left on a visit, not returned—1; there is now remaining in the Retreat—71 persons.

In addition, others were maintained elsewhere at the trustees' expense: funds were paid to the New York Hospital to maintain the seamen who had exceeded their four-month limit there, several were confined at Bellevue Hospital in Manhattan with small pox, and one patient was in the Bloomingdale lunatic asylum. Future plans announced in this first report included the establishment of a satellite clinic or "receiving house" in Manhattan to "obviate delay in giving of immediate relief." It was to be staffed by four physicians and a surgeon on a volunteer basis.

To provide these numbers with a human face, to suggest the particular health care problems that would remain the Retreat's principal concern, and to illuminate the rigors of life aboard ship (brigs and schooners) to which many of the problems were directly attributable, excerpts are included here from the hospital's first case histories (Fig 6):

> Jacob Schultze-Scorbutus-age 32-Born in Bremen-arrived last night in brig from round Cape Horn. Has been to sea 118 days and had had nothing but indifferent salt food to feed upon; and rice once a week only and a short allowance of water-was the carpenter on board. Twenty days after sailing his gums became sore and spongy and bled very freely . . . the ankles and insteps and so around the small of each leg caked hard and over the instep of a deep blue almost black color . . . during the whole time especially at first suffered universal pain, especially in the muscles of the thorax . . . very much prostrated and emaciated and was brought into the Retreat. Had no lime juice on board nor any other antiscorbutic so effectual in preventing as well as curing scurvy . . . it is in this shameful manner vessels are provided to the destruction of seamen . . . an object of pity to behold.
>
> Richard Thompson-age 15-born in Whitby, Yorkshire-arrived in brig last week in May 1832, from Havre. About a week before his arrival fell off the main loft gallant yard, catching in the main loft bowling and thence on deck striking his head on the long boat as he fell. His scalp was gashed and he was stunned senseless for some time . . . they carried him down in the cabin and bled him.
>
> Daniel Walker—Hernia Inguinalis-age 32-born in New York-arrived eight days ago in brig from London. Three weeks ago in consequence of bad weather night and day constantly reefing, a rupture took place in the right groin. Sir Gilbert Blane remarks that the practice of reefing causes among sailors an unusual proportion of injured ruptures.
>
> Peter McLean—Syphilis-age 26-born in Perth, Scotland-arrived on brig from Liverpool. About 10 weeks since (having put into Kinsale from stress of weather) a small cancra appeared on the plenum also on the top of the glans and a third within the prepuce behind and above the corona. On the passage the captain gave him near 100

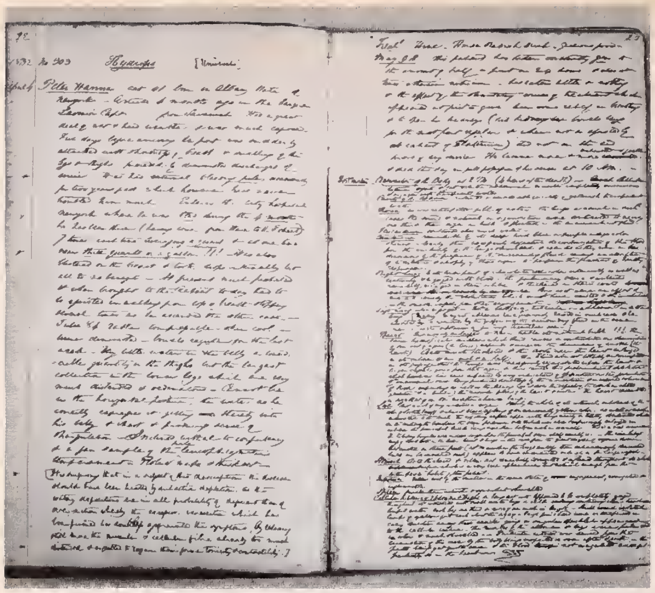


FIGURE 6. Case history: Peter Hanna, 1832, representative of the early case histories (Credit, US Public Health Service Archives, Staten Island, NY).

mercury pills (probably the Blue ones) which made his gums quite sore and about four weeks after the first appearance of the chancres a bubonic inflammation commenced in the right groin but did not proceed to suppuration.

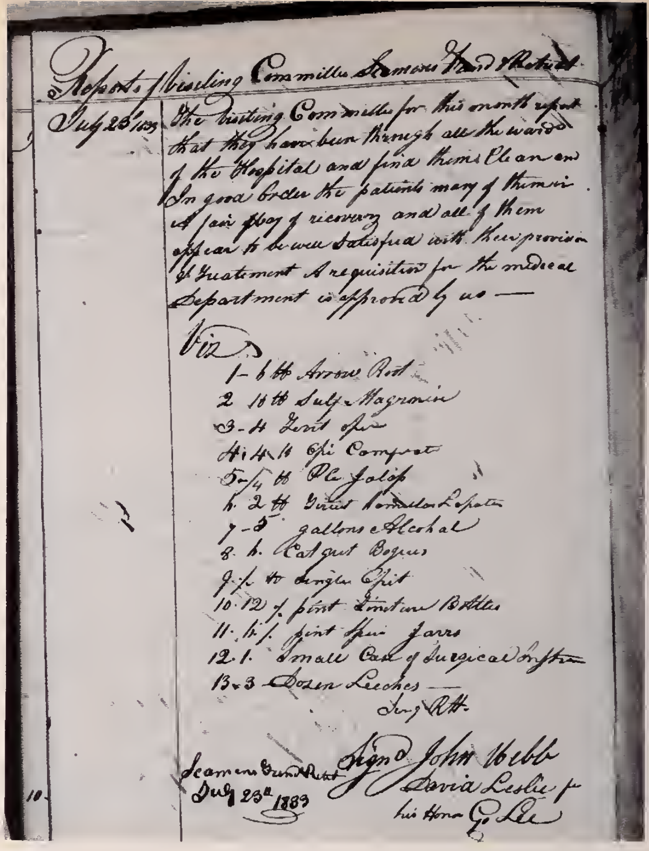
John Thompson—Rheumatismus mercurialis-age 26-born in Philadelphia-arrived four months ago in brig from Rio de Janeiro. About 3 years ago had a running or scalding. Then a seaman on board the US Frigate Constellation. The surgeon gave him 60 Blue pills and rubbed in Blue Ointment into both groins. The gums became very sore and the teeth loose. Immediately after and ever since the saturation has had distressing rheumatism pains, first in the arms of which he has lost the entire use to such a degree as to approach to a partial state of numbness and paralysis . . . the legs also became the seat of these pains extending from the hips to the toes . . . about five months ago the sternum became severely affected.

Henry Miller—Rheumatismus-age 55-born in Philadelphia-Got in a frolic since his arrival in port during the late wet weather and caught cold producing general soreness and rheumatic pains in the knees and shoulders.

Jacob Graver—age 30-born in New Orleans-arrived in brig from Carthagena. Was beating off the coast for 17 days before getting in during a gale and on the day of entering the port was frost bitten in all the toes of the left and the great toe and rest of the right foot.

James Jones—ulcus a porus-age 26-born on the Island of Madeira. Arrived on schooner from Trinidad about 4 months ago. The middle of the left leg anteriorly over the shin became inflamed with a small sore which now has increased to the size of a dollar . . . having all the usual characteristics of ulcerations in this part caused by rum drinking-what I call a regular rum leg. The patient is at present in a state of partial intoxication.

The treatments prescribed and recorded in the 1832 volume of case histories and the lists of supplies ordered by the Chief Physician are indicators of the early medical care available at the hospital during its first years. Intermittent fever cases received, for example, "Tel Sulph Quin" (quinne sulfate), "Tel Sulph Morph" (Morphine Sulfate), emetics were administered and the patients were bled; abscesses were lanced and "muriate ammonia" (ammoniated mercury) applied; urethritis was treated with "Mist Sin" (mixture senna); for dysentery an absence of liquid food and a diet of hard boiled biscuits was prescribed; strong lemonade, "chlor calc" (calcium chloride), "Syr Sarsaparilla" (syrup sarsaparilla), and wine were ordered for scurvy; frostbite and ulcus (ulcers) were treated with poultices of flaxseed oil and yeast; "ulcus a potu" and contusions both received "Mist Senna" (mixture senna), a medicine frequently prescribed for a wide variety of ailments. Bleeding was often ordered and for a range of ailments: for "hydrops" (dropsy), "contusio" (contusions), and fracture, for example. The Chief Physician's biweekly order always included a request for two to four dozen leeches (Fig 7). Of the operations performed, amputations appear to have been relatively frequent.[3]



Report of Visiting Committee Seamens Fund & Retreat

July 23d 1833 The Visiting Committee for this month report that they have been through all the wards of the Hospital and find them Clean and In good order the patients many of them in A fair way of recovery and all of them appear to be well Satisfied with their provision & Treatment A requisition for the medical Department is approved by us —

Viz

1- 6 lb Arrow Root
2 10 lb Sulf Magnesia
3- 4 [illegible]
4- 4 lb [illegible] Compost
5- 1/4 lb Pulv Jalap
6. 2 lb [illegible]
7- 5 gallons Alcohal
8. 6. Catgut Bogies
9. 1/2 lb Single [illegible]
10. 12 1/2 pint Tincture Bottles
11. 6 1/2 pint [illegible] Jars
12. 1. Small Case of Surgical Instru
13. 3 Dozen Leeches —

Seamens Fund Retreat
July 23d 1833
Signd John Webb
David Leslie
his Hon G Lee

10

FIGURE 7. Physician-in-Chief's order for medical supplies, 1833 (Credit, US Public Health Service Archives, Staten Island, NY).

In 1844, for example, there were 1,330 admissions, the average length of stay was 26 days, and the average daily census in the hospital was 107. Discharged cured totaled 1,197; relieved, 105; and by request, 40. In descending order of incidence the diseases diagnosed were as follows: Syphilis, 243; Rheumatism, 160; Gonorrhoea, 145; Febris Intermittens, 103; Ulcers, 86; Asthenia, 62. Of the 40 mortalities, 17 were attributed to "chronic" phthisis (tuberculosis). The relative standing of these diseases essentially did not change in succeeding decades, and phthisis produced the highest mortality amongst the chronic diseases, while pneumonia (and other lung-related diseases) ranked high among the deaths due to acute disease. In 1845, however, the epidemic of typhoid fever claimed the most lives.

Dr Anderson, in his annual report to the legislature, provides what appears to be the first summation of the hospital's mission, including the issue of long term care:

> This being the only institution in the country which affords a permanent resting-place to the merchant seamen, when prevented by the slow inroads of the numerous organic diseases, to which his life of hardship and of toil exposes him, from following any longer

> his laborious profession, he naturally turns towards it as the only retreat which legislation has provided.
>
> Hence, while we have a fair proportion of recent and acute cases within our walls, the number of these disabled, superannuated, and invalid ones is continually increasing, no other provision having been made for them, after the four months to which they are entitled in the United States Hospital, and which is the equivalent they receive for the twenty cents per month tax paid to the government.

Of particular interest in the hospital's early medical history is the treatment of the venereal diseases. There was a high incidence among those admitted, an observation confirmed by reports in the mid-1840s when it accounted for the largest number of annual admissions, a statistic that remained unaltered in the ensuing decades. The most commonly prescribed treatment, one used since the 16th century, was a mercury compound in the form of "blue pills" or applied ointments.[6] Because of the adverse drug reactions of "sore gums" and loss of teeth, Dr Townsend, the chief physician, was adamantly opposed to this therapy. The case histories of those affected, which he compiled, often are exceptionally lengthy; descriptions of symptoms and the effects of earlier treatment are described in detail. Dr Townsend's opposition to the treatment methods then in use was frequently and eloquently expressed:

> This system of malpractice (has) been the source of so much mischief and misery to those who have been the victims of it-all the presumed malignancy which has been ascribed to the syphilitic poison being in my opinion purely and exclusively the product of the mineral erroneously employed for the cure. What is still more unfortunate, the greater the ravages which have thus been made upon the constitution and organization, the greater has been the necessity, according to this false therapeutic of continuing the employment and increasing the quantity of that very metallic poison which has created the malignant and desolating symptoms it is intended to remove, thus adding fuel to the fire and ultimately destroying the life of the patient.[3]

In 1832, in place of mercury pills and ointments, a combination of other medicines were ordered by Dr Townsend and his assistant, which included senna, colchicine, and cubeb, in a variety of salves and lotions.

Additional commentaries in the case histories, underlinings, and marginal notations in key passages suggest Dr Townsend's investigations of venereal disease; although still in the embryonic stage (and abruptly terminated by his early dismissal), this might be considered the hospital's first research effort-a particularly appropriate one for the hospital at which, more than 100 years later, pioneering research in this field was to occur.

Daily Life of a Patient

The seaman's spiritual and moral state was no less the hospital's concern than his physical condition. The position of Chaplain had been established by the original bylaws and was one continuously occupied. Sabbath observances in the chapel, weekly prayer meetings, and services for the dead were among his formal responsibilities. The Chaplain's reports, included as a prominent feature of the hospital's annual reports, noted extensive proselytizing as well in his efforts to turn the hospital's patients away from their intemperate life of sin.

For the patients, a reading room had been formed and contained the daily papers from the city donations of books; one of the permanent patients acted as librarian.

Patients' daily lives were governed by bylaws (presented in the annual report) which defined appropriate behavior:

> No patient under medical treatment shall go out of the hospital yard, or enter any ward other than his own, the kitchen . . . without the permission of the physician.
>
> No patient shall play at any game of hazard . . . or introduce any spiritous liquors . . .
>
> No patient shall smoke in the house, except by consent of the physicians.
>
> Patients not under medical treatment, and such as, in the opinion of the physician, shall assist in nursing others, washing and cleaning the wards, and be employed in such other services as the Physicians or Superintendent may require.
>
> Patients are recommended as far as circumstances will permit, frequently to attend to reading the Holy Scripture.
>
> If any patient shall not conform to these regulations, or shall use profane or indecent language, get drunk, create disturbances, or behave rudely or indecently, the Superintendent may cause such person to be confined in the apartment provided for that purpose until the Visiting Committee shall further dispose of him.

Denouement of the Retreat

Annual admissions to the Seaman's Retreat peaked in 1852 at 3,150, and in 1853 began to decline; the hospital's existence as the Retreat was terminated in 1882. One contributing factor was the increase, after many years of effort by the Governors of New York Hospital, in admissions of seamen under federal coverage to hospital's in the Port of New York from 100 to 200 in 1852. However, the principal cause of the Retreat's decline was a decrease in revenues that resulted from several challenges to its legislative foundation. For example, the Retreat Trustees opposed the state legislation enabling construction of the Mariners Family Home which occupied five acres of the hospital's farmland and was a refuge for the distressed families of seamen, and opposed the use of seamen's tax funds for these purposes. In addition, the passenger tax was declared unconstitutional, and the revenues from this fund could not be used by the Retreat. The Civil War and the resulting absorption of merchant seaman by the Navy caused early declines in most admissions except for "fevers" (malaria, typhoid, and remittent cases). Contracting with the federal government as a maritime hospital charged the character of the institution.

References

1. Moffatt T: "A brief history of the Seaman's Retreat, Staten Island, from its origin in the year 1831 to the present time by its physician-in-chief." *New York*, 1863.
2. "Cornelius Corson of Staten Island, County of Richmond, farmer and people of the State of New York," Richmond County, *Liber of Deeds*, Vol T, p 165, 1831.
3. "United States Public Health Service Hospital, Staten Island, NY Archives." Department of Buildings and Grounds, 1831–1851.
4. New York State: "An act to provide for sick and disabled seamen, 1831." New York, Board of Trustees, Seaman's Retreat, 1845.
5. Hasse AR: *Index of economic material in the documents of the States of United States: New York, 1789–1904*. New York, Kraus Reprint Corp, 1965.
6. Clarke CW: *Taboo: The story of the pioneers of social hygiene.* Washington, DC, Public Affairs Press, 1961.

Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder?

Steven J. Turchan, MD; Valerie F. Holmes, MD; Charles S. Wasserman, MD

Although the term post-traumatic stress disorder (PTSD) is relatively new to psychiatry,[1,2] the concept that trauma may lead to psychological sequela has long been known. There is extensive literature written about combat-related psychological stress and an increasing body of information about psychiatric responses to other types of disaster. Helzer et al[3] found a 1% lifetime prevalence of PTSD in a general population study. Higher prevalences were found in civilians exposed to physical attack (3.5%) and among wounded Vietnam veterans (20%). Breslau et al[4] reported a 9.2% lifetime prevalence of PTSD in young adults, increasing to 24% in those who had previously experienced a traumatic event.

Eighty percent of the survivors of the West Virginia Buffalo Creek flood reported symptoms of PTSD.[5] Weisaeth[6] reported that over 80% of those who narrowly escaped from a major Norwegian industrial disaster had significant symptoms of PTSD, most within the first week after the incident. A four-year follow-up of the Chowchilla school bus kidnapping revealed that 100% of the children involved exhibited post-traumatic pathology.[7]

Studies on the pharmacotherapy of PTSD vary significantly in a number of areas including the presence of controls, sampling characteristics, selection of outcome measures, duration of treatment, and the dosages and types of medications used (ie, tricyclic antidepressants [TCAs], monoamine oxidase inhibitors, psychostimulants, lithium, carbamazepine, neuroleptics, β-blockers, and benzodiazepines).[8-16] TCAs are effective in decreasing the avoidant features (emotional numbing, distancing from loved ones, active suppression of memories of the trauma) and, to a lesser degree, the intrusive (nightmares, flashbacks, involuntary recollections) features of PTSD.[16,17]

An intriguing question is when to prescribe a TCA in a trauma patient. Should it be only after the patient meets all DSM-III-R criteria for PTSD, including the one month duration of symptoms, or should treatment begin earlier? Does immediate treatment, especially in suspected high-risk patients, help alleviate the symptoms of PTSD and prevent comorbidity, such as depression? We present two survivors of the January 26, 1990, Labianca airline crash on Cove Neck, Long Island, both experienced extensive physical injuries, prolonged entrapment and witnessed death first hand. Both received supportive and behavioral therapies from the same therapist; however, the timing of pharmacologic intervention differed.

Case Reports

Case 1. A 54-year-old with a history of lung cancer, alcohol abuse, and anxiety; for the latter, he had received benzodiazepines on an as-needed basis. He had had episodic periods of depression that never required medication and one episode of hypomania for which he briefly received lithium. He had resumed drinking two to three alcoholic beverages per day two weeks before the crash. Eight days after the crash, psychiatric consultation was requested because of suicidal ideation, explosive outbursts, disturbed sleep, and poorly controlled pain.

The patient had sustained bilateral femoral fractures and open fractures of both lower extremities and had undergone several surgical procedures. There was no head trauma or loss of consciousness. A psychiatrist' initial examination found him to be well-groomed and meticulous in appearance; his movements were restricted secondary to the injuries. He complained of nightmares, night terrors, and flashbacks of the crash. He had an increased startle response; appeared dysphoric and anxious; and was irritable, hostile, and argumentative. He was extremely demanding of staff and had great difficulty tolerating any pain.

Before the accident, he had difficulty coping with stress. He was recently separated from his second wife, his business was failing, and he had virtually no support system. Because of his past psychiatric history, the severity of his presentation, his current psychosocial stressors, and the presence of signs suggestive of PTSD, this patient appeared at high risk for developing further psychiatric complications; early psychopharmacologic intervention was begun. He was given amitriptyline (titrated to 150 mg/day over 1 week) and hydromorphone for pain. Treatment also included supportive and behavioral therapies, relaxation exercises, and desensitization. Within two weeks, the patient responded well to the combined therapies. The anxious and depressive complications frequently seen in PTSD did not occur.

Case 2. A 24-year-old with no previous psychiatric history was described as a successful, well-liked, and well-adjusted accountant who played semi-professional soccer. He had sustained injuries similar in severity to those of the first patient with bilateral pelvic fractures, an open fracture of the right ankle, and multiple serosal tears of the small and large bowels. Family or friends were always at the bedside.

Psychiatric consultation was requested four days after the crash to assess flashbacks, intrusive thoughts of the accident, nightmares, and disturbed sleep. The patient had an increased startle response and appeared mildly dysphoric. He was placed on lorazepam for sleep and hydromorphone for pain control. Because he had no psychiatric problems before these injuries, minimal affective symptoms, and a strong support system, antidepressant pharmacotherapy was not instituted. He received the same supportive and behavioral therapies as the first patient.

Although this patient showed initial reduction of symptoms, over the next four weeks the flashbacks began to intensify. There were occasional dissociative episodes, worsening anxiety, nightmares, and increased feelings of detachment and numbness. He refused to continue with desensitization and relaxation treatments. He developed a worsening self-image, feelings of hopelessness, helplessness, and guilt. His sleep and appetite decreased. At this point, he met criteria for both PTSD and major depression and was placed on nortriptyline (titrated to 100 mg/day). Shortly thereafter he was discharged from the hospital. Communication with his outpatient health care professionals revealed a positive response to nortriptyline within four weeks of discharge with improved self-image, appetite, and sleep as well as resolution of guilt, hopeless, and helpless feelings.

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This paper was presented in part at the 37th annual meeting of the Academy of Psychosomatic Medicine, Phoenix, Arizona, November 15–18, 1990.

DISCUSSION

The DSM-III-R criteria[2] for PTSD are well known. Biological research on PTSD has focused on the following areas: alterations in sympathetic arousal;[11–12,15,18–19] disturbance in the hypothalamic-pituitary-adrenocortical (HPA) axis;[12,20–22] endogenous opioid activity;[11,19] limbic system kindling;[11–12,14] and sleep physiology.[10,12,23]

Sympathetic Nervous System. In humans, the biochemistry of overwhelming trauma results in autonomic and extrapyramidal symptoms consistent with catecholamine depletion: masked facies, decreased eye blink, cogwheel rigidity, postural flexion, and coarse tremor of the extremities. A relative norepinephrine depletion hypothetically leads to decreased motivation, decreased occupational functioning, and global constriction.[11] Trauma-induced norepinephrine depletion may lead to adrenergic hypersensitivity, and PTSD symptoms may reflect tonic underactivity of norepinephrine-mediated behavior with aphasic overactivation of norepinephrine-driven behavior after transitory stimulation.[19]

The locus ceruleus is the primary source of noradrenergic innervation of the limbic system, cortex, cerebellum and, to a lesser extent, the hypothalamus. These areas participate in a hierarchical control over the autonomic nervous system and with the stress response.[11,19] The noradrenergic system is also intimately involved in short-term memory.[19] Connections of the locus ceruleus with the hippocampus, amygdala, and temporal neocortex are said to provide a "neurophysiological analogue of memory."[19] Noradrenergic depletion causes decreased memory storage; enhancement of noradrenergic activity with norepinephrine stimulating agents improves memory retention.[19] van der Kolk et al[19] showed that individuals had markedly decreased short-term memory for emotionally neutral stimuli during the nonarousal phase of PTSD compared with the same subjects during states of relative hyperarousal. In other words, highly charged emotional events (which stimulate the noradrenergic system) are easily remembered. Long-term potentiation of locus ceruleus pathways is seen as a possible cause of flashbacks (repetitive intrusive reliving of the trauma) and of the "photographic" quality of traumatic nightmares, as opposed to the less precise quality of more ordinary dreams or nightmares.[19]

These findings are also supported by data from studies of clonidine and yohimbine. Clonidine, an α_2-adrenergic agonist, inhibits central norepinephrine release. By blocking α_2-receptors in the locus ceruleus, clonidine down-regulates locus ceruleus activity, thereby decreasing heart rate, irritability, hyper-reactivity, and motor restlessness in PTSD.[24] Yohimbine, an α_2-adrenergic antagonist, has been shown to exacerbate the symptoms of PTSD.[25,26]

Trauma alone can have long-lasting effects on subsequent responses to stress and brain noradrenergic systems. Perry[24] examined the effects of traumatic stressors on the development of the autonomic nervous system in abused children and adolescents and found increased sympathetic tone and reactivity (higher baseline heart rate and blood pressure and down-regulation of α_2-receptors) after severe abuse, especially when the abuse occurred at or before age three. Trauma at any age may result in permanent autonomic dysregulation or central nervous system dysfunction with regard to affect modulation and impulse control.

HPA Axis. Psychophysiologic studies in man have shown peripheral alteration in the HPA axis in the form of low 24-hour urinary cortisol measurements in chronic PTSD and increased glucocortical receptor number on circulating lymphocytes; this suggests altered negative feedback at one or more levels of the HPA axis.[20–22] In addition, these studies have shown marked increases in 24-hour urinary epinephrine and norepinephrine (suggesting increased sympathetic nervous system activity), decreased platelet monoamine oxidase activity, and decreased platelet α_2-adrenergic receptor number.[27]

It is well known that TCAs block reuptake of various neurotransmitters such as norepinephrine and serotonin. One might hypothesize that TCAs relieved the dysphoric and anxious symptoms and prevented major depressive symptoms from occurring in this patient by stabilizing such neurotransmitter mechanisms within the sympathetic nervous system and/or HPA axis.

Endogenous Opioid System. Animals exposed to inescapable shock develop analgesia when re-exposed to a subsequent stressor within a brief period of time.[11,19] This response is mediated by endogenous opioids and is reversible by naloxone. Prolonged stress activates brain opiate receptors in a manner analogous to repeated applications of exogenous opiates.[11,19] Both naloxone and termination of stressful stimuli produce opiate withdrawal symptoms.[11,19] Therefore, severe chronic stress may result in a physiologic state resembling opiate dependence.[11,19]

In humans, an increase in plasma β-endorphins occurs after stress and in marathon runners and induces a centrally mediated analgesic response.[28] It is likely that re-exposure to traumatic situations in humans evokes an endogenous opiate response analogous to that seen in animals. Re-exposure to stress may have the same effect as the temporary application of an exogenous opiate. Metencephalins are increased in some patients who habitually mutilate themselves.[11,19] The increase in β-endorphins after stress may account for the sense of calm in the face of re-exposure to stress that is reported by many traumatized individuals.[11,19] Some individuals with PTSD voluntarily re-expose themselves to trauma because it produces a paradoxical calm. Although endogenous opioid release following re-exposure to stimuli may result in a temporary sense of control, it may also lead to withdrawal symptoms manifested by sleep disturbances, hyper-reactivity, and explosive outbursts.[11,19]

The reciprocal relationship between opioids and the noradrenergic system is well established. The locus ceruleus appears to mediate the opiate withdrawal syndrome. Encephalins are found in the locus ceruleus, hypothalamus, and limbic system, with the highest concentrations in the amygdala. Watson et al[22] postulated that PTSD involves an imbalance between noradrenaline and opioid release in the area of the locus ceruleus. Opioid depletion may remove an effective opioid inhibition of the locus ceruleus neurons so that noradrenaline is released, leading to relative catecholamine excess. The neurophysiologic changes with prolonged extreme (or acute catastrophic) stress may be poorly reversed due to ongoing imbalance between opioids and catecholamines and may contribute to the chronicity of PTSD.[28] There is a striking parallel between the symptoms of opiate withdrawal and the hyper-reactive symptoms of PTSD. Symptoms of opiate withdrawal have been effectively treated with clonidine,[29] which as previously mentioned, also helps to control symptoms of PTSD.

TCAs may enhance endogenous opioids by potentiating synergistic serotonergic mechanisms and by actual binding to opiate receptors in the brain.[12,30] Stable TCA plasma levels could theoretically prevent the need for re-exposure to trauma by some patients to stimulate endogenous opioid release. As previously mentioned, TCAs are known to affect noradrenaline. They may thus play a therapeutic role in patients at risk for PTSD by mediating in some fashion the reciprocal relationship between the noradrenergic system and opioids. Even though both patients received opiates for pain control, the addition of the TCA may have provided better analgesia for the one patient (Case 1), thus allowing him to take a more active part in his medical care and psychiatric management.

Limbic System Kindling. van der Kolk[11] posited that kindling leading to lasting neurobiological and behavioral changes is produced by repeated traumatizations, such as child abuse or by single trauma followed by intrusive re-experiences. Others have suggested that chronic sympathetic hyperarousal may kindle limbic nuclei in the same way as cocaine,[12] causing a stable neurobiological alteration and thus accounting for the frequency of neurologic abnormalities in trauma victims. TCAs could theoretically play a role in preventing or disrupting a kindling phenomenon by stabilizing sympathetic hyperarousal.

Sleep Physiology. Friedman[12] summarized abnormal sleep findings from several studies and found that there may be a unique biological pattern to sleep and dreaming in PTSD. Increased rapid eye movement (REM) latency, decreased REM and stage 4 sleep, lowered sleep efficiency, and disturbed dreaming (traumatic nightmares that do not correspond with either REM-related dream anxiety attacks or stage 4 night terrors) were reported.

Ross et al[23] reviewed a number of sleep disturbances that occur in PTSD, noting that anxiety dreams and nightmares are reported with higher frequency. PTSD dreams are usually vivid and frequently have elements of the traumatic scene, themes of death, dying, and threats to the self, often accompanied by the same emotions experienced at the time of the trauma. The authors suggest that the post-traumatic anxiety dreams resemble night terrors, in that they occur early in the night and are often accompanied by gross body movements (features generally not associated with REM sleep), and thus they hypothesize that the disturbed sleep of PTSD may represent a new class of parasomnias.

TCAs increase REM latency and decrease REM sleep possibly helping to decrease nightmares, although as noted, the sleep disturbance in PTSD is not fully understood. Perhaps for the patient in Case 1, the nightmares were quickly controlled and overall sleep quality was improved by the addition of the TCA.

In summary, one could make a case for the efficacy of TCAs in PTSD on the basis of any number of their biological activities that would complement the biological mechanisms discussed above. These include alterations in sympathetic arousal, disturbances in the HPA axis, effects on endogenous opioid activity, effects on limbic system kindling, and effects on sleep physiology. One could then hypothesize that the early use of TCAs may help prevent some of the biological disturbances described in PTSD, or perhaps modify their intensity and/or duration.

We have described a trimodal approach of patient care in PTSD, including supportive therapy, behavioral interventions, and pharmacotherapy. However, because of the different premorbid histories and affective presentations of the two patients presented here, TCA pharmacotherapy was introduced at different times. Because the first patient had poor premorbid functioning, minimal support system, and numerous personal stressors, he was placed immediately on TCA therapy. He responded very well, with a decrease in symptoms of PTSD. The second patient did not initially receive a TCA because of his good premorbid functioning, minimal affective symptoms, and strong support system. In spite of receiving supportive and behavioral therapies, he developed both PTSD and major depression. He was unable to proceed with the behavioral therapy and required antidepressant pharmacotherapy.

Individuals with PTSD are at risk for comorbid psychiatric disorders which may decrease treatment response.[16] Hence, the question raised is whether the early use of a TCA helps to prevent the development of PTSD and its associated comorbid illnesses (eg, depression) in individuals who would be considered by most to be at high risk. Although it is difficult to define who is at risk, immediate TCA therapy in an individual who has significant signs and symptoms of PTSD (even before meeting the one month criteria set forth in DSM-III-R) may be protective against the anxious and depressive complications of PTSD and allow earlier intervention and better response to traditional behavioral and supportive therapies. Controlled research needs to be done on the timing of pharmacologic intervention in the treatment of PTSD.

REFERENCES

1. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, ed 3. Washington, DC, APA, 1980.
2. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, ed 3, revised. Washington, DC, APA, 1987.
3. Helzer JE, Robins LN, McEvoy L: Post-traumatic stress disorder in the general population. *N Engl J Med* 1987; 317:1630–1634.
4. Breslau N, Davis GC, Andreski P: PTSD: Risks for event exposure and syndrome. *APA 1990 New Research Program and Abstracts*, Abs NR275, p151.
5. Titchener JL, Kapp FT: Disaster at Buffalo Creek: Family and character change at Buffalo Creek. *Am J Psychiatry* 1976; 133:295–299.
6. Weisaeth L: The stressors and the post-traumatic stress syndrome after an industrial disaster. *Acta Psychiatr Scand* (supp355) 1989; 80:25–37.
7. Terr LC: Chowchilla revisited: The effects of psychic trauma four years after a school-bus kidnapping. *Am J Psychiatry* 1983; 140:1543–1550.
8. Southwick SM, Yehuda R, Giller EL, et al: Antidepressant treatment of PTSD: A meta-analysis. APA 1990 New Research Program and Abstracts, Abs NR479, p231.
9. Bleich A, Siegel B, Garb R, et al: Post-traumatic stress disorder following combat exposure: Clinical features and psychopharmacological treatment. *Br J Psychiatry* 1986; 149:365–369.
10. Friedman MJ: Status report: Clinical psychopharmacology of PTSD. Syllabus American Psychiatric Association, 143rd Annual Meeting, 1990, #54E, pp204–205.
11. van der Kolk BA: The drug treatment of post-traumatic stress disorder. *J Affective Disord* 1987; 13:203–213.
12. Friedman MJ: Toward rational pharmacotherapy for post traumatic stress disorder: An interim report. *Am J Psychiatry* 1988; 145:281–285.
13. Sonnenberg SM: Victims of violence and post-traumatic stress disorder. *Psychiatr Clin North Am* 1988; 11:581–590.
14. Brodsky L, Doerman AL, Palmer LS, et al: Post traumatic stress disorder: A tri-modal approach. *Psychiatr J Univ Ottawa* 1987; 12:41–46.
15. Davidson J, Walker JI, Kilts C: A pilot study of phenelzine in the treatment of post-traumatic stress disorder. *Br J Psychiatry* 1987; 150:252–255.
16. Davidson J, Kudler H, Smith R, et al: Treatment of post-traumatic stress disorder with amitriptyline and placebo. *Arch Gen Psychiatry* 1990; 47:259–266.
17. Frank JB, Giller EL, Koster TL, et al: A randomized clinical trial of phenelzine and imipramine for post-traumatic stress disorder. *Am J Psychiatry* 1988; 145:1289–1291.
18. Kolb LC: A neuropsychological hypothesis explaining posttraumatic stress disorders. *Am J Psychiatry* 1987; 144:989–995.
19. van der Kolk B, Greenberg M, Boyd H, et al: Inescapable shock, neurotransmitters, and addiction to trauma: Toward a psychobiology of post-traumatic stress. *Biol Psychiatry* 1985; 20:314–325.
20. Charney DS, Krystal JH, Southwick SM: Animal models for PTSD. Syllabus APA 143rd Annual Meeting, 1990, #54A, p 204.
21. Southwick SM, Yehuda R, Perry BD, et al: Sympathoadrenal dysfunction in PTSD. Syllabus APA 143rd Annual Meeting, 1990, #54D, p 204.
22. Watson IPB, Hoffman L, Wilson GV: The neuropsychiatry of post-traumatic stress disorder. *Br J Psychiatry* 1988; 152:164–173.
23. Ross RJ, Ball WA, Sullivan KA, et al: Sleep disturbance as the hallmark of posttraumatic stress disorder. *Am J Psychiatry* 1989; 146:697–707.
24. Perry BD: Adrenergic receptors in child and adolescent PTSD. *Syllabus APA 143rd Annual Meeting*, 1990, #115D, p 291.
25. Krystal JH, Southwick SM, Charney DS: Yohimbine effects in PTSD patients. *Syllabus APA 143rd Annual Meeting*, 1990, #115E, p 291.
26. Southwick SM, Krystal JH, Charney DS: Yohimbine in PTSD. *APA 1990 New Research Program and Abstracts*, Abs NR478, p230.
27. Yehuda R, Perry BD, Southwick SM, et al: Platelet α_2 receptor binding in PTSD, generalized anxiety disorder and major depressive disorder. *APA 1990 New Research Program and Abstracts*, Abs NR286, p155.
28. Fairbank JA, Nicholson RA: Theoretical and empirical issues in the treatment of post-traumatic stress disorder in Vietnam veterans. *J Clin Psychol* 1987; 43:44–55.
29. Gold MS, Pottash AC, Sweeney DR, et al: Opiate withdrawal using clonidine: A safe, effective, and rapid nonopiate treatment. *JAMA* 1980; 243: 343–346.
30. Biegon A, Samuel D: Interaction of tricyclic antidepressants with opiate receptors. *Biochem Pharmacol* 1980; 29:460–462.

Progressive pulmonary thromboembolism in association with HIV disease

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Several cases of primary pulmonary hypertension have been reported in patients with acquired immunodeficiency syndrome (AIDS); all were of the classic plexiform variety.[1,2] The patient reported here had pulmonary hypertension with evidence of widespread thromboembolism.

CASE REPORT

A 26-year-old woman was admitted to the hospital with complaints of pain and swelling of the right upper extremity for four days. She also described increasing shortness of breath and progressive reduction in effort tolerance since early 1989. Her medical history was unremarkable. She was married and had two full- term normal deliveries. In 1983, she had a first trimester abortion. She denied use of oral contraceptives. She denied intravenous drug abuse or sniffing cocaine. There was no history of trauma. Her former husband had died of AIDS.

On examination, the patient was in mild respiratory distress, with a blood pressure of 100/70 mm Hg and a heart rate of 90 beats/min and regular. She was afebrile, fully alert, and oriented to time, place, and person. There was diffuse swelling of the right supraclavicular fossa and right upper extremity. The limb was bluish in color and diffusely tender. The brachial and radial artery pulsations were well felt. The neck was supple, and there was no lymphadenopathy. The jugular venous pulse was elevated. Examination of the heart revealed a right ventricular heave. The pulmonic component of the second heart sound was loud. There was a loud third heart sound at the left parasternal border, a grade II/VI ejection systolic murmur in the pulmonic area, and a grade III/VI pansystolic murmur in the tricuspid area increasing in intensity on deep inspiration. The lungs were clear. Tender hepatomegaly and bilateral pitting 4+ pedal edema were present.

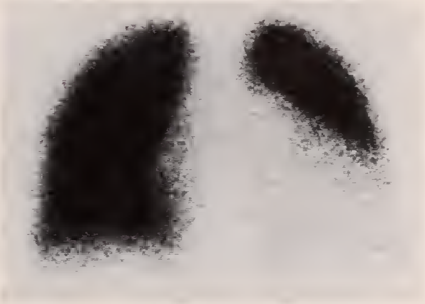

FIGURE 1. Perfusion lung scan showing cardiomegaly.

The laboratory data revealed a normal hemogram, electrolyte profile, and coagulation screen. The electrocardiogram showed regular sinus rhythm, incomplete right bundle block, and p-pulmonale. The chest film revealed cardiomegaly with clear lung fields. Ventilation/perfusion scanning of the lungs was normal (Fig 1). Pulmonary function tests showed normal lung volumes and flow rates. Diffusion was only mildly impaired (75% of estimated, with a normal lower limit of 80%).

A contrast venogram revealed obstruction in the right axillary vein. A computed tomography (CT) scan of the chest with contrast failed to show macrothrombi in the major pulmonary vessels. An echocardiogram showed a dilated right ventricle and right atrium with tricuspid regurgitation. The mitral and aortic valves were normal. There was no intracardiac shunt. Right heart catheterization revealed a right ventricular pressure of 65/10 mm Hg and a pulmonary artery pressure of 65/30 mm Hg. Oximetry showed oxygen saturation between 55% and 57% in the right atrium, right ventricle, and inferior vena cava.

She was given thrombolytic therapy followed by anticoagulation. We diagnosed primary pulmonary hypertension, but the patient refused vasodilator therapy under hemodynamic monitoring. She was empirically treated with oral anticoagulants and calcium channel blockers. Human immunodeficiency virus (HIV) testing proved positive, and the patient was put on the heart-lung transplant waiting list. One year and five months after initial admission, the patent returned with worsening shortness of breath. On the third day of the patient's hospital stay, she suddenly expired.

An autopsy revealed changes of recurrent pulmonary thromboembolism with macro- and microthromboemboli in various stages of organization. The right lower lobe branch was completely occluded by a recent thrombus with fibroblasts in the periphery. Many older thrombi with intimal eccentric collagenization and partial recanalization were noted in the smaller arteries. Colander lesions indicative of multiple recanalized channels were also seen. Medial hypertrophy was minimal in most arteries. Dilatation and plexiform lesions were not identified. The pulmonary veins were normal. No talc or foreign material with associated granulomatous reaction was present (Figs 2,3,4).

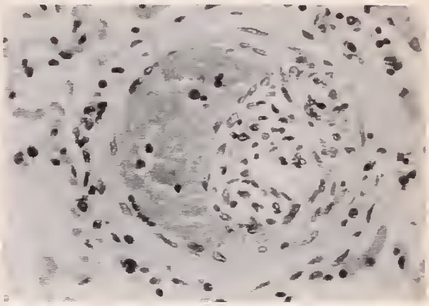

FIGURE 2. High power view of partially recanalized small pulmonary artery (hematoxylin and eosin stain; original magnification ×400).

DISCUSSION

The term "primary pulmonary hypertension" has been applied to three diseases with distinct histopathologic findings.[3] One of these disorders, pulmonary veno-occlusive disease, presents with pulmonary edema, is very rare, and was not a consideration in this patient. She had progressive pulmonary hypertension without left heart or chronic lung diseases but in association with axillary vein thrombosis.

The latter finding raised the strong possibility that this patient had thromboembolic pulmonary hypertension. On the other

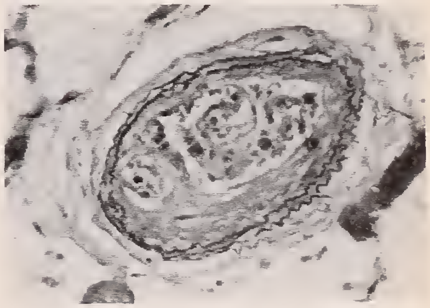

FIGURE 3. High power of recanalized small pulmonary artery with colander lesion (Elastic van giesen stain) original magnification ×400).

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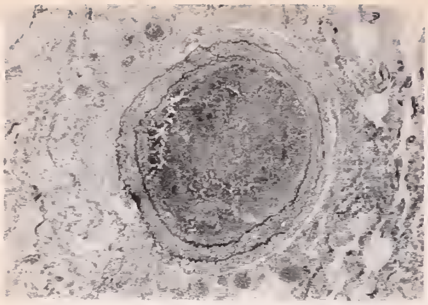

FIGURE 4. Low power view of a large pulmonary arterial branch with recent embolus (Elastic van giesen stain; original magnification ×40).

hand, the normal perfusion lung scan did not support this diagnosis, and so the third entity, plexiform pulmonary ateriopathy, was also considered. The autopsy confirmed the presence of thromboembolism and the absence of plexiform arteriopathy. It is possible that this patient had pulmonary embolism superimposed on plexiform arteriopathy that may have been obscured on histologic evaluation by widespread changes secondary to thrombi.

Primary pulmonary hypertension of the plexiform type has been reported in patients with the AIDS. Although antiphospholipid antibodies have also been demonstrated in these patients, the patients generally do not present with thromboembolic disorders.[4,5]

To our knowledge, the association of AIDS and thromboembolic primary pulmonary hypertension has not been previously reported. Intravenous drug abusers may develop pulmonary vascular disease in association with fibrosis and granulomas within the lungs. This patient was not an intravenous drug abuser and did not have the restrictive pattern and marked decrease in diffusion capacity characteristic of that subset of patients. Moreover, the pathologic findings were not consistent with the effects of intravenous drug abuse.

Also of interest in this case is the essentially normal perfusion lung scan, in spite of evidence of widespread macro- and microthromboembolism. A previous study found abnormal lung scans in eight of nine patients with histologically confirmed embolic pulmonary hypertension.[6] This issue has therapeutic implications. If one cannot rely on the lung scan to rule out the presence of thromboembolism and if a lung biopsy is not performed, it may be necessary to treat some patients with primary pulmonary hypertension with anticoagulants and pulmonary vasodilators.

REFERENCES

1. Coplan NL, Shimony RY, Iochim HL, et al: Primary pulomary hypertension associated with the HIV infection. *Am J Med* 1990; 89:96–99.
2. Goldsmith GH Jr, Baily RG, Brettler DB, et al: Primary pulmonary hypertension in patients with classic hemophilia. *Ann Intern Med* 1988; 108:797–799.
3. Rich S: Primary pulmonary hypertension. *Progress in Cardiovascular Disease* 1988; 31:205–238.
4. Tailla B, Roul C, Fuzibet JG, et al: Antiphospholipid antibodies associated with HIV infection. *Arch Intern Med* 1990; 150:1975.
5. Holland HK, Spivak JL: The hematological manifestations of AIDS. *Baillieres Clin Haematol* 1990; 3:103–114.
6. Rich S, Pietro G, Kieras K: Primary pulmonary hypertension radiographic and scintigraphic patterns of histologic subtypes. *Ann Intern Med* 1986; 105:499–502.

Unexpected reversal of an idiopathic dilated cardiomyopathy and the urgent need for a re-evaluation of the indicators of patient survival

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Idiopathic dilated cardiomyopathy (IDC) is a disease of obscure etiology characterized by high mortality and morbidity. The one-year mortality rate is estimated to range from 25% to 50% with no less than 75% of patients with class IV (New York Heart Association) expiring within that interval.[1] IDC constitutes a major cause of cardiac transplantation, and its clinical course is relatively unaffected by medical intervention. Stabilization and reversal of IDC have been recorded most frequently in the peripartum[2] and alcoholic[3,4] cardiomyopathies. Reversibility has also been documented in select cases of IDC,[5,6] prompting investigations to determine prognostic indicators and the appropriate timing of transplantation. This has resulted in the observation that IDC may be progressive or nonprogressive,[7] with disagreement as to the relative indicators of survival.

CASE REPORT

A 45-year-old man was in his usual state of excellent health until two weeks before admission when an upper respiratory infection developed, characterized by rhinitis, productive cough, myalgias, and dyspnea on exertion. The development of a nonradiating retrosternal chest "tightness" prompted him to visit his private physician, who on finding electrocardiographic changes and signs of congestive failure, recommended admission to the hospital. The patient's medical history was remarkable only for psoriasis for the previous 20 years. He had an approximate 20 pack/year smoking history, admitted to drinking one alcoholic beverage daily, and denied illicit drug use. Drug allergies were denied, and the only medication used was coal tar solution 5% applied to skin lesions four times a day.

The physical examination revealed a well-developed, well-nourished male who appeared anxious but in no acute distress. Vital signs were within normal limits, and an examination of the skin disclosed widespread psoriatic lesions. The neck was supple with positive jugular venous distention to the angle of the mandible. The lungs revealed scattered rhonchi and basilar rales bilaterally. The heart had a regular rate and rhythm without murmurs, rubs, or thrills. There was no S3, but an S4 gallop was heard at the left sternal border. The abdomen was soft and nontender with normoactive bowel sounds. The liver edge was palpable 3–4 fingerbreadths below the right costal margin. There was a positive hepatojugular reflux. The extremities displayed no edema, cyanosis, or clubbing.

A chest radiograph demonstrated cardiomegaly with a cardiothoracic ratio greater than 0.55. There was a mild increase in pulmonary vascular congestion without evidence of consolidation or effusion. The electrocardiogram revealed a normal sinus rhythm, indeterminate axis, left atrial enlargement, low voltage in the limb leads, and a right bundle branch block pattern with a poor R-wave progression in V1 to V5.

The complete blood cell count and the erythrocyte sedimentation rate were within normal limits, and the blood chemistry studies were notable: glucose level, 126 mg/dL; uric acid, 8.8 mg/dL; aspartate aminotransferase, 66 U/L; lactate dehydrogenase, 200 mIU/mL. The serum ethanol level was zero, and viral titers were not ordered.

An echocardiogram revealed biventricular

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enlargement with a generalized hypokinesis and a qualitative ejection fraction of less than 20%. No thrombi, pericardial effusions, or valvular pathology were identified. A resting, first pass, radionuclide cineangiogram confirmed a left ventricular ejection fraction (LVEF) of 17%, cardiac output of 2.8 with a cardiac index of 1.4. Wall motion was diffusely hypokinetic.

After an uncomplicated hospital stay, the patient was discharged on the sixth hospital day on captopril (25 mg/8 hr). He was advised to restrict severly his physical activity, and arrangements were made for cardiac catheterization and endomyocardial biopsy.

Catheterization findings were consistent with a nonischemic dilated cardiomyopathy: pulmonary wedge pressure, 25 mm Hg; right atrial pressure, 13 mm Hg; cardiac output, 3.4 with a 1.7 cardiac index. An endomyocardial biopsy revealed moderate fibrosis, myocellular hypertrophy, and vascular sclerosis consistent with a moderate nonspecific cardiomyopathy. There was no evidence of active myocarditis, and the myofibril volume (Weibel Method) was less than 60%, but greater than 40%.

The patient was started empirically on digoxin (0.125 mg daily), furosemide (40 mg daily), sodium warfarin (5 mg daily), and potassium supplementation in addition to the captopril. The patient was placed on a transplantation list in March 1987 (within six months on diagnosis). Three months later, asymptomatic, nonsustained, ventricular tachycardia developed while he was participating in a cardiac rehabilitation program. He was then readmitted to the hospital for amiodarone loading and transferred to another institution for programmed electrical stimulation. Although no ventricular tachycardia was inducible with the amiodarone, it was recommended by the electrophysiologist that the patient be maintained on the amiodarone until transplantation.

On this medical regimen the patient's clinical status stabilized and gradually improved to the extent that he was able to work part-time. Later, a repeat nuclear study revealed a resting LVEF of 36% increasing to 51% with exercise. Another study the following January showed a rest to stress change from 42% to 56% accompanied by normal wall motion except for mild apical hypokinesis. The amiodarone was discontinued, and a repeat electrophysiology study 12-months later was negative. All the other medications were gradually discontinued except for the captopril and digoxin. The patient has returned to work full-time, and he remains well four and a half years after diagnosis.

DISCUSSION

An extremely important and frequently unrecognized aspect of the natural history of IDC is that a substantial number (20%–30%) of patients demonstrate stability or improvement in their congestive symptoms.[8] The ability to predict accurately which patients are likely to exhibit complete or partial reversal of their disease would obviously be of inestimable clinical value. However, many recognized prognostic markers have not proven adequately discriminatory in the setting of potential transplantation.[9] For example, one study[6] concluded that for patients with ejection fractions between 11% and 19%, survival after transplantation exceeds that of the natural history of IDC. The investigators therefore concluded that surgery should be performed on such individuals as soon as six months after diagnosis. However, a more recent report[9] by the same principal investigator noted that the only factor found more frequently among those who improved was a duration of symptoms of heart failure of less than or equal to 12 months.

This latter finding must be carefully weighed by the clinician in estimating the rate of disease progression and the optimal time for transplantation, since it is clear from another study[10] that patients who are referred for transplantation and who have an ejection fraction of less than 25% deserve aggressive evaluation of their risk factors irrespective of symptomatology. In the patient presented here, a complete reversal of the disease was demonstrated, in spite of initial hemodynamic data that strongly suggested non-survival. Fuster et al[7] found a 97% mortality rate for patients aged 55 or older; 86% mortality rate when the cardiothoracic ratio was 0.55 or greater; 89% mortality rate if the cardiac index was less than 3.0; and an 87% mortality if the pulmonary wedge pressure was greater than or equal to 20 mm Hg. All of these statistics except for the first one apply to this patient. Furthermore, Hatle et al[10] reported a very poor prognosis when there was electrocardiographic evidence of low voltage and left atrial enlargement. Diaz et al[11] reported no survival if the LVEF was less than 25%; and Figulla et al[5] and Keogh et al[9] maintained that hemodynamic status at the time of diagnosis was not strongly prognostic. Likewise, a grim prognosis has been associated with pulmonary wedge pressures of 22[1] and 23[12] mm Hg and a cardiac index less than or equal to 2.1[1,13].

The prognostic value of the endomyocardial biopsy also remains controversial. Although it has been reported[9] that an abnormal biopsy is not related to survival in patients with IDC, such reports refer to histologic characteristics and not to volumetric measurements. It has been reported[5] that a myofibril volume less than 60% results in a 90% sensitivity and a 93% specificity for mortality. Some investigators[14] have found that the prognosis is poor when myocardial fibrosis is widespread, and other investigators[15] dispute this correlation. To further complicate matters, reports[16] of inter-observer variability in the interpretation of biopsy specimens is high, and the reproducibility of this Weibel Method remains to be proven.

It is indeed probable that the dilated cardiomyopathies represent multiple disease processes, and that some patients can do well and enjoy a good quality life without surgical intervention and with minimal medical support. Stevenson et al[13] have postulated that clinical prognosis correlates best with the therapeutic response of the pulmonary artery wedge pressure to medical therapy and to the absence of coronary artery disease. Keogh et al[9] believe that the duration of congestive symptoms is an important factor in survival. Indeed, in the patient described here there was evidence of improvement slightly more than one year after diagnosis. Although serial intracardiac pressures are not available in this case, one may infer a reduction in filling pressures coincident with clinical stabilization. In retrospect, this case demonstrates the unreliability of published prognostic indicators, and the need for an intensive investigation of prognostic factors in IDC, in conjunction with a standardization of the histologic diagnostic criteria.

Acknowledgments. The authors thank Karen Wright, BS, RN, and Joan Schanfeld, BS, RN for their contributions.

REFERENCES

1. Stevenson LW, Fowler MB, Schroeder JS, et al: Poor survival of patients with idiopathic cardiomyopathy considered too well for transplantation. *Am J Med* 1987; 83:871–876.
2. Demakis J, Rahimtoola S, Sutton G, et al: Natural course of peripartum cardiomyopathy. *Circulation* 1971; 44:871–876.
3. Regan T: Alcoholic cardiomyopathy. *Prog Cardiovasc Dis* 1984; 17:141–152.
4. Sacher ML, Siskind SJ, Boal BH: Gated radionculide angiocardiography in the noninvasive evaluation of a partically reversible alcoholic cardiomyopathy. *Am J Med* 1986; 80:1205–1207.
5. Figulla HR, Rahlf G, Nieger M, et al: Spontaneous improvement or stabilization and associated biopsy findings in patients with congestive cardiomyopathy. *Circulation* 1985; 71:1095–1104.
6. Keogh AM, Freund J, Baron DW, et al: Timing of cardiac transplantation in idiopathic dilated cardiomyopathy. *Am J Cardiol* 1988; 61:418–422.
7. Fuster V, Gersh BJ, Giuliani ER, et al: The natural history of idiopathic dilated cardiomyopathy. *Am J Cardiol* 1981; 47:525–531.
8. Kopecky SL, Gersh BJ: Dilated cardiomyopathy and myocarditis: Natural history, etiology, clinical manifestations, and management. *Curr Probl Cardiol* 1987; 12:10:569–647.
9. Keogh AM, Baron DW, Hickie JB: Prognostic guides in patients with idiopathic or ischemic dilated cardiomyopathy assessed for cardiac transplantation. *Am J Cardiol* 1990; 65:903–908.
10. Hatle L, Orjavik O, Storstein O: Chronic myocardial disease: Clinical picture related to long-term prognosis. *Acta Med Scand* 1976; 199:399–406.
11. Diaz RA, Obasohan A, Oakley CM: Prediction of outcome in dilated cardiomyopathy. *Br Heart J* 1987; 58:393–399.
12. Franciosa JA, Wilen M, Ziesche S, et al: Survival in men with severe chronic left ventricular function due either to coronary artery disease or idiopathic dilated cardiomyopathy. *Am J Cardiol* 1983; 51:832–836.
13. Stevenson LW, Tillisch JH, Hamilton M, et al: Importance of hemodynamic response to therapy in predicting survival with ejection fractions less than 20% secondary to ischemic and nonischemic dilated cardiomyopathy. *Am J Cardiol* 1990; 66: 1348–1354.
14. Shirley EK, Proudfit WL, Hawk WA: Primary myocardial disease. Correlation with clinical findings, angiographic and biopsy diagnosis: Follow-up in 139 patients. *Am Heart J* 1980; 99:198–207.
15. Unverferth DV, Fetters JK, Unverferth BJ, et al: Human myocardial characteristics in congestive heart failure. *Circulation* 1983; 68:1194–1200.
16. Baandrup U, Florio RA, Olsen EG: Do endomyocardial biopsies represent the morphology of the rest of the myocardium? A quantitative light microscope study of single versus multiple biopsies with the King's bioptome. *Eur Heart J* 1982; 3:171–178.

Acute emphysematous cholecystitis: A case report and review of the literature

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Acute emphysematous cholecystitis is a rare variant of acute cholecystitis in which gas is produced by infecting bacteria. Typically, on the plain roentgenogram of the abdomen, a characteristic pattern of air is seen in the gall bladder lumen, the wall of the gall bladder, and the pericholecystic area.

Acute emphysematous cholecystitis occurs more frequently in men, with the greatest incidence between 50 and 60 years of age. The incidence of gangrene and perforation is high in acute emphysematous cholecystitis, and the overall mortality rate averages 15%, compared with 4.1% for acute cholecystitis.[1]

We report a case of emphysematous cholecystitis that illustrates the importance of plain abdominal films in the diagnosis of this entity and the role of CT scans for confirmation.

CASE REPORT

A 67-year-old black male was admitted to the VA Medical Center in Castle Point, NY, for evaluation of chest pain, hypertension, abdominal pain, and constipation with occasional diarrhea. The patient had a history of a cerebrovascular accident with left hemiparesis. Subsequently, he became febrile and had increased abdominal pain and leukocytosis.

Examination of the abdomen showed epigastric pain and tenderness with rebound and guarding in the right upper quadrant and a globular mass in the area. The lower abdomen was unremarkable. Radiographic analysis of the abdomen showed globular air around the gall bladder (Fig 1).

The liver profile was normal, and the white blood cell count was 25,000 cells/mm^3. A computed tomography (CT) scan of the abdomen also showed an air fluid level around the gall bladder in the region of the fossa, indicative of emphysematous cholecystitis (Fig 2). At the time of surgery, the patient was found to have acute emphysematous cholecystitis, gangrene, and perforation with cholelithiasis. Approximately 45 cc of cloudy purulent material was aspirated from the gall bladder, yeilding *C perfringens* on culture. The patient responded well to antibiotics and had an uneventful recovery.

DISCUSSION

In 1901, Stolz[2] first described emphysematous cholecystitis at necropsy. He reported on three patients with gas present in the gall bladder and common duct. In 1908, Lobinger[3] described gas in the gall bladder lumen and wall in his operative findings. In 1931, Hegner[4] documented the first preoperative diagnosis of emphysematous cholecystitis by plain abdominal films showing a pear-shaped gas shadow in films of a 62-year-old man with symptoms of acute cholecystitis. In 1958, Edinburgh et al[5] published the 50th case reported in the medical literature of the clinical and pathologic entity then known as acute pneumocholecystitis, pyopneumocholecystitis, or acute gaseous cholecystitis. In 1966, Rosoff et al[6] reviewed 94 cases in the English language literature and added an analysis of ten cases of his own. In 1966, Sarmiento[7] reviewed 105 cases including a brief review of 101 previously published cases and four new cases.

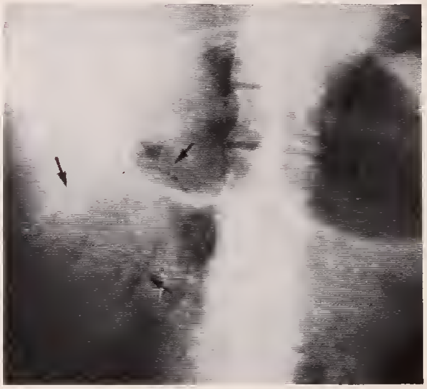

FIGURE 1. Radiograph of the abdomen globular showing air around the gall bladder.

In 1975, Mentzer et al[1] reviewed a total of 164 cases and offered a variety of descriptive synonyms such as pneumocholecystitis, pyopneumocholecystitis, gas phlegmon of the gall bladder, and aerocholecystitis. Since then, more reports have been added to the literature.

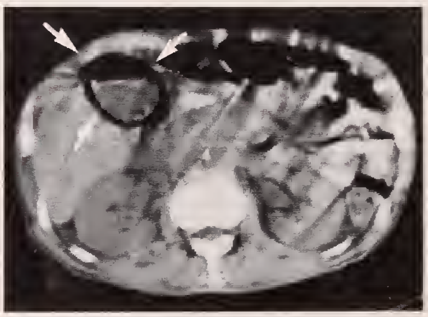

FIGURE 2. CT scan of the abdomen showing air fluid level around the gall bladder in the region of the fossa, indicative of an emphysematous gall bladder.

Emphysematous cholecystitis is more prevalent in men, and acute cholecystitis is more common in women. Sarmitento[7] reported that 73.3% of cases of emphysematous cholecystitis were in men and 26.7% in women. Kune and Birks[8] reported that 27% of cases of acute cholecystitis occurred in men and 73% in women. Sarmiento's[7] review of emphysematous cholecystitis indicated that 74% of patients had gangrene and 21% had perforation. Kune and Birks,[8] reporting on their experience with acute cholecystitis, indicated that 2.5% of patients had gangrene and 4.5% had perforation with a peak age incidence between 50 and 60 years of age.

Gall bladder bile was cultured in 109 cases of emphysematous cholecystitis reviewed by Mentzer,[1] and 46% showed a positive culture for clostridial organisms; 79% were noted to be *C welchii*.

Emphysematous cholecystitis is found primarily in males and in persons with diabetes mellitus. May and Strong[9] suggested a vascular etiology since arteriosclerosis is fairly common among males and those with diabetes mellitus. Vascular occlusion of the cystic artery and its branches are thought to be the major contributory factors of emphysematous cholecystitis. Clostridial organisms would be expected to be more effective invaders in the presence of ischemia. Arteriolar narrowing has been identified in patients with emphysematous cholecystitis. Gangrene, so prominent in this acute process, is usually regarded as a facilitating factor for clostridial infection rather than a result of infection. Finally, the relatively high incidence of cholecystitis after major trauma or surgery with vascular compromise appears to play a major role in the occurrence of emphysematous cholecystitis.

The overall majority from emphysematous cholecystitis noted by Mentzer[1] was 15%, in contrast to 4.1% for ordinary acute cholecystitis. This emphasizes the importance of the differential diagnosis and the early treatment regimen for acute emphysematous cholecystitis.

Graham et al[10] and Kadama[11] noted that bacteria may reach the gall bladder directly from the liver by way of the extensive lymphatic network existing between the two organs. Mason and Hart[12] reported on culturing *C perfringens* from the liver in normal human subjects. In a study involving 305 cases, Edlund, Mollstedt, and Ouchterlong[13] reported that bile obtained at surgery from apparently normal gall

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bladders was sterile. However, a variety of bacteria was cultured from gall bladder bile in 50% of patients with acute cholecystitis and 20% of those with chronic cholecystitis. Popper et al[14] suggested that bile is bactericidal if the bile acid concentration is 70% of normal. McCorkle and Fong[15] speculated that gas-forming anaerobes were extremely virulent in some instances and very inactive in others.

The ideal treatment for acute emphysematous cholecystitis is early cholecystectomy in conjunction with intravenous fluids and antibiotics. The emphasis is on early surgical intervention due to the potential complications of gangrene and perforation.[1,5,6,7] The surgical intervention of choice is cholecystectomy and cholecystostomy if primary surgery is not feasible. The case presented here emphasizes the importance of plain abdominal films confirmatory in establishing the diagnosis, and the use of CT scans.

REFERENCES

1. Mentzer RM, Golden GT, Chandler JG, et al: A comparative appraisal of emphysematous cholecystitis. *Am J Surg* 1975 129:10–15.
2. Stolz A: Ueben gasbildung in den gallenwegen. *Virchow Arch Path Anat* 1901; 165:90–123.
3. Lobinder AS: Gangrene of the gall bladder. *Ann Surg* 1908; 48:72–79.
4. Hegner CF: Gaseous pericholecystitis with cholecystitis and cholelithiasis. *Ann Surg* 1931; 22: 993–1000.
5. Edinburgh A, Geffen A: Acute emphysematous cholecystitis. *Am J Surg* 1958; 96:66–75.
6. Rosoff L, Meyers H: Acute emphysematous cholecystitis. *Am J Surg* 1966; 111:410–423.
7. Samiento R: Emphysematous cholecystitis. *Arch Surg* 1966; 93:1009–1004.
8. Kune GA, Birks D: Acute cholecystitis: An appraisal of current methods of treatment. *Med J Aust* 1970; 2:218–221.
9. May RE, Strong R: Acute emphysematous cholecystitis. *Br J Surg* 1971; 58:453–458.
10. Graham GA, Peterman MG: Further observation on the lymphatic origin of cholecystitis, choledothitis and the associated pancreatitis. *Arch Surg* 1922; 4:35–50.
11. Kadama S: The lymphatics of the extrahepatic biliary passages. *Surg Gynecol Obstet* 1926; 43: 140–146.
12. Mason GC, Hart MS: The Welch-like bacillus in human liver. *J Lab Clin Med* 1940; 25:835–838.
13. Edlund YA, Mollstedt BO, Ouchterlong O: Bacteriological investigation of the biliary system and liver in biliary tract disease correlated to clinical death and mictrostuction of the gall bladder and liver. *Acta Chir Scand* 1958; 116:461–476.
14. Popper H, Shaffner F: *Liver Structure and Function*. New York, Blakiston Davison, McGraw-Hill Co, 1957, 81–82.
15. McCorkle H, Fong EE: The clinical significance of gas in the gall bladder. *Surgery* 1942; 11: 851–868.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

The *New York State Journal of Medicine*

TO THE EDITOR: I read with great interest "An update on the *New York State Journal of Medicine*" by Dr Milton Gordon in the July issue.[1] I fully agree with his conclusion and that of the Committee on Publications, Library, and Archives not to discontinue the *Journal* or replace it with a lesser grade "throw-away" publication.

I have been the New York correspondent for the *British Medical Journal* for the past three years and can assure you that the *Journal* is widely read and respected in the United Kingdom. New York is regarded as a keystone state in medical progress, and the *Journal* is on the cutting edge of current advances.

I was a little surprised to read that out of a total circulation of 27,595, there are only 161 (5.84%) foreign subscriptions. I feel sure that there is scope for increasing the number of foreign subscriptions, especially to foreign medical schools.

Let us recall what Sir Robert Hutchison had to say:[2] "...the amount of writings of a profession is a measure of its vitality and activity, while their quality is a rough indication of its intellectual state."

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1. Gordon M: An update on the *New York State Journal of Medicine*. *NY State J Med* 1992; 92:293–294.
2. Hutchison R: Medical literature. *Lancet* 1939; II:1059–1062.

The future of the *New York State Journal of Medicine*

TO THE EDITOR: I hope the future of the *New York State Journal of Medicine* has not been predestined. My impression of the 1992 House of Delegates meeting was significantly supportive for the continuation of the *Journal*. I hope the task force evaluating the Journal will bring back recommendations to the full house to enable the house to decide the fate.

Today's medicine is often viewed largely in socio-economic terms. Hopefully, the high quality of our *Journal* under its outstanding Editor will be preserved.

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The physically handicapped children's program: A helping hand for children with serious health problems

TO THE EDITOR: Children with serious long-term health problems can often be helped by the Physically Handicapped Children's Program (PHCP). The program is over 70 years old and has helped more than a million children in New York State. Last year alone, it paid for services for more than 7,000 children. In spite of these impressive statistics, many parents and health professionals do not know of its existence. It has been referred to as "the best kept secret in the State."

PHCP's main purpose is to make sure that lack of money does not prevent children with serious health problems from getting the medical services they need. The program is designed to help families who are not covered by Medicaid and who do not have adequate health insurance or private means to pay for the services their children need.

PHCP pays for both diagnostic and treatment services. It covers surgery; examinations in clinics and doctors' offices; speech; hearing, and physical therapy; medicines; braces; hearing aids; and other medical services and equipment. It pays both hospital and doctors' bills.

A common misconception is that the program helps only children with congenital and orthopedic handicaps. This was indeed the case in the first years of the program's existence but is no longer true. Today the program can help any child with a serious physical handicap or chronic illness. PHCP covers both congenital and acquired disabilities of all types.

The second major purpose of PHCP is to ensure that the services for which it pays are of high quality. The state program establishes standards for medical eligibility and for medical services and designates specialists and specialty centers qualified to treat PHCP children.

Each county administers its own PHCP, usually through the county health department. The local programs establish their own standards for financial eligibility, and determine which of the State-authorized services and conditions they will cover. Most county programs elect to cover all eligible conditions and services.

Applications for the help of the PHCP must be made to the program in the county in which the family lives. A list of county PHCP contact persons is available from the Bureau of Child and Adolescent Health of the State Department of Health.

Families and health social services professionals who need more information about the program should call their County Health Department. In New York City, they should call the New York City Health Department's Bureau for Families with Special Needs.

The state PHCP also invites inquiries from physicians, parents, and others interested in services for children with serious chronic physical health problems. PHCP is located in Room 219 of the Corning Tower Building, Empire State Plaza, Albany 12237. The telephone number is (518) 474-2001.

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Choice of surgical procedure for prostatic surgery

TO THE EDITOR: Given the improvements in medical practice and the public's awareness of their right to participate in critical decisions regarding malignant tumors of the prostate, I think that the time has come to seek new approaches when discussing surgery with patients and their families. If physicians have proof that the cancer is still limited to the prostate gland and has not spread beyond it, they have an ethical duty to offer to patients not only standard surgery, namely transurethral resection of the prostate, but also more radical surgery, namely the suprapubic (retropubic) resection of the prostate which carries a higher surgical risk but also provides a much higher hope of cure.

Why shouldn't these patients, who face an implacable disease, be given the right to decide for themselves, between a potential cure with one operation and a poorer outlook with more limited surgery.

It is no longer possible nor permissible, in my judgment, to rely on statistical evidence when advising individuals. In these matters, one individual is as important as a large population.

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Leiomyoma of the nipple

TO THE EDITOR: We report an uncommon tumor of the nipple arising from the muscularis mamillae. During its growth, it involved the entire nipple circumference.

Case Report. A 31-year-old black female presented with a painful mass on her nipple which had been present for six months. It had recently shown a sudden increase in size. No

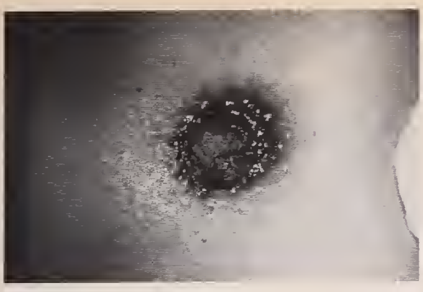

FIGURE 1. Gross appearance of lesion. The lighter colored portion is the tumor.

nipple discharge or any other symptoms were noted. Examination of the breast revealed a 1 cm "growth" protruding from the nipple and attached around its circumference. The rest of the examination was negative (Fig 1).

The lesion was surgically excised. Pathologic examination of the specimen showed the characteristic whorled bundles of benign smooth muscle cells indicative of leiomyomas (Fig 2).

Discussion. Leiomyoma of the nipple is a rare lesion with fewer than 25 cases reported in the literature. In the 99 cases of superficial leiomyoma collected by Stout,[1] there was no report of a mamillary leiomyoma. Of the 54 cases reported by Fisher et al[2], only one originated in the nipple.

The first case was probably described by Virchow in 1854[3] in the journal that bears his name. The second case was also published in that journal by Sokolow, 19 years later.[4] However, the detailed study of the anatomy of the smooth muscle of the nipple giving rise to these tumors was not described until 1916, by Bauer.[5]

FIGURE 2. Microscopic section of the nipple showing dense crossing bands of smooth muscle forming a leiomyoma in subareolar tissue. (hematoxylin and eosin × 13)

There are now thought to be four types of cutaneous leiomyomas, based on their origin:[6] multiple pileoleiomyomas and solitary pileoleiomyomas, which arise from the arrectores pilorum muscle; solitary genital ones' which arise from dartoic/vulvar/mamillary muscle, and solitary angioleiomyomas' which arise from the muscles of veins.

Multiple pileoleiomyomas are more common than solitary ones with the extremities being the primary site followed by the trunk. The average age of patients is 25 years, and a familial incidence has been observed.[2]

These tumors are often clinically mistaken for sebaceous cysts, nevi, fibromas, dermatofibromas, or neurofibromas. They are generally removed and sent to the pathologist labelled as "cyst/tumor/nodule." Nipple tumors that should be considered in the differential diagnosis are adenomas, intracystic papillomas, fibromas, hemangiomas, Paget's disease or even the rare condition known as lymphocytoma benignum Bafverstedt.

Patients often complain of pain, as with other cutaneous leiomyomas,[8] and this may be secondary to contractions of smooth muscle.[1] The other equally common presenting symptom is a mass. Treatment is by simple excision. However, complete removal is essential, as these tumors, although benign, can recur if incompletely resected.[9] Leiomyosarcoma arising in the nipple has been reported by Hernandez.[10] However, malignant transformation of a benign lesion has not been reported.

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2. Fisher ER, Helwig, EB: Leiomyomas of the skin. *Arch Derm* 1963; 88:510–520.
3. Virchow R: Ueber cavernose (erectile) geschwulste und telengiektasien. *Virchows Arch of Path Anat* 1854; 6:525.
4. Sokolow K: Myoma laevicellulare (leiomyoma) der rechte Brustwarze. *Virchows Arch of Path Anat* 1873; 58:316.
5. Bauer T: Zur normalen und pathologischen anatomie und Histologie der menschlichen Brustwarze. Beitr, Z Path, Anat, UZ. *Aug Path* 1916; 233–264.
6. Lever WF, Schamburg-Lever G: *Histopathology of the skin*, 6 ed. Lippincott, Philadelphia 1983, pp 658–659.
7. Doctor VM, Sirsat MV: Florid papillomastosis (adenoma) and other benign tumors of the nipple and areola. *Br J Cancer* 1971; 25:1–9.
8. Tsujoka K, Kashihara M, Imamura S: Cutaneous leiomyoma of the male nipple. *Dermatology* 1985; 170:98–100.
9. Nasciemento AG, Rosen PG, Karas M: Leiomyoma of the nipple. *Am J Surg Path* 1979; 3:151–154.
10. Hernandez FJ: Leiomysarcoma of the male breast originating in the nipple. *Am J Surg Path* 1978; 2:299–304.

DID THIS REALLY HAPPEN?

By Herbert Berger, MD, 405 pp. New York, Vantage, Press, Inc, 1991. $18.95 (hardcover)

The more I read the autobiographies of our elder physicians, the more I come to see them as a testament to a vanishing world. As such, each is precious archival material with a value to coming generations we can only guess. Dr Berger's memoir puts me in mind of the verbal and written testimonies now being produced in a veritable torrent by survivors of the Nazi Holocaust, not in content, of course, but because there is something about impending mortality that makes memories will out. No longer personal possessions of the mind, they become repositories of history too important to be maintained selfishly within the individual. "KNOW WHAT IT WAS LIKE" becomes the overriding imperative, and tongues and pens go into action.

Herbert Berger, it seems, has always been a story teller, although feminists, I am sorry to say, will not be pleased with some of his tales! For this reason, I enjoyed his account of his life and times, from the vantage point of 80 years. It is true that some personal narratives are exercises in narcissistic indulgence, but this is not one of them. A humanist dedicated to the treatment of the patient rather than the disease, a frontiersman in rural practice, a life-long student (and the first board-certified internist on Staten Island), a medico-political activist, a veteran of a stint in the Navy during the Second World War, a teacher of young physicians, a prodigious traveller and lecturer abroad, a family man, and now a wise old bird, he has lived a life that will be replicated by few who are beginning their careers in medicine.

Dr Berger's narrative style, writing ability, sense of humor, and ability to create a sense of immediate intimacy with the reader have enabled him to produce a book that is interesting. Wisely refraining from an "I was born in . . ." chronicle, he has divided what he has to say into 142 "chapters," each of which deals with the adventures of certain periods in his life, challenges he has met, cases he has studied, people he has known, and interventions that prove you can't win 'em all. These anecdotes serve to reinforce the feeling that one is lucky to be spending an evening with an entertaining fellow whose phenomenal memory, unflagging energy, and *joie de vivre* make him the *raconteur par excellence*.

Dr Berger describes the strength of his nuclear family, which contributed a level of moral support and expectation that enabled him to develop his enduringly generous and disciplined outlook toward life. He describes the hardships of a poor young student, struggling against odds that would seem unthinkable to the kids of today. He explains why he turned his back on city practice and went to Tottenville to begin over a half a century of a "do everything" practice. He confesses why his meddling came close to ruining one life and did just about ruin another, but he acknowledges his culpability with such good grace we are hard pressed not to forgive him. His ideas about what is incalculably stupid about the practice of medicine today ring with honest truth and, impressively, his right to speak has been earned by years of experience and informed observation.

One problem with this book reflects the author's inability clearly to define his audience. He has included some pathophysiological explanations that will be unnecessary to medical readers (although they are not overly long and intrusive), and one can only make a guess whether or not they will be sufficient for readers without a medical background.

Another major problem is the catastrophic copy editing and physically poor presentation offered by the publisher. Repetition that should have been weeded out has been permitted to occur up to four times over, and whole pages are reiterated. The typographical errors detract from the professional look of the volume, and some of the more serious failures of copy editing should prove a grave embarrassment to the author. I am an old Gilbert and Sullivan buff, but it took me a while to identify "Kateshaw," (Amelita). "Galle Curchi" and "Benjamino Gili" would not be singing praises for the massacre of their names, although Enrico Caruso escaped unscathed. Nor could Dr Berger's medical buddies hide from the copy editor's sloth. "Gouverner" and "Montifiore" hospitals are probably used to having their names garbled, but real people like Dr "Rachmeil" Levine and Dr "Gus Echna" (formerly Chairman of the Department of Medicine at SUNY Downstate) would have to be saints to take gracefully what has been done to them by the "perp." While I am still trying to imagine what "ascetic" fluid from ascites looks like, I have given up trying to figure out what to do with "Dubrovink" and "peloponnisos," which not even a copy editor should be expected to spell correctly.

The failure to clarify whether or not the names of patients presented in this book were real or fictionalized is a major gaffe, and the inclusion of an anecdote about Arturo Toscanini, which reflected the aged maestro in an unflattering light, is in questionable taste.

A goodly portion of Dr Berger's book will be of interest only to armchair travellers, for he presents thumbnail sketches of every country he has visited, undoubtedly from notes maintained through the years. Once again, he treads these treacherous waters with a fair amount of skill.

Dr Berger may be forgiven a prideful amount of self-congratulatory messages, for they appear well-earned; and what might seem to be name-dropping is probably only a sincere pleasure in the interesting people he has cultivated through the years. Ultimately, what is most impressive is the remarkable, astounding, and almost unbelievable stories of the love affair between doctor and patient that no one, not even Dr Berger, could have made up. They serve as the central nidus of the book and are accountable for its name. Neither Dr Berger, the reviewer, nor the reader would wish for a return to the era of helplessness and watchful waiting for the crisis; however, in reading his stories, one can only mourn what has happened to the practice of medicine, where human beings and their families are reduced to their mindless parts, diagnosed by machines, monitored by instruments, cured by technology, and discharged by computers, while the doctor stands by scratching his head and watching the traffic.

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IN THE SHADE OF AN ACACIA TREE

By Frank L. Lambrecht, 418 pp, illustrated. Philadelphia, American Philosophical Society, 1991. $40.00 (hardcover).

Frank Lambrecht's memoirs of 15 years in the Belgian Congo vividly present the pleasures and the problems of scientific research in the tropics. On the basis of recollections, diary entries, and correspondence, they reflect the last years of the colonial experience in the Congo. Dr Lambrecht reached the Congo in the spring of 1945, just before the German surrender at the end of World War II, and left in 1959, within a year of Belgium's relinquishing its colonial enterprise.

He was initially employed by the Congo Red Cross as a District Health Officer in the Eastern Congo; after three years he joined the staff of the Institut pour la Recherche Scientifique en Afrique Centrale (IRSAC) headed by Dr Vanden Berghe of the Antwerp Institute of Tropical Medicine. Thereafter, his major official responsibilities were the establishment of various IRSAC centers, and conducting medical surveys in Urundi (now Burundi) and Ruanda (Rwanda). In the performance of these duties, the author first investigated human and other mammalian and avian malarial parasites but was gradually drawn into the field of glossinology. Serendipity and astute observation contributed to his discoveries of the ecology and habits of several Glossina species, basic information required for the better control of human and animal trypanosomiasis.

The author records an era of tropical disease research that is now gone, an era characterized by enthusiasm, improvisation and low funding. To read him is to listen to an old-timer tell how it once was. He brings to life struggles against rot and termites, and against bureaucracy as well. Birdsong at the cool dawn of a new day, a sudden, drenching tropical downpour, dust drawn into the open back of a vehicle on laterite roads—his descriptions recall personal experiences in Africa in words not all of us are able to command.

Dr Lambrecht describes his scientific work in the context of warm accounts of the lives and practices of the African peoples among whom he and his family lived. Mutima the house boy, for example, early assumed responsibility for Lambrecht's well-being, to the point of making certain he was dressed appropriately for official functions. Whenever Lambrecht moved to a new post, he made certain that Mutima was brought to it. He trained young village men in conducting Glossina surveys, even in the discussion and examination of the tsetses for the presence of trypanosomes. While accepting, even justifying many colonial practices, his accounts reflect an appreciation of local customs and social systems.

The book is handsome in appearance. Photographs of people and places are augmented by African scenes sketched by Jessica Rice, the author's daughter. A mild complaint: When I tried to follow Dr Lambrecht's travels on the maps, I could not find many of the localities to which he alluded.

This book will be a joy to the person who has conducted research in the tropics. It will afford pleasure and edification as well to anyone interested in tropical diseases, Africa, the history of medicine, or the human condition.

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CRUSADING DOCTOR: MY FIGHT FOR COOPERATIVE MEDICINE

By Michael A. Shadid, MD. 238 pp. Norman, Oklahoma, University of Oklahoma Press, 1992 (reprint). $18.95 (hardcover)

What does it mean when consumer crusader Ralph Nader writes a foreword for a book authored by a physician? Is there greater meaning in the fact that the book is entitled, *Crusading Doctor: My Fight for Cooperative Medicine?* Does the following laudatory remark by Nader help? "No better example of service can be recounted than that rendered by Dr Shadid and his sons. They practiced medicine as if only people mattered." These questions and Nader's evaluation lead to the inevitable conclusion that organized medicine will not be wearing white hats in this good against evil epic battle.

Readers should recall the nature of American society and organized medicine during the great depression as they read this book. However, even many years after the great depression, some physicians may recall similar bitter events relative to the opposition to Medicare and the development of the Health Insurance Plan of Greater New York (HIP). At one time, no HIP obstetrician on Staten Island had a hospital appointment there. All HIP deliveries had to be made at hospitals in other boroughs.

After 22 years as a successful practicing physician, Michael A. Shadid established America's first cooperative hospital in the farming community of Elk City, Oklahoma, in 1929. Shadid copied the existing concept of farmers' cooperatives that banded together for mutual benefits and reduced expenses. Eventually, five basic principles emerged as the cornerstone of a cooperative approach to health care:

- Prepayment: Budgeting the cost of medical care.
- Comprehensive medical health care: Preventive and curative
- Group medical practice
- Ownership and management of facilities by prepayment members
- No interference by laymen with the professional practice of medicine by those licensed so to do.

Today, organized medicine accepts these principles. Prepaid insured medical care is part of the American way of providing health care. During the great depression of the early 1930s, Shadid's ideas were labeled radical at the least and communist or socialist or both at the extreme. How could anyone provide health care to farming families for $25 a year?

Shadid's medical colleagues resolved that he was a charlatan and bitterly did everything they could to vilify him and to destroy the Elk City Community Health Association. At a special 1931 meeting of the Beckham County Medical Society, a physician, allegedly a Ku Klux Klan member, aroused his colleagues. "Doctor Shadid is a schemer. He is fooling these poor farmers and radicals. He is a spell-binder and has hypnotized these poor people into building a hospital for him . . . I do not want to associate myself with crooks—at least not foreign crooks. I am not going to tolerate foreigners coming over here and telling us how to practice medicine. This country belongs to the people who inhabit it and not to immigrants. If I had my way about it, I would expel them all and send them back to where they came from." Then, the members considered a motion to "drop Dr Shadid's name from the roll, cancel his membership and bring charges against him for steerage and unprofessional conduct." Steerage involved paying agents to make referrals to a physician. However, it was pointed out that the Society could be sued for libel if the charges were not substantiated. A member rose to make an astounding motion. "I move, Mr Chairman, that we surrender our charter as a county medical society and that we disband and adjourn *sine die* (without a date set for resumption)." The motion was duly seconded and when put to a vote was carried by a large majority. Believe it or not, the medical society disbanded rather than let Shadid continue be a member in good standing!

Politics, licensure revocation, outright lies, intimidation, and a motley blend of dirty tricks and double dealing all come together in this battle of David against Goliath. However, Dr Shadid is the author, and we only have his side of the story.

Born after his father's death in 1882, Shadid was the 12th child, one of three surviving, of Syrian parents living in what is now Lebanon. In 1898, the family came to America. Shadid put himself through college and medical school peddling cheap jewelry door-to-door. At 24 years of age, he earned his MD from the Washington University School of Medicine in St. Louis. During the 1940s, he was an unsuccessful candidate for the US Congress from Oklahoma's seventh district. He campaigned urging national legislation for socialized medicine.

Later, from 1946 to 1949, he was president of the Cooperative Health Federation of America. Two of Shadid's six children also followed his footsteps by becoming physicians.

Many younger physicians may have no idea whatsoever about organized medicine's continuous caustic opposition to the concepts of "socialized medicine" and prepaid health care. To them, this book will be an eye-opener. Others will find the book enjoyable as a Horatio Alger story, the personal odyssey of an immigrant who made good. Still others will empathize with Shadid's reparative idealistic efforts to make life and health care easier for others. Whatever your views, this book is strongly recommended.

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INTERPRETATION OF DIAGNOSTIC TESTS: A SYNOPSIS OF LABORATORY MEDICINE

Fifth Edition. By Jacques Wallach, MD. Boston, Massachusetts, Little, Brown and Company, 1992. $29.50 (softcover)

The stated purpose of this book is to serve as a quick pocket-sized reference to the large number of laboratory tests now available for medical diagnosis (Preface, pp ix–x).

Dr Wallach has seen his work through five editions, and is a recognized expert in the field. He uses a terse writing style, appropriate for works such as these. Tables are also used whenever possible in an attempt to organize data and save space.

The book is 933 pages in length, divided into four sections. The first presents normal ranges for a wide variety of tests. The second lists the diseases that may result in an abnormal result. The third section, composed of 13 chapters, makes up the bulk of the book. Each chapter is dedicated to an organ system, and presents an exhaustive list of laboratory abnormalities seen in the major diseases of that system. Section Four discusses the effects of drugs on laboratory test results, therapeutic drug monitoring, and toxicology. The book ends with a short appendix of abbreviations and conversion factors. This is followed by a very complete index.

Wallach's work is superior to others in the field in three particulars: It is better organized, more complete, and more current. For example, Bakerman's *ABC's of Interpretive Laboratory Data* was last revised in 1984, is organized solely according to test, and lacks an index. Two other general laboratory reference books, Rollings' *Facts and Formulas* (also © 1984) and Goldenberg, Barnes and Redding's *Diagnostic Testing Handbook for Clinical Decision Making* (© 1989), are also much less complete and current than is Wallach's work.

The most significant shortcoming of this fifth edition of Wallach's book is its size. It is a full octavo, measuring 8″ by 5″ and 1″ wide. It barely fits in an otherwise empty laboratory coat pocket. For all practical purposes, this takes it out of the "pocketbook" category, and defeats the author's intended purpose of providing a "pocket-sized" reference.

Wallach's book also shares the shortcomings inherent to all such single-authored, "quick" references. I take the following examples from the endocrinology section.

The author tends at times to list outdated procedures in his compendium of tests. For example, the iodine tolerance test is still mentioned in the thyroid section (p 454), and the glucagon and histamine provocative tests are included in the section on pheochromocytoma (p 522).

In addition, the myriad laboratory abnormalities described for each disease are not always weighted according to the probabilities of their occurrence. We are therefore informed that in hyperthyroidism, hyperglycemia, glycosuria, and abnormalities of liver function tests are found, without being told how often this is the case (p 455). Similarly, generalizations are often too sweeping. For example, thyroid function studies are simply considered "not indicated" for hospitalized patients with acute medical or psychiatric illnesses (p454).

Finally, Wallach's diagnostic flow diagrams are not always in an order that most specialists in the field would choose to follow. In the diagnosis of hyperthyroidism, an elevated serum thyroxine level immediately leads to a radioactive iodine uptake test (p459); and in the work-up of pheochromocytoma, selective venous sampling is recommended before localizing studies such as computed axial tomography and magnetic resonance imaging (p523).

In spite of the above shortcomings, in the field of "pocket" laboratory references, Wallach's *Interpretation* is probably still the best of the bunch provided, of course, that one's pocket is large enough.

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THE KIDNEY

Fourth Edition (2 vol). Edited by Barry M. Brenner, MD, and Floyd C. Rector, Jr, MD. 3,881 pp, illustrated. Philadelphia, WB Saunders, 1991. $275.00 (hardcover)

This is the fourth edition of this highly successful, multi-authored textbook on the kidney in health and in disease. The first edition appeared in early 1976, and the second in 1981. Over the interval, the number of pages has grown from about 2,700 to almost 3,900, but the shelf space occupied has not increased. The price, of course, has grown.

The editors' approach is uncompromising. The authors are generally leaders in the field, and each has been given adequate space to develop the topic in substantial detail. This is intended to be an encyclopedic coverage. Advances in the understanding of transport, metabolic support of renal function, and molecular biology are all covered with up-to-date chapters. The range is also intended to be all encompassing: The final chapter deals with ethical, social, and financial aspects of end-stage renal disease, and they are substantial.

With almost 4,000 pages of text, it is likely that only fellows preparing for board examinations will read every page. This is a book where one goes in search of specific information when faced with a problem in a patient or the need to prepare a lecture or seminar. As part of the review process, I tried to employ the book in both ways.

As a non-expert, I found the chapters on metabolism (Chapter II) and molecular biology (Chapter IV) easy to follow, and informative. They make few assumptions about prior knowledge and handle their complicated subject very effectively.

In my own area of interest, hypertension, I found substantial redundancy and some interal inconsistency among the various chapters. That is not a complaint. One reasonable approach to the editorial responsibility allows authors to develop their subject, and confesses that at our current state of knowledge there will be apparent inconsistency. How does this book compare with others available? The *Textbook of Nephrology* edited by Masseri and Glassock does not attempt to be encyclopedic and thus is probably more readable. On the other hand, the amount of information is substantially less. *The Kidney, Physiology, and Pathophysiology,* edited by Seldin and Giebisch, as the title suggests, focuses much more on physiology and pathophysiology, and much less on clinical problems. The book by Maxwell, Kleeman, and Narins, *Clinical Disorders of Fluid and Electrolyte Metabolism* does a splendid job in the area of electrolyte physiology and pathophysiology but does not cover the broad range that Brenner/Rector does. It is of some interest that Giebisch and Glassock, editors of other textbooks, contribute to this one. Clearly, they believe that their texts have a different function and are addressed to a different audience.

The escalating costs of texts has simplified the final question that each reviewer must address: Who should consider purchase of this text? At $275, the purchasers are likely to be those who practice primarily renal medicine, fellows in training, and every library. Indeed, no medical library is complete and up-to-date without this text.

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PSYCHOSOCIAL ASPECTS OF END-STAGE RENAL DISEASE

Edited by Mark A. Hardy, MD, John Kiernan, MBA, Austin H. Kutscher, PHD, Lynn Cahill, MSW, and Alan I. Benvenisty, MD. 218 pp. Binghamton, NY, Haworth Press, 1991. $29.95 (hardcover)

This volume is the most recent in a series that examines thanatology. It focuses on the psychosocial aspects of end-stage renal disease (ESRD). Edited by Hardy et al, it is a collection of articles and papers on current psychosocial issues in end-stage renal disease. It touches on several key issues that health professionals face in decision making with the increased load of very sick and very old patients. This, of course, is especially applicable to the urban setting in which most of us and our patients live.

This book will be of use to members of the interdisciplinary ESRD professional community. The papers have appeal for social work, nutrition, and nursing staff who manage crisis intervention, family problems, compliance, and AIDS. As such, the book adds little to the knowledge base of the psychosocial aspects of ESRD. The book suffers as a compilation of papers because a central theme or unifying thread fails to emerge. In spite of these flaws, graduate students and other health care professionals will find the volume useful as it highlights the major problems one confronts in everyday clinical work.

A discussion of the various stages one encounters when confronted by the ESRD diagnosis is framed within the context of a family system response. A pilot study is described, but the research methodology is not explicitly stated and statistical analysis is lacking.

The special problems faced by the pediatric renal team are examined within a model of strategic short-term therapy. The paradigm can readily apply to the adult because the ESRD patient's family is also part of the process.

In "Is Ignorance Bliss," Degan goes to the core of the matter when she studies spousal fidelity. We are reminded of the need to provide team counseling and support to couples at risk for sexual dysfunction. The topic of ethics and decision making is discussed and illustrated with appropriate vignettes. Six useful principles of decision making are outlined in "Passions in the Arena." Finally, "Psychosocial Factors in the Care of the Geriatric Nephrology Patient" is a useful and well organized paper and offers valuable insights regarding a population with special needs.

As with all multi-authored works, there is an unevenness in the quality of the analyses presented in the text. Nevertheless, many physicians, social workers, and other health professionals can pick up this tome confident that the materials presented will add to their ability to better understand our patients.

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OBSTETRICS AND GYNECOLOGY REVIEW 1991

By Harrison H. Sheld. 435 pp. Philadelphia, Pergamon Press, 1991. $34.95 (softcover)

This book is a comprehensive study guide whose purpose is to review the most current publications from the leading journals in obstetrics and gynecology. The author, an associate professor from the University of Nevada, has prefaced his review with the clear intentions of providing a "tool for keeping abreast of contemporary developments in obstetrics and gynecology." This guide is divided into several sections: gynecology, reproductive endocrinology, gynecologic oncology, obstetrics, and perinatal medicine. Each section contains a series of questions, emphasizing facts from the most recent publications. Each question is followed by the correct answer and a thorough discussion regarding the publication. All questions are accompanied by multiple references if further information is needed. The author has identified the more pertinent articles that illustrate both classic obstetrics and more importantly newer developments in the fields of clinical obstetrics and research. The book is very easy to use and well-organized. The teaching modality of instruction through examination is very effective, this is particularly so when all information needed is given after the questions and references are available, if time and interest allow. The question format used is the same as used by the American Board of Obstetrics and Gynecology and The Council on Resident Education in Obstetrics and Gynecology (CREOG) with multiple choice, matching, and K-type multiples.

I found this publication a challenge. If you feel you are current with today's literature you may be surprised! How useful is this review in preparation for the CREOG and written board exam? Having recently taken both the written board in June 1991 and the CREOG in January 1992, I feel that Dr Sheld's review is beyond the scope of what is needed to perform successfully on those examinations. However, it may be an excellent exercise in preparation for the oral exam. As a fellow in maternal-fetal medicine, I salute Dr Shelds for his thorough and accurate review of both obstetric and perinatal medicine. I found these sections to be terrifically worthwhile and enlightening.

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Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1992; 92:122–125). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Samples references:

1. Bowen J: Autoimmune paraneoplastic diseases of the nervous system. *NY State J Med* 1991; 91:2–3.
2. Cotran RS, Kumar V, Robbins SL: *Robbins Pathologic Basis of Disease*, ed 4. Philadelphia, WB Saunders Co, 1989, pp 842–845.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine,* PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

(*continued from p5A*)
Credits. Columbia University Medical Center, Alumni Auditorium. Contact: Center for Continuing Education, 630 W 168th St, New York, NY 10032. Tel: (212) 305-3682.

Oct 21. **Update Your Medicine: Update Cardiology.** 16 Cat I Credits. Contact: Association of Practicing Physicians, Cornell University Medical College, 445 E 69th St, Olin 328, New York, NY 10021.

Oct 22–23. **15th Annual Current Concerns in Adolescent Medicine.** 12 Cat I Credits. The Hilton Hotel. Contact: June Lewis, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

AROUND THE NATION

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Oct 23–27. **Advanced Rhinoplasty.** Wynfrey Hotel, Birmingham. Contact: Rhinoplasty '92, 1600 20th St S, Birmingham, AL 35205.

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Oct 23–24. **Pediatrics Update.** Arizona Health Sciences Center, Tucson. Contact: The University of Arizona Health-Sciences Center, Office of Continuing Medical Education, Tucson, AZ 85724. Tel: (602) 626-7832.

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Oct 1–4. **Western Neuroradiological Society Annual Meeting.** Ritz-Carlton, San Francisco. *Oct 26–30.* **17th Annual San Diego Postgraduate Radiology Course.** 25.25 Cat I Credits. Hotel Del Coronado, San Diego. *Oct 29–Nov 1.* **Current Concepts of Magnetic Resonance.** 19.5 Cat I Credits. Ritz-Carlton, Laguna Nieguel. *Oct 30–Nov 1.* **12th Annual Interventional Radiology Course.** 16.5 Cat I Credits. Hotel Del Coronado, San Diego. Contact: Dawne Ryals, Ryals & Associates, PO Box 1925, Roswell, GA 30077. Tel: (404) 641-9773.

Oct 10. **The Diabetic Eye.** 6 Cat I Credits. University of California, Davis, Cancer Center Auditorium, Sacramento. Contact: Office of Continuing Medical Education, UC Davis Medical Center, 2701 Stockton Blvd, Sacramento, CA 95817. Tel: (916) 734-5390. □

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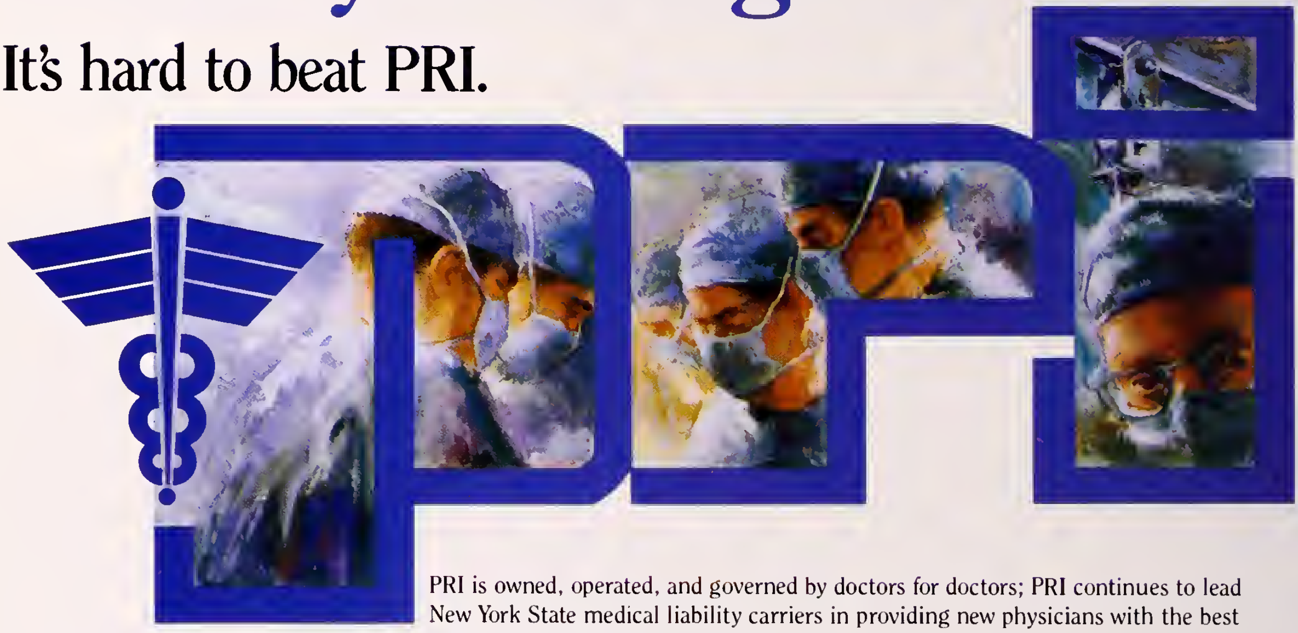

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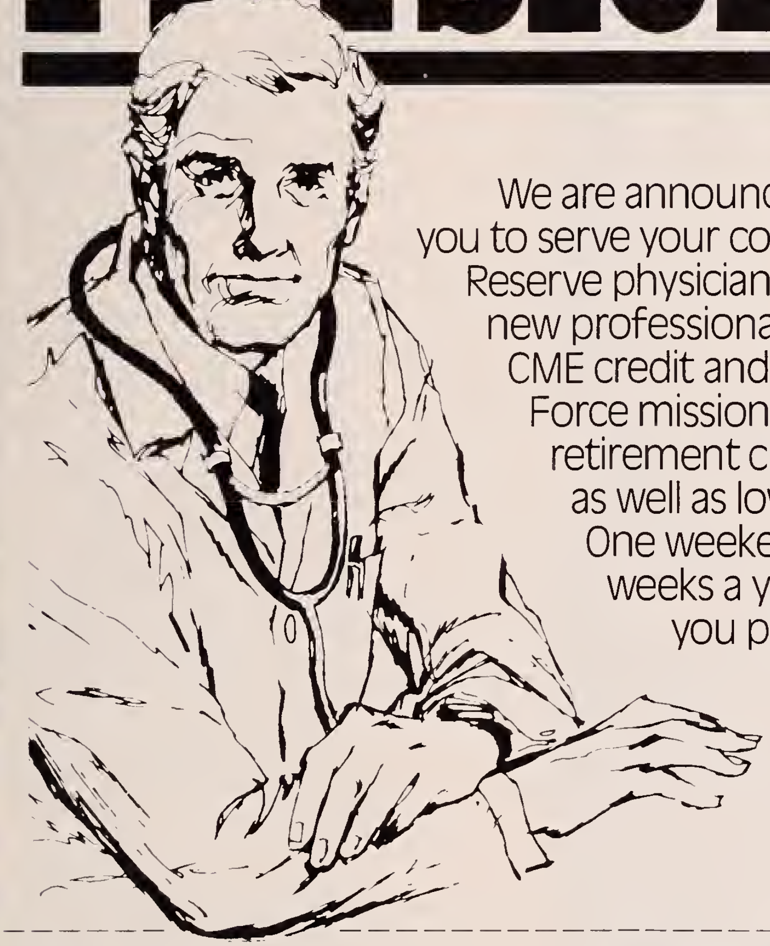

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BRENTWOOD

Nov 17. **How to Achieve Excellence in Patient-Client Relationships.** CW Post College. Contact: Office of Continuing Education, CW Post Campus, Long Island University, Brookville, NY 11548.

HUNTINGTON

Oct 17. **Immunization Update.** *Oct 20.* **Failure to Thrive.** Cat I Credits. Schneider Children's Hospital Grand Rounds at Huntington Hospital. Tuesdays, 8:30 AM to 9:30 AM. *Nov 10.* **Menstrual Disorders I.** *Nov 17.* **Menstrual Disorders II.** *Nov 24.* **Neonatal Bleeding Disorders.** Cat I Credits. Hospital Grand Rounds at Huntington Hospital. Tuesdays, 8:30 AM to 9:30 AM. Contact: Arthur J. Schwager, MD, 164 E Main St, Huntington, NY 11743. Tel: (516) 427-0630.

MANHATTAN

Nov 4–6. **Second Annual Post-Graduate Course: Transplant 1992.** 15.25 Cat I Credits. NYU Medical Center. *Nov 7.* **Fiberoptic Endoscopy Workshop for the Management of the Difficult Airway.** 8 Cat I Credits. NYU Medical Center Alumni Hall. *Nov 13.* **Biologic Response Modifiers as a Therapeutic Strategy for Cancer.** 3.5 Cat I Credits. New York University. Contact: NYU Medical Center Post-Graduate Medical School, 550 Fifth Avenue, New York, NY 10016. Tel: (212) 263-5295.

Nov 18. **Update Your Medicine: Update Infectious Diseases.** Cornell University Medical College Uris Auditorium. Contact: Association of Practicing Physicians, Cornell University Medical College, 445 E 69th St, New York, NY 10021. Tel: (212) 746-4752.

(*continued on p 9A*)

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1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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New York State Journal of Medicine

OCTOBER 1992 / Volume 92, Number 10

Contents

COMMENTARIES

415 **The immunization crisis in New York**
GUTHRIE BIRKHEAD, MD, MPH

418 **Transesophageal echo: Defining the indications**
THOMAS C. HILTON, MD, JOSEPH L. BLACKSHEAR, MD

419 **Esophageal perforation**
JEFFREY A. NIEZGODA, MD

RESEARCH PAPERS

421 **Pap smear histories in a medical clinic: Accuracy of patients' self-reports**
RACHEL G. FRUCHTER, PHD; KATHIE RONES, MD; TRACY ROTH, BA; CAROLYN A. WEBBER, MD; LOUIS CAMILIEN, MD; JOHN G. BOYCE, MD

424 **Extracranial carotid arterial disease: A prognostic factor for atherothrombotic brain infarction and cerebral transient ischemic attack**
WILBERT S. ARONOW, MD; CHUL AHN, PHD; MYRON SCHOENFELD, MD; HAL GUTSTEIN, MD

REVIEW ARTICLE

426 **AIDS prevention by needle exchange**
ANDREW D. FIRLIK, BA, KATRINA SCHREIBER, BA

SPECIAL ARTICLE

431 **The experience of an American medical student with an Aboriginal community in central Australia**
ELIZABETH KAVALER, MD

HISTORY

436 **Leonardo da Vinci's anatomical drawings**
NORA KRISCH SHIRE

440 **The story of Lassa fever—Part III: The disease in the community**
JOHN D. FRAME, MD

(continued on p 6A)

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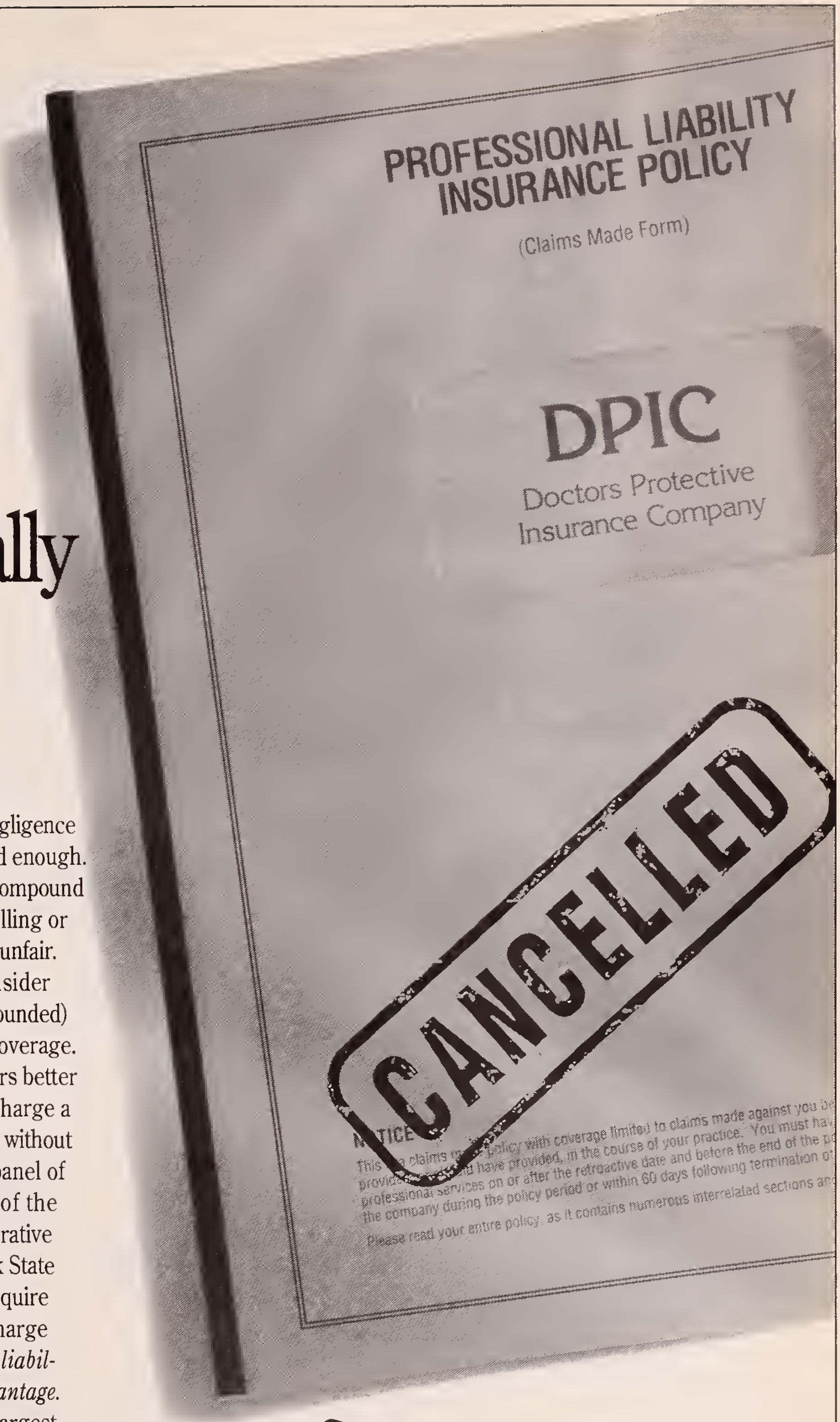

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CONTENTS, CONT'D

New York State Journal of Medicine

CASE REPORTS

445 **Acute tricuspid valve incompetence secondary to blunt trauma**
CHARLES C. CANVER, MD; BLAZE SEKOVSKI, MD; PAUL A. TAHERI, MD; ROBERT M. MENTZER, JR, MD

446 **Cervical esophageal perforation—A unique presentation**
ROBBIN S. DICK, MD, DONALD R. BORDLEY, MD

447 **Diltiazem-induced myoclonus**
JOSEPH S. JERET, MD; MAHENDRA SOMASUNDARAM, MD; SHAILESH ASAIKAR, MD

LETTERS TO THE EDITOR

449 **The *New York State Journal of Medicine***
ALEXANDER L. STRASSER, MD, PETER P. McKELLAR, MD

449 **The role of third party payors in medical care**
FRANCIS J. DURGIN, MD

449 **The changing winds**
RANDALL D. BLOOMFIELD, MD

449 **Pseudoleukemia in a patient with Hodgkin's disease**
SHARON MANNHEIMER, MD, JACOB M. ROWE, MD

ESSAY REVIEW

451 **Rationing America's medical care**
ALLEN D. SPIEGEL, PHD, MPH

454 BOOK REVIEWS

458 OBITUARY LISTINGS

460 AUTHOR GUIDELINES

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New York State
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COMMENTARIES

The immunization crisis in New York

As the 30th anniversary of the introduction of the measles vaccine in 1963 approaches, New York State is facing an immunization crisis in the return of vaccine-preventable diseases like measles, in the continued morbidity from diseases like pertussis (whooping cough), and in the unrealized potential to control *Haemophilus influenzae* type B (Hib) and hepatitis B infections through immunization. The cause of this crisis is the failure to immunize preschool-age children on time. This is due, in part, to a breakdown in primary, preventive health care for a large segment of New York's children.

Immunizations are among the most effective resources in the public health armamentarium. For example, since measles immunization was introduced in New York in 1963, measles morbidity has fallen by over 95% (Table I). In 1962, a total of 47,155 measles cases, and 36 deaths were reported to the New York State Department of Health (NYSDOH). Measles morbidity and mortality reached a nadir 21 years later in 1983 with 90 cases and no deaths reported (source: NYSDOH). A similar pattern can be seen with the other vaccine-preventable diseases. Each year in New York State, tens of thousands of illnesses and hundreds of deaths are prevented by immunizations (Table I).

In spite of the availability of these effective public health tools, the state has seen a resurgence of vaccine-preventable diseases in the past three-to-five years, epitomized by the large measles epidemic that occurred in New York City and surrounding counties in 1990 and 1991. Almost 4,000 confirmed measles cases and 23 measles deaths, including a number among persons infected with the human immunodeficiency virus (HIV), were reported in the state during this outbreak (source: New York City Department of Health and 57 local and county health departments). Many more cases were reported than were able to be confirmed. An examination of the epidemiology of measles in the epidemic shows that the preschool-aged population, those under age five years, makes up more than half of measles cases, especially in New York City. Most of these preschool-age children with measles are unvaccinated. The minority black and hispanic populations have been the most severely affected with two thirds of cases occurring in these groups. Although the epidemic waned in the second half of 1991 and the number of cases has remained low in recent months, the underlying problem is far from solved. Rather, measles may be resuming the seasonal pattern of outbreaks in the spring with a periodicity of every two to three years that characterized measles epidemiology in the pre-vaccine era.[1] A similar but smaller outbreak with over 1,500 reported cases occurred in New York City in 1986 and 1987.

The successes and failures of immunization efforts are also seen with the other vaccine preventable diseases. Diphtheria, tetanus, and polio have been virtually eliminated in New York State, with only two, 31 and no cases, respectively, reported from 1981 through 1991. Although reduced dramatically from the pre-vaccine era, excessive morbidity continues to occur in New York from pertussis, rubella, and mumps, with 363, 26, and 226 reported cases, respectively, in 1990. The success of immunization in reducing morbidity from these diseases is being jeopardized by poor immunization levels, much as the initial success with controlling measles has been. Measles is arguably the most contagious of the immunizable diseases, and it is with this disease that a recurrence of epidemics, reflecting a breakdown in immunization coverage, would be expected to occur first. The immunization crisis may also interfere with efforts to control *H influenzae* type B (Hib) disease, the major cause of meningitis and post-meningitis brain damage in children under age five.[2] Even in the brief time that Hib vaccine has been used in infants, levels of Hib disease have decreased dramatically from 279 reported cases in New York State outside New York City in 1988 to a provisional total of 78 cases in 1991 (source: NYSDOH). However, most cases are still preventable. Finally, the recent efforts to control hepatitis B disease, a leading cause of chronic liver disease and the first cancer prevented by a vaccine, through universal infant immunization[3] are threatened by the failure to achieve adequate immunization levels for the routine childhood diseases.

The proximate cause of the current immunization crisis is the failure to immunize children on time. The

TABLE I. Morbidity and Mortality from Vaccine-preventable Diseases New York State, 1917–1991

	Morbidity Mortality					
Disease	75 Years Ago 1917	60 Years Ago 1932	50 Years Ago 1942	30 Years Ago 1962	25 Years Ago 1967	1991
Diphtheria	19,183	4,235	457	17	1	0
	1,755	270	13	0	0	0
Measles	60,860	60,618	23,265	45,155	1,229	2,306
	904	212	13	36	1	15
Mumps	6,852	10,618[*]	NA	NA	2,214[†]	125
	NA	9	NA	NA	0[†]	0
Pertussis	18,355	23,178	22,269	802	354	199
	894	300	144	1	1	0
Polio[‡]	295	334	292	60	2	0
	92	59	28	2	0	0
Rubella	14,266	1,482	NA	4,246	2,258	541
	NA	1	NA	0	2	0
Tetanus	50[§]	68	63	15	13	0
	73[§]	49	24	3	0	0

*Data for 8 months only.
†1975 data.
‡Due to wild polio virus.
§1919.
Source: Bureau of Communicable Disease Control (CDC), New York State Department of Health.

best data showing this come from the state-wide retrospective immunization survey of kindergarten enterers conducted by the city and state health departments each year. At a sample of schools across the state, the dates of birth and immunization are collected for a sample of kindergartners. The adherence of each child with the recommended immunization schedule is calculated at age two years, the bench mark for determining immunization status. In the 1991 survey, only 56% of kindergartners were up-to-date with the recommended schedule of four DTP, three polio, and one MMR vaccine doses at age two years. The level in New York City was 52% (source: New York City Department of Health), but interestingly the rest of the state achieved only 59% up-to-date at age two. The rest of the state may have been spared a large measles outbreak only because the disease was not introduced into the vulnerable preschool population. A similar study of immunization levels conducted by CDC in the Bronx in 1990 showed only 36% of kindergartners and first grade students were up-to-date at age two.[4] This level was similar to those found in other inner city areas in the United States.[4]

Who are these children who are behind on their immunizations? The available information is based on the characteristics of measles cases, but as indicated above, many more children are under-immunized than acquire measles. Surveillance data indicate that black race and hispanic ethnicity are risk factors for measles disease. There are limited data to suggest that unvaccinated children with measles are poor and that many, but not all, receive publicly funded medical and social services. A CDC study conducted in 1990 showed that in New York City, 63% of unvaccinated, preschool age children with measles were receiving services from the Women, Infants, and Children (WIC) nutrition program, 65% received Aid for Families with Dependent Children (AFDC), and a large proportion were on Medicaid at a time when they could have been immunized (CDC: unpublished data).

Why are these children not vaccinated on time, and what can be done about it? The National Vaccine Advisory Committee has identified three general barriers to adequate immunization; inadequate public awareness of and demand for immunizations; lack of availability of primary preventive health care; and missed opportunities and barriers to immunization within the health care system.[5] Failure to present for routine, preventive health care visits is the first barrier to immunization. Parents won't bring their children for routine care if they don't understand the importance of well-child visits. In some cases, they may have misconceptions about immunization. Anecdotal reports suggest fear that immunizations can cause sterility or AIDS may be keeping parents in some communities from seeking routine care for their children. Another probable factor is the stress of daily living for parents in difficult socioeconomic circumstances that may push health care for their children too far down the list of priorities.

The second barrier to immunization is the collapse of the primary health care system in many of these communities. Even if parents are motivated to bring their children for care, they are often faced with months of wait for an appointment and dilapidated facilities when they finally do get in for care. The public sector has seen cutbacks in personnel in recent years, and private hospitals and physicians are increasingly squeezed by the escalating health care costs. Convenient evening, lunch time, and weekend clinic hours for children of working parents are often not available.

Failure to immunize children who do present for care is the third barrier to immunization. The same unpublished CDC study of measles cases cited above indicated that 60% of unvaccinated measles cases had received medical care in a private doctor's office or clinic, 33% in public clinics, and 71% in a hospital emergency department before contracting measles, suggesting that missed opportunities to vaccinate children were occurring in these settings. This is partly a problem with health care workers who may defer immunizations for inappropriate contraindications, such as an upper respiratory infection or low grade fever, and fail to immunize a child with all needed antigens on a single visit. Clinic procedures may impede immunization efforts by requiring a physician examination. Finally, the high costs of vaccines in the private sector is a barrier to immunization. The cost of a doctor's office visit for a child without insurance coverage could be as much as $60 (over $200 for the full recommended series) for vaccine alone at current catalog prices, not including hepatitis B. As a result, children may go unvaccinated or be referred to the increasingly stressed public clinics for free immunizations. All these factors serve to make the current system of delivering immunizations anything but "user friendly." Recently, national immunization practice standards have been promulgated, which address many of these barriers to immunization.[6]

In the past year, a consortium of governmental, professional, and community-based groups has come together to develop comprehensive plans to address the immunization crisis. These include the State Departments of Health and Social Services, the New York City Health Department, county health departments, the State Medical Society, the American Academy of Pediatrics and other professional and community organizations. Individual components of the plans seek to address many of the barriers to immunization identified above and carry a common theme to improve the capacity of the primary health care system. Immunization is arguably the *sine qua non* of primary, preventive health care. If immunization services are deficient, the other components of primary care are also likely to be deficient. A corollary of this is that the immunization crisis cannot be solved without addressing the crisis in primary health care. The solutions to both are interrelated.

The components of the New York State immunization plan include the following: to fund local and city health departments to expand clinic hours, to offer evening and weekend hours, to conduct more and better assessments of immunization levels in their own patients and in their communities, to conduct outreach to high risk, underimmunized populations; to screen immunization status and refer for needed immunizations at all WIC clinics, and to pilot a similar programs in Income Support Centers at the time of AFDC certification, in some cases establishing a link with Medicaid Managed Care sites, and in hospital emergency departments in an attempt to take advantage of missed opportunities for immunizations; to fund community-based organizations in high risk communities enabling them to perform outreach in their communities and to refer children to primary care sites where immunizations can be given; to encourage parents to seek appropriate primary preventive health care for their children and to develop a telephone hot line with a directory of health care providers offering immunization services for use in referrals; to conduct professional education for health care providers to encourage appropriate immunization practices; and to provide needed vaccines to participating primary health care providers to make these efforts successful.

An additional $1.25 million from the current State budget under the Public Health Campaign will support these efforts and additional funding is being sought from the federal government by both the city and state health departments. The state is also funding seven hospitals to develop model immunization programs to serve emergency departments, WIC, and other out-patient settings. The state and city health departments are working together to plan and develop an immunization registry to track the immunization status of all children through a central data base. Finally, the State Departments of Health and Social Services are developing a system to purchase and distribute vaccines to private physicians to use for their Medicaid patients. If successful, this bulk vaccine distribution system could be a model for wider distribution of vaccines in the future.

Taking these steps will not be enough to solve the current crisis, but they are a start. Efforts to improve immunization status can be a seed that will grow to benefit the whole effort to provide adequate, primary, preventive health care. The logic of immunizations and other preventive measures in health care is strong enough and the costs in human and monetary terms of the failure to apply them are great enough, that there is no other course.

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1. Preblud SR, Katz SL: Measles vaccine, in Plotkin SA (ed). *Vaccines*. Philadelphia PA, W.B. Saunders Co, 1988, p187.
2. Immunization Practices Advisory Committee. Haemophilius b conjugate vaccines for prevention of *Haemophilus influenzae* type b disease among infants and children two months of age and older. *MMWR* 1991; 40:1–7.
3. Immunization Practices Advisory Committee: Hepatitis B virus: A comprehensive strategy for eliminating transmission in the United States through universal childhood vaccination. *MMWR* 1991; 40:1–25.
4. Retrospective assessment of vaccination coverage among school-aged children—selected US cities, 1991. *MMWR* 1992; 41:103–107.
5. National Vaccine Advisory Committee: The measles epidemic: The problems, barriers and recommendations. *JAMA* 1991; 266:1547–1552.
6. National Vaccine Advisory Committee: Standards for Pediatric Immunization Practices. Bethesda, MD. Department of Health and Human Services. April, 1992.

Transesophageal echo: Defining the indications

Although transesophageal echocardiography (TEE) was developed in the late 1970s, it was not until the latter half of the 1980s that commercially available flexible transesophageal endoscopes were widely available in this country. Since its introduction, TEE has served to complement rather than replace transthoracic echocardiography (TTE). The risk, safety, and clinical use of the technique is established. Unsuccessful insertion of the TEE probe occurs very rarely, and the risk of serious complications is less than 1%.[1]

The esophagus is an ideal acoustic window to the posterior aspect of the heart and complements the anteriorly oriented TTE examination. Absence of an air interface, close proximity to cardiac structures, and the use of higher frequency transducers are some of the reasons that TEE produces superior image quality. TEE is a "semi-invasive" technique with a small, but not negligible morbidity and mortality. Thus, TEE is indicated only when test results may alter therapy. The technique is most likely to affect patient management when it is applied to the assessment of posterior cardiac structures such as the left atrium, the left atrial appendage, the interatrial septum, the mitral valve, and the thoracic aorta. TEE is also useful in the assessment of critically ill patients, many of whom have technically limited surface echocardiograms.[2]

The case described by Canver et al, in this issue of the *Journal*,[3] exemplifies the important role of TEE in the evaluation of critically ill patients. TEE in the intensive care unit is considered safe, and observational data have shown that the yield of clinically important information is high.[2]

With TEE, Khandheria et al demonstrated an important abnormality in 48% of patients with suspected porthetic mitral dysfunction and a normal TTE.[4] Improved diagnostic accuracy results from superior image quality and Doppler assessment of mitral regurgitation without intervening prosthetic material, which may shadow or mask the presence and severity of the regurgitant jet. Assessment of pulmonary venous flow (available only with TEE) may also provide important quantitative information about regurgitation severity. These key aspects of TEE also make it extremely useful for the assessment of native mitral valvular disease.

TEE has largely replaced or supplemented the use of digital palpation, open chest surface echocardiography, and double sampling indocyanine green dye curve assessments of mitral valve regurgitation during operative repair.[5] In this setting, TEE is safe, practical, and likely to yield important additional information about left ventricular function and associated valvular abnormalities.

In patients with systemic embolism, TEE can identify a potential cardiac source of embolus in 20% of patients with non-diagnostic TTE.[6] Intracardiac masses, undetected aortic disease, and abnormalities of the interatrial septum are the principle findings.

Contrast injections during TTE can demonstrate the presence of a right-to-left shunt in a high percentage of patients. However, 20% of atrial septal defects are not seen by TTE. These are chiefly sinus venosus atrial septal defects, and they are readily seen with TEE. Atrial septal aneurysm, which has an association with stroke, is readily identified by TEE.[7]

In the diagnosis of aortic dissection, TEE, computed tomography (CT), and magnetic resonance imaging (MRI) all have sensitivities and specificities that exceed 95%. TEE offers advantages over other modalities in that it is portable and, thus, can be performed in the emergency room, intensive care unit, or operating room.[8] Additionally, TEE provides useful information regarding aortic valve competency, left ventricular function, and pericardial effusion or tamponade. CT and MRI are superior in identifying the extent of infradiaphragmatic dissection. However, this information rarely alters surgical management.

Additional indications for TEE include assessment of anatomical complications of endocarditis,[9] intraoperative monitoring of cardiac function, and intraoperative detection of intracardiac air. Although not uniformly required in these circumstances, a compelling case can be made for the use of TEE in selected patients with these conditions.

In summary, TEE provides information that is unique and complimentary to surface echocardiography in patients with certain important cardiovascular disorders. In large centers, approximately 10% of patients undergoing TTE are subsequently referred for TEE. Current indications for TEE are based on observational data rather than randomized controlled trials. However, its impact on patient management is often dramatic, and thus, it has become viewed as an accepted and necessary technique in certain clinical conditions. It is unlikely that randomized controlled trials will be performed that document the benefits of TEE in clinical management. Thus, the decision to use TEE should be based on an urgent clinical question and the adequacy or inadequacy of TTE to provide sufficient data.

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1. Matsuzaki M, Toma Y, Kusukawa R: Clinical applications of transeosophageal echocardiography. *Circulation* 1990; 82:709–722.
2. Pearson AC, Castello R, Labovitz, AJ, et al: Safety and utility of transesophageal echocardiography in the critically ill patient. *Am Hear J* 1990; 119:1083.
3. Canver CC, Sekovski B, Taheri PA, et al: Acute tricuspid valvular incompetence secondary to blunt trauma. *NY State J Med* 1992; 92:445–446.
4. Khandheria BK, Seward JB, Oh JK, et al: Value and limitations of transesophageal echocardiography in assessment of mitral valve prostheses. *Circ* 1991; 83:1956–1968.
5. Sheikh KH, de Bruijn NP, Rankin JS, et al: The utility of transesophageal echocardiography and doppler color flow imaging in patients undergoing cardiac valve surgery. *J Am Coll Cardiol* 1990; 15:363–372.
6. Pearson AC, Labovitz AJ, Tatineni S, et al: Superiority of transesophageal echocardiography in detecting cardiac source of embolism in patients with cerebral ischemia of uncertain etiology. *J Am Coll Cardiol* 1991; 17:66–72.
7. Pearson AC, Nagelhout D, Castello R, et al: Atrial septal aneurysm and stroke: A transesophageal echocardiographic study. *J Am Coll* 1991; 18:1223–1229.
8. Kotler MN: Is transesophageal echocardiography the new standard for diagnosing dissecting aortic aneurysms? *J Am Coll Cardiol* 1989; 14:1263–1265.
9. Daniel WG, Muggs A, Martin RP, et al: Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. *N Engl J Med* 1991; 324:795–800.

Esophageal perforation

Perforation of the esophagus, esophageal inlet, or pharynx can result from many causes, including blunt external trauma, penetrating wounds, iatrogenic trauma during operation, endoscopy, or intubation, postemetic perforation as seen in Boerhaave's Syndrome, or from trauma related to swallowed foreign bodies.[1] Fortunately, esophageal perforation is a relatively uncommon medical or surgical presentation. It can be associated with disturbingly high mortality rates, reported in excess of 50% in some series.[2] These high mortality rates correlate well with missed or late diagnosis.

A knowledgeable clinician with a high index of suspicion is best able to establish the diagnosis of esophageal perforation early and limit the impact of the rupture on his patient. In this issue of the *Journal*, Dick and Bordley describe yet another unusual presentation of perforation.[3] This case and its discussion provide valuable information for physicians employed on the front line of medicine,[3] the emergency rooms and the urgent care clinics, where these patients are most likely to present for medical care initially.

It is important to emphasize that the diagnosis of esophageal perforation is based on a constellation of the patient's presentation, the clinician's examination, and appropriate radiographic studies. Exclusion of any of these can result in a missed or delayed diagnosis. As illustrated by the case reported in this issue of the *Journal*, the presenting symptoms can vary. The patient's symptoms are influenced by multiple factors including the location, the severity, and the etiology of the perforation, as well as the duration between perforation and diagnosis.

In addition to vomiting, dysphagia, hematemesis, and shortness of breath, other initial symptoms may include chest pain, nausea, pleurisy, back pain, diaphoresis, neck pain, or abdominal pain.[4] As this potpourri of symptoms will not usually significantly narrow the list of potential diagnoses, the present case also illustrates the importance of a complete history. Symptomatology spatially related to an episode of vomiting must raise the index of suspicion so as to include esophageal perforation in the differential diagnosis.

Physical examination can reveal subcutaneous emphysema and crepitation, especially in a patient with a high esophageal tear. These findings mandate radiological studies. It has been reported that abnormal air streaking seen on plain films is nearly diagnostic for pharyngoesophageal perforation especially in those patients presenting after neck trauma.[1] Contrast swallow studies are also useful to delineate location and extent of perforation. However, as Dick and Bordley discuss, after initially basing the diagnosis on radiographs demonstrating air in the soft tissue, it is not unusual to be unable to locate the perforation site with meglumine diatrizoate swallow or during surgical exploration.

Dick and Bordley postulate that the rupture in their patient resulted from esophageal trauma from a foreign body in combination with large intraesophageal pressures produced by the patient's episode of vomiting. This hypothesis is certainly plausible as various foreign bodies including bones, coins, dentures, a pull-tab from a soda can, and a sword (as in sword swallowing) have all been implicated in causing perforation. The pressure rise during emesis has been documented to cause a five-fold increase in the size of the diameter of the lower esophagus[5] and the Boerhaave's Syndrome describes esophageal perforation caused by such pressure changes during forceful vomiting. The combination of trauma and vomiting is logically the likely culprit in this case.

The treatment of pharyngoesophageal perforation is based on the location and extent of the perforation, as well as the patient's clinical condition and associated injuries if present. Prompt and aggressive surgical treatment is the standard for large perforations, perforations extending to and involving the esophagus, and for those patients who are critically ill or with extensive damage to the esophagus and mediastinum. A recent series of high cervical and pharyngeal perforations establishes the fact that small perforations, less than 2 cm, which are limited to the pharynx, can be managed with conservative, nonsurgical management.[1]

Esophageal perforations are usually described as diagnostic dilemmas with the associated severe consequences of high morbidity and mortality rates, especially when definitive treatment is delayed. Clinicians armed with a basic understanding of this disease process, a high index of suspicion, reasonable physical examination skills, and the liberal use of appropriate roentgenograms, will be prepared to diagnose and treat successfully the patient with pharyngoesophageal perforation.

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1. Niezgoda JA, McMenamin P, Graeber GM: Pharyngoesophageal perforation after blunt neck trauma. *Ann Thorac Surg* 1990; 50:615–617.
2. Patton AS, Lawson DW: Reevaluation of the Boerhaave's: A review of fourteen cases. *Am J Surg* 1979; 137:560–565.
3. Dick RS, Bordley DR: Cervical esophageal perforation—a unique presentation. *NY State J Med* 1992; 92:446–447.
4. Graeber GM, Niezgoda JA, Burton NA, et al: A comparison of patients with endoscopic esophageal perforations and patients with Boerhaave's syndrome. *Chest* 1987; 92:995–998.
5. Donner MW: Hemorrhage at the esophagogastric junction (including bleeding esophageal varices) in Katz D, Hoffman F (eds). *The Esophagogastric Junction*. Amsterdam, *Excerpta Medica*, 1971, pp 76–77.

FROM THE LIBRARY

AN OUTBREAK OF DIPHTHERIA IN A SECOND-CLASS CITY

Late in November the attention of the officials of a second class city, having a population of about 54,000, was called to the fact that diphtheria had become so prevalent there that the municipality was charged with the highest case rate for this disease of any city in the State. Following a request for assistance, from local officials, the writer was assigned to assist in efforts to control the disease.

The outbreak was not of the rapidly developing type which, as a rule, yields readily to the application of scientific preventive measures, but one which had been developing progressively for four years and had become generally disseminated. The records indicated that during 1912 but 28 cases had been reported; in 1913 there were 60; in 1914, 143; in 1915, 201; and finally, in 1916, there were 358, with the greatest incidence in the last three months of the year. Nearly a year before, the attention of local health officials had been called to the fact that the cases were largely localized in one city ward. During 1916 it had invaded nearly all parts of the city.

From a preliminary survey, we made the following deductions. Generally speaking, 79.5 per cent of the cases had occurred among persons under twenty years of age, the greater number localized to some extent in districts covered by a few public schools. A detailed study of cases in one or two such districts showed that a majority of the cases were among school children, while in a number of other families with cases under or over school age, there were school children, many of whom had had sore throats.

There had been, continuously occurring, cases of “sore throat” and “tonsilitis,” so called, to some of which no physician had been called.

Following the survey, our first procedure was to arrange a meeting of local physicians with city and school officials. The situation was presented, regulations reviewed, culture material and report cards distributed, and the planned campaign outlined. Immediately following this meeting there was a marked increase in the number of cases reported and cultures submitted to the laboratory.

The plan of campaign included the culturing of school children and contacts, control of carriers, close supervision and enforcement of rational quarantine, hospitalization where necessary, the seeking out of unreported cases, and the opening up of various channels of communication between the schools and the Health Department. Our plans were carried out as fully and effectively as local conditions and a limited corps of workers permitted. It soon became necessary for the city to provide additional hospital facilities, the result being permanent provision for at least thirty patients. The local laboratory facilities were found inadequate to meet the increased demand and it was necessary at times to send from one to two hundred cultures daily to the State laboratory at Albany.

A rule was promulgated providing that cultures for release from quarantine should be taken by the attending physician in any case if so desired, until one negative culture had been obtained, after which subsequent cultures would be taken by a representative of the Health Department.

In consideration of the foregoing, the writer is confirmed in his personal opinion that, both out of consideration of the physician and in the interest of effective quarantine, the responsibility for the taking of release cultures should invariably rest upon the health authorities.

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(*NY State J Med* 1917; 17:295–297)

Pap smear histories in a medical clinic: Accuracy of patients' self-reports

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ABSTRACT. **Women using the medical clinic of a public hospital were interviewed about their Pap smear histories to assess the accuracy of self-reported smears and to identify groups in need of further screening. Interview data from 263 women were compared with cytology files and hospital records. In spite of considerable agreement between patient report and record, patients reported significantly more recent smears than were documented. Accuracy of recall was not dependent on age or birthplace, but on the length of time since the last smear. About half the women had been screened within the past two years, whereas one tenth had never been screened. Women aged 65 years or older had fewer recent smears than younger women, while foreign born women were more likely never to have had a Pap smear than were United States born women. We conclude that self-reported information is useful in assessing Pap smear histories, but the screening rates that result should be treated as high estimates. Significant opportunities for screening exist in ambulatory care sites in low-income communities.**

(NY State J Med 1992; 92:421–424)

Although most women in the United States have had a Pap smear at some time,[1,2] national and local studies consistently show that older and low-income women are less frequently screened than younger and more affluent women.[1,2] Previous studies in Brooklyn and New York State[3,4] have demonstrated that there are major opportunities to screen older women when they seek ambulatory medical care for non-gynecologic problems. In central Brooklyn, NY, invasive cervical cancer continues to be a problem, with incidence rates of 17–27/100,000 in the black and Latina communities.[5] A study at Kings County Hospital Center (KCHC) and the University Hospital of Brooklyn (UHB) of 174 women with invasive cervical cancer found that although 87% had received ambulatory medical care during the previous five years, only 39% had had a Pap smear performed as part of that care.[6] The latter report documented inaccuracies in patients' self-reported smear histories. The current study was undertaken to assess the need for Pap smear screening in hospital ambulatory care sites, and to assess whether a short interview was a reliable means of assessing screening history in patients who do not have cancer.

PATIENTS AND METHODS

Two hundred and sixty-three female patients attending six sessions of the medical clinic at KCHC in Brooklyn, NY, were interviewed by a medical student while waiting to see a physician. All women were previously registered in the clinic and most had appointments. The clinics included two morning, two afternoon, and two evening sessions. The interview instrument was developed, and the interviewer trained during a pilot study of 50 medical clinic patients who were not included in the main study. Patients were interviewed using a uniform instrument with 13 questions including age, birthplace, length of residence in the United States, number of years since the last Pap smear (and approximate date if known), the facility where the previous smear was taken, and the usual source of medical care. If patients did not know what Pap smears were, this was explained at the beginning of the interview. (A Pap smear is a test for cancer of the womb; the test involves a vaginal examination). Interviews took about three minutes each and were conducted in English or French with interpreters as needed.

The cytology records of KCHC and the adjacent UHB were searched to document any Pap smears of the 263 women. The cytology records are stored in manual alphabetic index files that include the date and result of each smear. The files were reviewed first by a student, and six months later, by the study director to allow for delays in filing reports. In addition, the hospital charts (combined inpatient and outpatient) were reviewed to locate those for 29 patients who reported a smear at KCHC of which there was no evidence in the cytology files, and 25 patients who reported a smear more than 12 months later than the cytology records show.

For patients with documented smears, the patients' self-reported screening interval and the documented screening interval were compared in two ways. First, the self-reported and documented screening intervals were compared using the κ-statistic as an estimate of concordance.[7] The amount of skew of overreporting compared to underreporting was analyzed using a modified McNemar's χ^2 test as described by Walter and coworkers.[8] Second, the discrepancy between each self-reported and documented screening interval was determined, and the proportions of patients who overestimated or underestimated the interval was compared using a χ^2 test.[7]

RESULTS

In this study, 86% of 306 women attending six clinic sessions were interviewed, with 14% leaving before the interviewer could talk to them. The study population of 263 women included 32% aged less than 50 years and 27% aged 65 years or more. More than 95% were black and Latino. Most patients (53%) were born outside the United States or Puerto Rico,

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with 75% residents in the United States for five years or more, and 55% residents here for ten years or more.

Two hundred and forty-five women (93%) claimed to know what a Pap smear was, while four (2%) were unsure, and 14 (5%) did not know of the test. Seventeen of these 18 women (94%) were foreign born, including ten (59%) who had been in the US for less than five years. Six women who had lived in the United States for more than ten years, but did not know about Pap smears, had ages ranging from 64 to 88 years. After an explanation of a Pap smear, 15 of the 18 lacking knowledge of smears said that they had never had a smear, and three reported having a smear.

Two hundred and thirty-four of the 263 women (89%) claimed to have had a previous Pap smear, whereas 24 women (9%) denied any previous test, and five (2%) were not sure. Of the 234 with a previous smear, 165 claimed that the most recent smear was taken at KCHC or UHB. The KCHC/UHB cytology files included records of smears of 131 of the 165 women (18%), of two of 24 women who claimed they had never had a smear, and of two of five women who were unsure of their smear history. In addition, review of the hospital charts identified smears in three women who claimed to have had smears and identified two more recent smears in women with earlier smears in the cytology files. No attempt was made to verify the records of 65 women who reported their most recent smears at 52 other medical facilities.

The self-reported and documented screening intervals could be compared in 138 women (Table I). Two women who were unsure of their screening histories were included and were considered as reporting smears more than three years previously because they reported gynecologic care at that time. The overall accuracy of the patients' self-reported screening interval was 72%, with the κ statistic of 0.46 indicating fair agreement beyond chance between patient and record. Thirty-one women reported shorter screening intervals than the records indicated, whereas seven women had been screened more recently than they reported, a symmetry ratio of 4.4:1.[8] This ratio indicated a significant tendency for patients to report more recent smears than actually occurred. Using recorded data, the proportion of women screened within the previous two years decreased from 75% to 63% ($p < 0.05$ in χ^2 test), and within three years from 93% to 83% ($p < 0.05$).

The absolute discrepancy between the self-reported and the documented screening intervals was more than on year in 22% of the patients, and more than three years in 10% (Table II) The documented smear was more than one year more recent than the self-reported smear in 6%, and more than one year less recent in 16%, a significant skewing toward recent self-reporting. Of the 30 women with discrepancies of more than one year, 27 (90%) gave KCHC as their usual source of health care as well as the place the most recent smear was taken. If three patients who gave other facilities as their usual source of care are removed from this analysis, the data remain significantly skewed toward patients reporting more recent smears than could be documented.

TABLE I. Self-reported and Documented Intervals Since Prior Pap Smears*

		Cytology Record			
		<2 years	2–3 years	>3 years	Total (%)
Patient	<2 years	83	12	8	103 (75)
Report	2–3 years	1	13	11	25 (18)
	>3 years	3	3	4	10 (7)
	Total (%)	87 (63)	28 (20)	23 (17)	138 (100)

*Accuracy: 100/138 = 0.72.
κ statistic = 0.46 ($p < 0.01$, 4 degrees of freedom).
Symmetry ratio = 31:7 ($p < 0.01$).

TABLE II. Discrepancies Between Self-reported and Documented Screening Intervals

Discrepancy	N	%
Within one year	108	78
More than one year		
Record more recent than self-report	8	6*
Record less recent than self-report	22	16*
Total	138	100

*Proportion with record more recent than self-report vs record less recent than self-report: $p < 0.05$ in χ^2 test.

There was no significant difference in the accuracy of the screening intervals by age or by birthplace (Table III). However, the longer the screening interval, the lower the accuracy and the greater the discrepancy between reported and documented screening intervals.

The distribution of screening intervals using self-reported intervals was compared with an adjusted distribution that used documented intervals when available and self-reported intervals when documentation was unavailable. The proportion of the 263 women who had smears within the previous two years fell from 56% for the self-reported to 49% for the adjusted distribution (Fig 1). There were 69 women whose self-reported smears at other health facilities were not documented, and 31 women whose reported smears at KCHC were not documented. Although these 100 women were younger (40% less than age 50) and more likely to be United States born (60%) than the 138 women with documented smears, a similar proportion (67%) reported a smear within the past two years. If there were similar discrepancies in the screening interval in this undocumented group of 100, the true proportion screened within two years would be 44% of the total.

There was no significant difference among the age groups in the proportion never screened, but women aged 65 years and over were significantly less likely to have undergone recent screening than were women under 65 years (Table IV). Compared to United States born women, immigrants were more likely never to have been screened, but there was no significant difference between United States born and foreign born women in the proportion with recent screening. Among the foreign born, duration of residence in the United States was inversely associated with lack of screening. Thus, 33% of 43 women who had resided in the United States for five years or less had never had a smear compared to 6% of 71 women who had resided in the United States for more than ten years.

DISCUSSION

This study demonstrates significant inaccuracies in women's self-reported Pap smear histories, including both the

TABLE III. Factors Associated with Accurate Reporting of Pap Smears

Factor		Concordant* Reports	Total Reports	Accuracy	Significance
Age	<50 years	28	35	80%	
	50–64 years	44	61	72%	$p > 0.05$
	65+ years	28	42	67%	
Birthplace	USA/Puerto Rico	44	60	73%	$p > 0.05$
	Elsewhere	56	78	72%	
Self-reported Screening Interval	<2 years	83	103	81%	$p < 0.05$
	>2 years	17	35	50%	
Total		100	138	72%	

*Concordant reports are defined as those in which the patient's self-reported interval falls within the same interval as in the smear record (as in Table I).

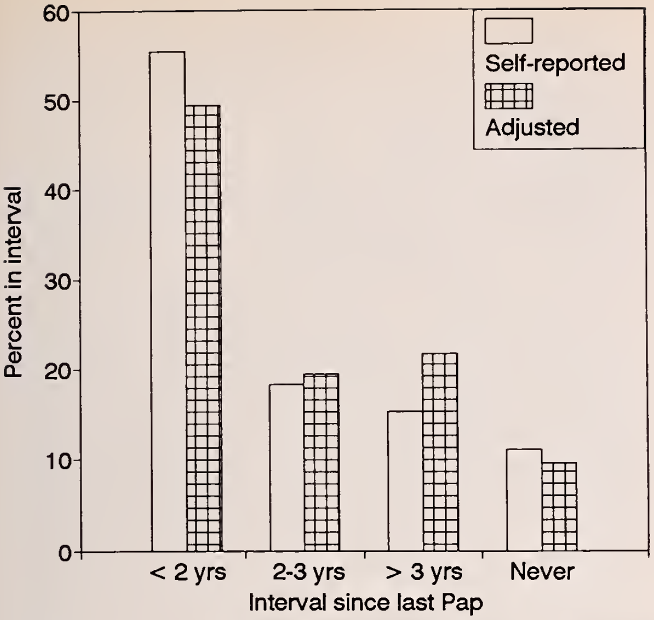

FIGURE 1. Proportional distribution of intervals since last Pap smear test according to patients' self-reports, compared to an adjusted distribution using documented information when available and self-reports when documentation was unavailable.

identification of the facility where the smear was taken and the interval since the last smear. We found a significant tendency for the patient to report a more recent smear than could be documented. Although some discrepancies may reflect inadequacies of the hospital's record system rather than misreporting by the patient, our review of the cytology files and of medical charts suggests that misfiling of records can account for no more than 10% of the discrepancies. Loss of actual slides might also contribute to discrepancies, but was not assessed in this study. We also considered whether the recent smears the patients reported as having been taken at KCHC might have been taken at other facilities. However, 90% of the patients with discrepancies reported that KCHC was the usual site of all their medical care, so it is most likely that the discrepancies are in time rather than place.

Our findings are consistent with other studies evaluating the accuracy of women's self-reported screening histories[5,8–11] in both urban and rural areas and in women with and without cervical neoplasia. All the studies were consistent in two major respects. First, while agreement between self-report and documentation was always greater than chance, nevertheless, each study showed significant discordance, ranging from 11% to 41%. Second, subjects tended to report that smears were taken more recently than the record documented. Thus, all interview data on frequency and recency of Pap smears must be considered to be overestimates.

TABLE IV. Screening History by Age and Birthplace

		Interval Since Last Pap Smear			
		Less than 2 yrs (% of Patients)		Never (% of Patients)	
	Number of Patients	Self-reported	Adjusted*	Self-reported	Adjusted*
Age					
<50 years	83	61.4	60.2	10.8	9.6
50–64 years	108	57.4	50.0	8.3	7.4
65+ years	72	45.8[†]	36.1[†]	13.9	12.5
Birthplace					
USA/Puerto Rico	125	61.6	53.6	4.0	4.0
Elsewhere	138	50.0	45.6	17.4[‡]	14.5[‡]
Total	263	55.5	49.4	11.0	9.5

[*] Adjusted histories include documented reports when available and self-reported data when documentation was unavailable.
[†] Age <65 vs age 65+: $p < 0.05$ in χ^2.
[‡] USA/Puerto Rico vs elsewhere: $p < 0.05$ in χ^2.

Reports on the recall of health-related behaviors suggest that health behaviors considered desirable, such as low-fat diets, are over-reported,[12] whereas undesirable behaviors, such as smoking, are under-reported.[13] We suggest that since regular Pap smears are desirable health behaviors, patients are likely to report more smears and more recent smears than are actually taken. We found no difference in the accuracy of reporting with respect to age or birthplace but could not assess the effect of socioeconomic status, since all the study patients were of low income. As in other reports, the major factor affecting accuracy of recall was the recency of the smear, with recent smears reported more accurately.

Our finding of over-reporting of the recency of Pap smears has significance for clinicians and health planners alike. Clinicians must be aware that the self-reported screening history cannot be relied on exclusively to determine whether an individual patient should be rescreened or referred for screening. The American College of Obstetricians and Gynecologists recommends that after three consecutive, annual, normal smears, the screening interval can be varied at the discretion of the physician.[14] Clinicians exercising their discretion must have confidence in the screening history. In low-income urban communities, where women are at higher risk for cervical cancer, women use multiple providers and relocate frequently, so that it is extremely difficult to document screening histories. Possible solutions include computerized cytology records within an institution or giving the patient written records. In the absence of accurate documented cytology records, we recommend that annual Pap smear screening be performed. This standard has recently been affirmed by the New York State Department of Health's Cancer Advisory Panel on cancer screening guidelines.[15]

Discrepancies in screening histories are important to health planners who usually use survey data of patients' self-reported histories to assess a population's screening level. National Health Interview Survey (NHIS) data indicate that in 1985, 65% of United States women reported a Pap smear within the previous two years.[1] However, NHIS data are not verified from provider records, and therefore the levels reported are likely to be overestimates.

Our study suggests that interviews of a clinic population can provide a useful estimate of the Pap smear screening level if the proportion reporting recent screening is reduced by 5%–10%. Although an audit of cytology records would give a more accurate estimate of smears taken at the facility surveyed, there is no inexpensive way to document smears taken at other health facilities.

We found that only about half the medical clinic patients had been screened within the past two years and one tenth had never had a Pap smear. Women aged 65 years and over were less likely to have had recent screening than were younger women, and immigrants were most likely never to have been screened. Low-screening levels among Brooklyn immigrants have been reported in community studies.[16,17]

Our earlier study of the Pap smear histories of cancer patients[5] indicated that there were significant missed opportunities for screening when women attended ambulatory care facilities. This report confirms that such opportunities exist in hospital clinics, particularly among older women who are unlikely to seek out preventive gynecologic care. Intensifying screening in clinics in low-income areas may be one of the most cost effective way of further preventing the development of invasive cervical cancer.

REFERENCES

1. Makuc DM, Freid VM, Kleinman JC: National trends in the use of preventive health care by women. *Am J Public Health* 1989; 79:21–26.
2. National Center for Health Statistics: Provisional estimates from the National Health Interview Survey supplement on cancer control—United States, January–March 1987. *MMWR* 1988; 37:417–420.
3. Fruchter RG, Boyce J, Hunt M: Invasive cancer of the cervix. I. Previous Pap smear tests and opportunities for screening. *NY State J Med* 1980; 80:740–744.
4. Howe H, Bzduc H: A survey of Pap smear screening in upstate New York. *NY State J Med* 1986; 86:291–296.
5. Boyce JG, Fruchter RG, Romanzi L, et al: The fallacy of the screening interval for cervical smears. *Obstet Gynecol* 1990; 76:627–632.
6. Fruchter RG, Nayeri K, Boyce JG, et al: Gynecologic cancer in Brooklyn, New York, 1978–82. *NY State J Med* 1988; 88:466–473.
7. Fleiss J: *Statistical Methods for Rates and Proportions*. New York, John Wiley, 1981, pp 221–224.
8. Walter SD, Clarke EA, Hatcher J, et al: A comparison of physician and patient reports of Pap smear histories. *J Clin Epidemiol* 1988; 41:401–410.
9. Warnecke RB, Graham S: Characteristics of blacks obtaining Pap smears. *Cancer* 1976; 37:2015–2025.
10. Sawyer JA, Earp JA, Fletcher RH, et al: Accuracy of women's self-report of their last Pap smear. *Am J Public Health* 1989; 79:1036–1037.
11. Bowman JA, Redman S, Dickinson JA, et al: The accuracy of Pap smear utilization self-report: A methodological consideration in cervical cancer screening research. *Health Serv Res* 1991; 26:97–107.
12. Dwyer JT, Krall EA, Coleman KA: The problem of memory in nutritional epidemiology research. *J Am Diet Assoc* 1987; 87:1509–1512.
13. Krall EA, Valadian I, Dwyer JT, et al: Accuracy of recalled smoking data. *Am J Public Health* 1989; 79:200–220.
14. American College of Obstetricians and Gynecologists: Report of task force on routine cancer screening. Washington DC, The American College of Obstetricians and Gynecologists, 1989.
15. New York State Department of Health: Cancer screening guidelines. *Epidemiol Notes* 1990; 5:1–2.
16. Fruchter RG, Wright C, Habenstreit B, et al: Screening for cervical and breast cancer among Caribbean immigrants. *J Community Health* 1985; 10:121–135.
17. Fruchter RG, Nayeri K, Remy JC, et al: Cervix and breast cancer incidence in immigrant Caribbean women. *Am J Public Health* 1990; 80:722–724.

Extracranial carotid arterial disease: A prognostic factor for atherothrombotic brain infarction and cerebral transient ischemic attack

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ABSTRACT. A prospective study investigated the prevalence of extracranial carotid arterial disease by carotid duplex ultrasonography, and evaluated its correlation with the incidence of atherothrombotic brain infarction or cerebral transient ischemic attack in 949 patients, mean age 82 ± 8 years. The mean follow-up period was 45 months. Of 949 patients, 37 (4%) had 80%–100% extracranial carotid disease (mean follow-up, 24 ± 15 months), 113 (12%) had 40%–80% extracranial carotid disease (40 ± 19 months follow-up), and 799 (84%) had 0%–40% extracranial carotid disease (47 ± 14 months follow-up). The average annual incidence of atherothrombotic brain infarction was 37% in patients with 80%–100% extracranial carotid disease, 9% in patients with 40%–80% extracranial carotid disease, and 4% in patients with 0%–40% extracranial carotid disease. The average annual incidence of transient ischemic attack was 3% in patients with 80%–100% extracranial carotid disease, 2% in patients with 40%–80% extracranial carotid disease, and 1% in patients with 0%–40% extracranial carotid disease. The Cox proportional hazard model showed that among the variables evaluated, the severity of extracranial carotid disease correlated with the highest relative risk of developing atherothrombotic brain infarction (2.5× higher relative risk) or transient ischemic attack (2.8× higher relative risk). Patients with an earlier atherothrombotic brain infarction had a 2.1× higher probability of developing atherothrombotic brain infarction and a 1.9× higher chance of developing transient ischemic attack than those without an earlier atherothrombotic brain infarction. Age was a prognostic variable for new atherothrombotic brain infarction, and male sex was a prognostic variable for new transient ischemic attack.

(NY State J Med 1992; 92:424–425)

Asymptomatic patients with extracranial carotid arterial disease (ECD) diagnosed by carotid Doppler ultrasonography have an increased incidence of atherothrombotic brain infarction (ABI) or cerebral transient ischemic attack (TIA) at follow-up.[1–5] We report the results of a prospective study (45-month mean follow-up) correlating the incidences of ABI and TIA in 37 elderly patients with 80%–100% ECD stenoses (24-month mean follow-up), 113 elderly patients with 40%–80% ECD stenoses (40-month mean follow-up), and in 799 elderly patients with no significant ECD (47-month mean follow-up).

PATIENTS AND METHODS

Bilateral carotid duplex ultrasonograms were obtained prospectively in 949 unselected elderly patients (271 men and 678 women), mean age 82 ± 8 years (range, 60–101 yr), in a long-term health-care facility. Bilateral carotid duplex ultrasonograms were performed as previously described with an Interspec XL machine, using a 7.5-MHz transducer with combined two-dimensional real-time and pulsed or high-pulse-frequency Doppler capabilities.[6] The severity of ECD was semiquantified by using conventional Doppler criteria (V_{MAX} <0.80 m/s = $<40\%$ arterial luminal diameter reduction; V_{MAX} 0.80–1.75 m/s = 40%–80% arterial luminal diameter reduction; V_{MAX} >1.75 m/s = 80%–99% arterial luminal diameter reduction; V_{MAX} 0.00 m/s (no Doppler signal on at least two separate tests = 100% arterial luminal diameter reduction).

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TABLE I. Incidence of New ABI at Follow-up in Elderly Patients With and Without Extracranial Internal or Common Carotid Arterial Disease

	New ABI		Mean Follow-up (Months)	Average Annual Incidence (%)
	No.	(%)		
80%–100% ECD	27/37	(73%)	24 ± 15	(37%)
40%–80% ECD	34/113	(30%)	40 ± 19	(9%)
0%–40% ECD	127/799	(16%)	47 ± 14	(4%)

New ABI was diagnosed when the patient experienced a documented sudden, focal neurologic deficit lasting longer than 24 hr in the absence of a known source of embolism, bloody cerebrospinal fluid, known hypercoagulable conditions, or other diseases causing focal brain deficits. ABI was diagnosed if the documented focal neurologic deficit lasted longer than 24 hr and left residual or no residual focal neurologic deficit. A careful neurologic examination was performed in each patient for signs of a focal neurologic deficit that conformed to the typical vascular distribution of an ABI. ABI was also confirmed by computed axial tomography (CAT) in 179 of 188 patients (95%) with ABI. TIA was diagnosed by a neurologist if the patient had transient (<24 hr), contralateral (to the ECD) hemiparesis or hemianesthesia, or transient ipsilateral blindness or visual field defect.

None of the patients with ECD in this study underwent carotid endarterectomy. Thirty-seven of 37 patients (100%) with 80%–100% ECD were prescribed aspirin, 325 mg daily. Of 113 patients, 107 (95%) with 40%–80% ECD were prescribed aspirin 325 mg daily. Mean follow-up was 45 months (range, 3–63 mo), with 24 ± 15 months for patients with 80%–100% ECD, 40 ± 19 months for patients with 40%–80% ECD, and 47 ± 14 months for patients with 0%–40% ECD. The relation between prognostic variables and time to the development of either ABI or TIA was analyzed using the Cox proportional hazard model. χ^2 analysis was also used to analyze the data.

RESULTS

In 949 patients, 80%–100% ECD was present in 37 patients (4%), 40%–80% ECD was present in 113 patients (12%), and 0%–40% ECD was present in 799 patients (84%). Forty-seven of 271 men (17%) and 103 of 678 women (15%) had 40%–100% ECD. Before ABI had occurred in 72 of 150 patients (48%) with 40%–100% ECD and in 216 of 799 patients (27%) with 0%–40% ECD ($p < 0.001$).

Table I shows the incidence of new ABI, and Table II indicates the incidence of new TIA. Tables III and IV show the prognostic variables for new ABI and new TIA, respectively, and their regression coefficients in the Cox proportional hazard model.

Patients with 80%–100% ECD have a 2.5× higher chance of developing new ABI and a 2.8× higher probability of developing new TIA than patients with 40%–80% ECD. Patients with 40%–80% ECD have a 2.5× higher probability of developing new ABI and a 2.8× higher chance of developing new TIA than patients with 0%–40% ECD. Patients with 80%–100% ECD have a 6.4× higher probability of developing new ABI and an 8.0 times higher chance of developing new TIA than patients with 0%–40% ECD.

TABLE II. Incidence of New Cerebral TIA at Follow-up in Elderly Patients With and Without Extracranial Internal or Common Carotid Arterial Disease

	New TIA		Mean Follow-up (Months)	Average Annual Incidence (%)
	No.	(%)		
80%–100% ECD	2/37	(5%)	24 ± 15	(3%)
40%–80% ECD	9/113	(8%)	40 ± 19	(2%)
0%–40% ECD	14/799	(2%)	47 ± 14	(1%)

TABLE III. Prognostic Variables for New ABI and Their Regression Coefficients in the Cox Proportional Hazard Model

Variable	Regression Coefficient	Standard Error	p Value	Relative Risk
Severity of ECD*	0.930	0.110	0.000	2.534†
Prior ABI	0.754	0.152	0.000	2.126
Age	0.024	0.010	0.012	1.024
Sex‡	−0.045	0.160	0.777	0.956

*ECD = extracranial internal or common carotid arterial disease.

†Relative risk = 2.534 comparing 40%–80% ECD with 0%–40% ECD, 2.534 comparing 80%–100% ECD with 40%–80% ECD, and 6.421 comparing 80%–100% ECD with 0%–40% ECD.

‡0 if men, 1 if women.

TABLE IV. Prognostic Variables for New Cerebral Transient Ischemic Attack and Their Regression Coefficients in the Cox Proportional Hazard Model

Variable	Regression Coefficient	Standard Error	p Value	Relative Risk
Severity of ECD*	1.041	0.291	0.001	2.832†
Sex‡	−0.810	0.406	0.050	0.445
Age	0.040	0.027	0.127	1.041
Prior ABI	0.640	0.420	0.133	1.897

*ECD = extracranial internal or common carotid arterial disease.

†Relative risk = 2.832 comparing 40%–80% ECD with 0%–40% ECD, 2.832 comparing 80%–100% ECD with 40%–80% ECD, and 8.020 comparing 80%–100% ECD with 0%–40% ECD.

‡0 if men, 1 if women.

DISCUSSION

The mean age of the population in this study was 82 years, whereas the mean ages of the patients in some other studies were 65 years for men and 63 years for women,[1] 62 years,[2] and 60 years.[3,4] These studies[1–4] included asymptomatic patients, whereas 30% of the population in the current study (288 of 949 patients) had an earlier ABI.

The severity of ECD was the prognostic variable with the highest relative risk for predicting new ABI or new TIA. After we controlled for other prognostic variables, the patients with an earlier ABI had a 2.1× higher probability of developing new ABI and a 1.9× higher chance of developing new TIA than those without an earlier ABI. Age was also a prognostic variable for new ABI, and male sex was a prognostic variable for new TIA. Although significant ECD increased the incidence of ABI or TIA, 141 of 213 cerebrovascular events (66%) developed in patients without significant ECD.

REFERENCES

1. Chambers BR, Norris JW: Outcome in patients with asymptomatic neck bruits. *N Engl J Med* 1986; 315:860–865.
2. Hennerici M, Hulsbomer HB, Rautenberg W, et al: Spontaneous history of asymptomatic internal carotid occlusion. *Stroke* 1986; 17:718–722.
3. Bogousslavsky J, Despland PA, Regli F: Asymptomatic tight stenosis of the internal carotid artery: Long-term prognosis. *Neurology* 1986; 36:861–863.
4. Ford S, Frye JL, Toole JF, et al: Asymptomatic carotid bruit and stenosis: A prospective follow-up study. *Arch Neurol* 1986; 43:219–222.
5. Schoenfeld MR, Aronow WS, Paul P: A prospective study of the neurologic outcome of carotid stenoses amongst the elderly. *J Cardiovasc Tech* 1989; 117–120.
6. Schoenfeld MR, Aronow WS, Paul P: Are strokes more likely to result from severe carotid atherosclerotic stenoses? If not, why not? *Angiology* 1988; 39:720–724.

AIDS prevention by needle exchange

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Intravenous drug use is the second most common risk factor for the acquisition of the human immunodeficiency virus (HIV) in the United States, resulting in 23% of all adult cases of the acquired immunodeficiency syndrome (AIDS).[1] In New York City, and in many other large American and European cities, intravenous drug use has become the foremost risk factor for HIV infection; at least 60% of New York City's estimated 250,000 intravenous drug users (IVDUs) are believed to be infected.[2]

Reducing transmission of HIV among IVDUs, their sexual partners, and their children is a crucial element of the public health battle against AIDS. Questions about how best to achieve this reduction, however, remain a constant source of debate. Controlled studies of IVDUs demand meticulous planning as well as sufficient time, funding, and public support. Governments and funding agencies have impeded many programs and research projects in their early stages; grass roots movements, on the other hand, face criticism for their questionable legal status and lack of a factual foundation.[3–6]

The major proposals that have emerged from this debate are HIV antibody testing, drug treatment, education, and needle distribution.[7] Although a combination of all these approaches would be most effective, each presents dilemmas of its own: Should testing be voluntary or mandatory? How can we protect against discrimination? What is the optimal drug treatment program? What kind of education will most likely effect behavioral change? What is the best way to distribute clean needles? How effective are needle exchange programs? This paper focuses exclusively on the last two questions.

The Needle Exchange Principle

Needle exchange involves the one-for-one trade of a used needle for a new one. This increases the probability that any given needle in the environment is sterile, without increasing the total number of needles in circulation or facilitating drug use. Exchanging needles also ensures that needles traded in are disposed of safely, reducing the pool of needles that would otherwise be left behind in public places or discarded in a potentially harmful way, a benefit not derived from selling needles in pharmacies or vending machines. Furthermore, most needle exchange programs use the repeated encounter with IVDUs as an intervention point for the distribution of information about treatment options and for AIDS education, which may encourage voluntary HIV antibody testing. Since the same population is also at risk for contracting HIV from, or passing it on to, sexual partners and offspring, free condoms are frequently offered in the exchange. Finally, needle exchange programs serve populations of IVDUs who, in return, provide information for behavioral, epidemiologic, and ethnographic studies of the interplay between drug use and infectious diseases.

Needle exchange programs are predicated on four underlying concepts: sharing needles transmits HIV; many IVDUs share needles; they share because needles are difficult to obtain; they are motivated to stop sharing if another option exists.[8] Studies have validated each of these premises.

That sharing needles among IVDUs effectively passes blood, and HIV, from one person to another is well proven.[9,10] A study in New York City revealed that 53% of IVDUs interviewed on the street reported that they usually or always shared needles.[11] In a study of a needle exchange in Tacoma, WA, 154 IVDUs were interviewed about their drug use during the month before their enrollment in the exchange. On average, the IVDUs in the study reported obtaining used syringes 57 times per month per person, and they passed on used syringes at an average rate of 100 times per month per person.[12] These studies support the notion that IVDUs commonly share needles. Nevertheless, since these figures represent only IVDUs who use the exchange, usually males with a mean duration of drug use of 15 years, they may not be representative of individuals whom the program does not commonly attract, such as women or those who have recently started intravenous drug use. Obtaining information about the practices of IVDUs outside of the exchange is difficult, and follow-up is almost impossible.

By far, the most common reason for needle sharing reported by IVDUs is difficulty in obtaining needles and syringes. In a study in New York City, 46% of IVDUs interviewed from a sample of prison inmates and methadone treatment patients stated that they shared needles mostly because they could not obtain clean ones.[13] In Melbourne, Australia, 66% of IVDUs interviewed shared needles because they did not have a clean needle when they needed it.[14]

The difficulty in obtaining needles stems mainly from legal and distributional obstacles. In 48 states, the possession of needles is illegal under drug paraphernalia laws. Since carrying needles puts IVDUs at risk of arrest, many are motivated to go to "shooting galleries" (usually abandoned buildings, where IVDUs congregate) to share used contaminated needles.[15] In addition, because of the shortage of needles on the streets, drug dealers have attempted to meet the demand by selling repackaged used needles and syringes that are unsterile and possibly contaminated by

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HIV.[16] A report on street interviews conducted in New York City showed a remarkably high demand for clean needles, citing that illicit needle vendors on the street obtain between 50% and 100% profits on the sale or rental of needles that they either stole, obtained illegally, or resold without sterilizing.[11]

In general, IVDUs are aware of AIDS risk behaviors, and they are motivated not to share if sufficient needles are available. One recent study, not involving needle exchange programs, clearly demonstrated the influence of access to sterile injection equipment on HIV transmission. The study compared the HIV seroprevalence of diabetic versus nondiabetic IVDUs. Out of a group of 2,921 IVDUs, it was found that diabetic individuals had a significantly lower HIV seroprevalence (9.8%) than nondiabetic individuals (24.3%). Both groups reported similar intensity and duration of drug use and similar sexual practices, but diabetic IVDUs were less likely to share their "works" (drug injection equipment) and attend shooting galleries than nondiabetic IVDUs. The researchers believed that this difference was due to the safer injection practices of diabetic IVDUs, who had easy access to sterile needles and syringes.[17] Critical evaluation of this study reveals a potential source of bias in its methodological design: diabetic IVDUs, having more interaction with the medical community than nondiabetic IVDUs, may be more aware of AIDS risks, and this difference may account for their safer injection practices. A study in New York City, however, showed that 97% of a random sample of 261 IVDUs knew that HIV could be transmitted between two individuals sharing a needle.[13] Clearly, lack of information is not the cause of dangerous injection practices.

OPPOSITION

It is worthwhile to consider the reasoning and motivations of those who oppose the principle of needle exchange on ideological grounds. Unlike other public health measures designed to curtail the spread of infectious disease, such as the inspection of water supplies or the establishment of cleanliness standards for restaurants, needle exchange necessarily invokes several complicated and controversial public policy issues, namely, drug use and addiction, minority group membership and racism, law enforcement, and AIDS.

Although needle exchange programs have existed in Europe and elsewhere for many years, actually predating the AIDS epidemic in certain countries (where they were established to counter hepatitis B), action was delayed in the United States. Interestingly, disapproval of the needle exchange concept stems from both extremes of the liberal-conservative continuum. President George Bush has stated that he opposes the distribution of clean needles "under any circumstances."[3] Many Democratic and Republican congressional leaders also oppose needle exchange. Representative Charles B. Rangel, Democrat of Manhattan, head of the Select Committee on Narcotics Abuse and Control and head of the National Council on Drug Abuse, has refused to fund needle exchanges, saying, "Needle exchange is a way of sweeping addicts under the rug, instead of restoring their dignity and giving them drug free lives."[5] This statement implies that drug users do not have dignity and want to lead drug free lives, an arguable point. Even more extreme is the position of City Councilman Hilton B. Clark of Harlem, who accused Stephen C. Joseph, then New York City's Commissioner of Health, of using clean needle programs to conduct a "genocidal campaign against black and Hispanic people."[6] New York City's former Police Commissioner, Benjamin Ward, opposed needle exchange, stating, "As a black person, we [sic] have a particular sensitivity to doctors conducting experiments, and they too frequently seem to be conducted against blacks."[6]

One author has pointed out that some opponents of needle exchange, who have long valued a strong police force in the war against drugs, criticize needle exchange programs for cooperating with IVDUs, including drug dealers. The argument is that handing out free needles condones illegal drug use, encourages new IVDUs, and prevents long-time users from quitting.[18] The issue of whether or not acceptance of needle exchange signals cooperation with drug users or condones illegal drug use is a matter of moral inquiry; specific data exist, however, to refute claims that needle exchange programs increase drug use or prevent abstinence.

There are also ethnographic studies, widely cited by those opposing needle exchange programs, observing that the sharing of works is an indelible cultural aspect of the drug subculture because it engenders a sense of trust and bonding in an arena where mistrust and competition prevail.[19] These studies lead certain opponents to argue that IVDUs cannot and will not change their habits; therefore, needle exchange programs are destined to fail. A survey of 100 IVDUs conducted in New Haven, CT, showed that, contrary to this belief, addicts did not share needles as part of a drug culture ritual, but instead shared because of their fear of arrest for possessing needles and because of the scarcity and cost of needles sold illegally on the streets.[20] One researcher noted that "sharing" is a misleading term: the use of contaminated needles is called "going after" or "using someone else's works," by IVDUs themselves.[15] In other words, what most researchers term "sharing" is really what IVDUs consider a desperate attempt to find any needle when one is needed.

ESTABLISHED PROGRAMS

The most well-established needle exchange programs exist in Europe and Australia, where criminalization of needle possession is not nearly as common as it is in the United States. A group of IVDUs in Amsterdam created the first program in 1984 as a means of reducing transmission of the hepatitis B virus. The program was greatly expanded in 1985 when HIV antibody testing of previously collected serum samples of IVDUs revealed that HIV was already prevalent.[21] The program currently serves approximately 3,000 IVDUs and exchanges 700,000 needles per year.

The government of the United Kingdom established 15 pilot programs in 1987 with the intent of closely monitoring their results.[22] The number has grown to over 70, ranging from shopfronts, caravans, medical sites, to special agencies.[8] Australia established its first programs in 1986; there are now more than 100 spread throughout the country. Almost two million sterile needles and syringes are distributed, exchanged, or sold each year in New South Wales.[23]

The first needle exchange program in North America was created in Tacoma, WA, in 1988 by David Purchase, a former drug counselor. He based the operation on private donations and ran it himself from a card table set up near a local shooting gallery. Popular support for the program, which provided AIDS and drug treatment information and distributed bleach and condoms in addition to needles, led the local health department to conduct a survey of exchange participants. The local police force cooperated with the exchange in spite of drug paraphernalia laws by not confiscating the needles of IVDUs in the program. After several months of operation, the Tacoma-Pierce County Health Department assumed official responsibility for the program. The Tacoma Syringe Exchange maintains a nonjudgmental attitude toward IVDUs and accepts anonymous participation. Presently, an estimated 550 different IVDUs visit the two exchange locations weekly.[12]

In spite of heated opposition, New York City's needle exchange pilot program (NEPP) operated for ten months beginning in November 1988. The NEPP, designed specifically to address the feasibility and success of a needle exchange in New York City, was designed and promoted as a "bridge to treatment." It served 294 people and generated much data before it was discontinued by the Dinkins administration. Unlike other American and most European exchanges, the NEPP was a negative example of how to run an effective program. Admission procedures were complicated, services such as tuberculosis testing were mandatory, and the NEPP was located on the first floor of the New York City Department of Health building. This site, surrounded by criminal justice facilities and far from most IVDUs' homes, was not user-friendly.[24] Unofficial groups in New York City, such as ACT-UP (The AIDS Coalition to Unleash Power) have developed unapproved needle exchanges of their own since the closing of the NEPP.

The New Haven Needle Exchange Program, in cooperation with Yale University researchers, is the most well-evaluated program in the United States. Connecticut passed legislation in 1990 allowing needle exchange programs, with the knowledge that the vast majority of AIDS cases in New Haven and other major Connecticut cities are directly or indirectly attributable to intravenous drug use. The program operates six hours a day, four days a week, out of a brightly painted van that visits several sites known for high levels of illicit drug use. Each of the more than 700 participants is given a card with a fictitious nickname that he or she presents to exchange workers at each visit. In this way, participation is anonymous, but data can be collected accurately on individuals. All syringes are marked with a different number as part of a unique syringe tracking and testing system, and a limit of five syringes are exchanged at one time.[20] The report of the program's eight-month study was released in July 1991, the results of which have led New York City government officials to reconsider needle exchange.

Needle exchange programs, both legal and illegal, exist in several other United States cities, including Portland, OR; Seattle, WA; and Boulder, CO. Prevention Point, in San Francisco, was established in 1988 and serves the largest number of IVDUs in the country. The legal status of Prevention Point remains in question, but the government has not interfered with this program, presumably because of its success.[21]

EVALUATING NEEDLE EXCHANGE

Assessing the public health benefits of a needle exchange program involves asking three questions: Does the program meet its intended goal of establishing clientele and exchanging needles? Does the program decrease HIV-risk behavior among its clients? Does the program bring about a decrease in HIV seroconversion and seroprevalence? In addition, there must be clear evidence that needle exchange programs do not increase intravenous drug use.

All of the programs mentioned have established sufficient-numbers of clientele, commonly enrolling far more IVDUs than expected. The New Haven program, for example, started out with a goal of enrolling 300 IVDUs in the first year. In the first seven and a half months of operation, however, the program had already enrolled over 700 IVDUs.[20] The busiest needle exchange program in the United Kingdom, The Exchange, London, attracted on average 257 clients per month, with whom a monthly average of 8,950 needles were exchanged in 1987 and 1988.[25]

It is evident that many needles are exchanged, but are programs getting back the needles they give out? The ideal program, in which 100% of distributed needles are returned promptly, implies a consistent use of clean needles, lower probability of sharing, and safe disposal. This question was addressed in a study in San Francisco (Prevention Point). Half of the distributed needles (2,068/4,239) were returned within two weeks of distribution, and 61% were returned during the study period of nine weeks.[26] At The Exchange in London, return rates of 51% to 90% have been achieved for any given month, with a mean of 77% over a one year period.[25] During the eight months of the New Haven study, 52% of the distributed needles (5,263/10,180) were returned. This figure, does not take into account needles that were distributed at the end of the eighth month and returned afterwards.[20]

That a client is sufficiently motivated to exchange a used needle for a sterile one implies a desire to reduce HIV risk factors and to make positive behavioral changes. Positive changes in behavior can be classified as any one or more of the following: decreased use of used needles, decreased needle sharing, decreased risky sexual behavior, decreased drug use, or increased willingness to enter treatment. In spite of the obvious methodologic obstacles in studying these variables, evidence of positive behavioral change is accumulating.

Data from the 15 UK government-sponsored NEPPs indicate that many clients adopted or maintained low-risk behaviors: 79% sustained or adopted low or lower levels of syringe sharing. Many clients also reported changes in sexual behavior; those with sexual partners decreased from 77% to 69% of the sample, and those with two or more sexual partners decreased from 26% to 21%.[22]

After ten months of operation, the New York City NEPP reported that 81% of clients reported having shared needles before the exchange, and 62% having shared needles within the past 30 days. After receiving needles and revisiting the program at least once, only 5% reported sharing since their last NEPP contact. Furthermore, 78% accepted enrollment into treatment programs, mostly methadone maintenance.[24] In the New Haven study, 188 clients asked for help finding treatment. Of these clients, 57% entered treatment programs. Because of an inadequate number of treatment spots, many clients who requested treatment were not able to receive it.[20] Judging the success of this program's placement of IVDUs into treatment is difficult because there was no control group for comparison, such as IVDUs not enrolled in the program who requested treatment from other sources.

In a study of the Tacoma needle exchange, a 76% reduction in use of potentially contaminated equipment and a 63% reduction in passing on needles after use was reported among interviewed IVDUs since enrollment.[21] No increase in intravenous drug use was reported among clients. The mean number of injections per month pre-exchange (151) was almost identical to the reported number of injections after participating in the exchange (150).[12]

Whether or not needle exchange programs bring about reductions in HIV seroconversion or seroprevalence is the central issue, the truest measure of the utility and public health importance of needle exchange. After all, it is theoretically possible that the programs are well attended and effect risk reduction but do not solve the original problem. Researchers from The Exchange in London published the first study demonstrating a direct link between HIV status and reported behavior indexes among needle exchange clients; they also performed repeat testing of a cohort to evaluate directly prevention of HIV infection. Of 133 IVDUs interviewed and tested (from saliva samples by anti-HIV immunoglobulin-G antibody capture radioimmunoassay, or GACRIA), only two seroconversions occurred at the end of one year of the study. The actual infection times of these two individuals were likely to have occurred before starting the exchange.[25] Unfortunately, there was no control group in this study. We do not know therefore how the seroconversion rate of the group compares with that of IVDUs not enrolled in an exchange program.

The Tacoma needle exchange has demonstrated a non-statistically significant trend toward lower HIV seroprevalence among IVDU exchange users (3%) compared to a group of IVDU nonexchange users (8%) during eight months of exchange use,[27] as well as a decline in hepatitis B incidence among IVDUs in the city since the start of the exchange.[28] The incidence of hepatitis B virus has been proposed as a marker for needle sharing and HIV transmission among IVDUs. The Tacoma program represents the third example of a decrease in hepatitis B virus in an area with a major exchange program.

Yale University researchers at the New Haven needle exchange used a mathematical model to project that the program is already reducing the incidence of new HIV infections in the city by 33%. Incidence was calculated from data produced by the syringe tracking and testing system, which tests needles for the presence of HIV using the polymerase chain reaction. One part of the study involved testing needles from different sources. Needles taken from shooting galleries were found to be 91.7% HIV positive; needles from the street were 67.5% positive; needles distributed by and returned to the exchange were 50.3% positive; and, of these exchange needles, those determined by the tracking system to be "dedicated," or unshared, were 44.5% positive.[20] These comparisons clearly illustrate the risks of using needles from shooting galleries or from the street, and point out the necessity of replacing these needles with sterile ones from the exchange program. This study also indicates the potential financial benefits of prevention by needle exchange versus treatment of AIDS. The New Haven program, operating at a total cost of around $150,000 per year, offers the promise of preventing many new HIV infections and premature deaths on a city wide level. In contrast, the medical cost of caring for one person with AIDS for one year can easily exceed $35,000.[20]

SOURCES OF ERROR AND BIAS

Overall, the data from these studies support the notion that needle exchange programs reduce HIV risk-behavior and HIV transmission, without promoting or increasing intravenous drug use. The methods of evaluation, however, are not perfect. Most data about reduction in needle sharing, for example, are obtained from interviews with IVDUs, who may not report their use of drugs and behavioral patterns accurately. In addition, it is difficult to discern changes in behavior arising from a needle exchange program from those brought about by increased awareness and education from other sources. A study from Cleveland has shown that AIDS and risk-reduction educational sessions given by a health educator in a one-on-one format can reduce the sharing of syringes. Three months after educational sessions, the percentage of IVDUs in the study who reported sharing needles decreased from 67.4% to 24.3%.[29] These results suggest that AIDS education should be combined with needle exchange programs. The effectiveness of education in this study, however, also shows that the increasing amount of information about AIDS conveyed by the media and within the public in general may contribute to the decrease in sharing that is attributed to needle exchange programs alone.

Another problem in evaluating needle exchange programs stems from the fact that IVDUs participating in exchanges are self-selected, usually males with a long history of intravenous drug use. IVDUs who are motivated enough to participate in needle exchange may be more willing or able to change behavior, including sexual practices, than IVDUs who do not choose to participate. Increased use of the condoms distributed by many needle exchange programs, for example, may also be a complicating factor in assessing the impact of sterile needles on HIV transmission.

CONCLUSIONS

Although the concept of needle exchange is relatively new to the United States, and much definitive data have yet to be presented, some conclusions may be drawn. Needle exchange programs can be run efficiently and cost effectively. They attract and satisfy drug users who would otherwise not be part of other services, opening the channels of education and treatment. Most important, needle exchanges, without increasing drug use or preventing abstinence, give IVDUs the means to adopt and maintain drug injecting behaviors that reduce HIV risk. Preliminary evidence exists to suggest that the lowering of these risk factors leads to decreases in the transmission of HIV among IVDUs, their sexual partners, their drug injecting partners, and their children.

Needle exchange programs in the United States are likely to be more effective without the existing drug paraphernalia laws, as in many European countries. Under these laws, IVDUs who faithfully exchange their needles will remain apprehensive about carrying injection equipment and may still feel the need to seek needles at shooting galleries or from other IVDUs. Repeal of drug paraphernalia laws in this country, however, is not likely unless needle exchange programs are accepted first.

Political opposition to needle exchange programs stems mostly from moral, racial, and emotional concerns and not from clear and logical studies of the data. This is not to be belittled. Culturally sensitive studies need to be performed that address the concerns and worries of leaders who oppose, and who often stand in the way of, large-scale needle exchange programs.

REFERENCES

1. Center for Disease Control: HIV/AIDS surveillance. *February*, 1992, 9.
2. AIDS morbidity and mortality. *NY State J Med* 1990; 90;572–575
3. Ginzburg HM: Needle exchange programs: A medical or a policy dilemma? *Am J Public Health* 1989; 79:1350–1351
4. Lewis NA: Koch cancels needle test near school. *NY Times*. November 3, 1988.
5. Johnson K: New Haven plans to give drug addicts new needles. *NY Times*. May 24, 1990.
6. Marriott M: Needle exchange angers many minorities. *NY Times*. November 7, 1988.
7. Brickner PW, Torres RA, Barnes M, et al: Recommendations for control and prevention of human immunodeficiency virus (HIV) infection in intravenous drug users. *Ann Intern Med* 1989; 110:833–837.
8. Stimson GV: Syringe-exchange programmes for injecting drug users. *AIDS* 1989; 3:253–360.
9. Des Jarlais DC, Friedman SR, Hopkins W: Risk reduction for the acquired immunodeficiency syndrome among intravenous drug users. *Ann Intern Med* 1985; 103:755–7591.
10. Marmor M, Des Jarlais DC, Cohen H: Risk factors for infection with human immunodeficiency virus among intravenous drug users in New York City. *AIDS* 1987; 1:39–44.
11. Hopkins W: Needle sharing and street behavior in response to AIDS in New York City, in Battjes RJ and Pickens RW, eds: *Needle Sharing Among Intravenous Drug Abusers: National and International Perspectives*. Washington, NIDA Research Monograph 1988; 80:18–27.
12. Hagen H, Des Jarlais DC, Purchase D, et al: The Tacoma Syringe Exchange, in Friedman SR and Lipton DS, eds: *Cocaine, AIDS, and Intravenous Drug Use*. New York, The Haworth Press, Inc. 1991.
13. Selwyn PA, Feiner C, Cox CP, et al: Knowledge about AIDS and high-risk behaviour among intravenous drug users in New York City. *AIDS* 1987; 1:247–254.
14. Paine S, Tonuma M, Monheit B: AIDS in drug abusers [letter]. *Med J Australia* 1985; 143:631.
15. Fernando D: Sterile needles: Human immunodeficiency virus in diabetic intravenous drug users [letter]. *JAMA* 1991; 267:1340.
16. Des Jarlais DC, Friedman SR: AIDS and the sharing of equipment for illicit drug injection: A review of current data. Albany, New York State Division of Substance Abuse Services, 1987; pp9–10.
17. Nelson KE, Vlahov D, Cohn S, et al: Human immunodeficiency virus in diabetic intravenous drug users. *JAMA* 1991; 266:2259–2261.
18. Imperato PJ: Needle exchange and the prevention of AIDS. *J Comm Heal* 1989; 14:61–63.
19. Connors MM, Galea RP: Anthropological investigation of the meaning and practices of needle use and sharing among intravenous drug users (IVDU) (Abstract 8518). *Proceedings of the IV International Conference on AIDS*. Stockholm, 1988.
20. O'Keefe E, Kaplan E, Khoshnood K: *Preliminary Report: City of New Haven Needle Exchange Program*. New Haven, CT, New Haven Health Department, July 31, 1991.
21. Des Jarlais DC, Friedman SR: AIDS and legal access to sterile drug injection equipment. *Ann Am Acad Pol Soc Sc* (in press).
22. Donoghue MC, Stimson GV, Dolan K, et al: Changes in HIV risk behaviour in clients of syringe-exchange schemes in England and Scotland. *AIDS* 1989; 3:267–272.
23. Wodak A: Australia: Preventing HIV spread among injecting drug users. *Lancet* 1991; 337:482.
24. New York City Department of Health: The pilot needle exchange study in New York City: A bridge to treatment. December 1989.
25. Hart GJ, Carvell ALM, Woodward N, et al: Evaluation of needle exchange in central London: Behaviour change and anti-HIV status over one year. *AIDS* 1989; 3:261–265.
26. Guydish J, Clark G, Garcia D, et al: Evaluating needle exchange: Do distributed needles come back? *Am J Public Health* 1991; 81:617–619.
27. Hagan H, Des Jarlais DC, Purchase D, et al: Lower HIV seroprevalence, declining HBV incidence and safer injection in relation to the Tacoma Syringe Exchange (Abstract 3291). Delivered at the Seventh International Conference on AIDS, Florence, Italy, June 1991.
28. Hagan H, Des Jarlais DC, Purchase D, et al: The incidence of HIV infection and syringe exchange programs [letter]. *JAMA* 1991; 266:1646–1647.
29. Stephens RC, Feucht TE, Roman SW: Effects of an intervention program on AIDS-related drug and needle behavior among intravenous drug users. *Am J Public Health* 1991; 81:568–571.

The experience of an American medical student with an Aboriginal community in central Australia

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As a fourth year medical student, I spent eight weeks living and working among Aboriginal people at the Papunya settlement in central Australia in March and April of 1992. This experience was made possible through an international health elective sponsored by the Department of Preventive Medicine and Community Health, State University of New York, Health Science Center at Brooklyn. It was funded by the Alumni Fund of the Center's College of Medicine.

Aboriginal people have maintained their spiritual, cultural, and tribal identity in spite of numerous attempts at integrating them into white Australian society. Their native languages are still the primary tongue, with some people speaking only a few words of English. They hunt *tucker* (food) in the harsh bush of the Tanami Desert and consult the *ngangkaris* (medicine man) when illness strikes. When the Europeans settled Australia in the 1800s and 1900s, the Aborigines were exploited and murdered, their lands plundered, and their spirit trampled. The legacy of that era is carried today; by the "white fella," who feels guilty and responsible for righting the wrongs of the past and the "countrymen," who are resentful of the attempts made at destroying their culture and their dependence on government handouts. It was in this climate that I began to understand the complexity of the issues and learned of the demoralization and disenchantment of a people.

ABORIGINAL CULTURE TODAY

Aboriginal culture is the oldest on earth. The first archeological site dates back to 35,000 BC in New South Wales. Aborigines probably entered from Indonesia during one of the Ice Ages, when the shallow seas in northern Australia dried out, allowing for movement between Australia, Indonesia, and New Guinea. In spite of the theories that they arrived on the island of Australia via Indonesia, no association can be traced from native populations in the north and Aborigines. Genetic and linguistic studies have failed to show any connection between Aborigines and any other group on earth.

The Aboriginal people who I came to know during my stay at Papunya still hold strongly to their ancient customs and traditions. The Aborigines are hunters and gatherers. They live off of the arid land of central Australia, eating kangaroo, goana, and dingo. They do not raise any livestock of their own. Because food is scarce during periods of drought (which occur fairly often), each tribe once had to roam huge expanses of land in order to feed their families. Periods of starvation and dehydration occurred frequently, perhaps explaining their predisposition to diabetes mellitus. Today, they still hunt the great desert for food.

The land holds more for aborigines than just a source of physical sustenance. They endow it with profound spiritual qualities. The mythology that they associate with certain landmarks is called the Dreamtime. They depict the stories and mysteries of their Dreaming in their art, which also serves as a means of communication. It is their written language, so to speak. When they were forbidden from roaming around their native territories, more was lost to them than food and water. Their identity and history lie within the mountains and dry river beds of the bush.

Because of complex individual and group relationships, political, and social units are difficult to distinguish. For the sake of identity, I will call the largest unit a tribe. All of its members share a common language, but dialects exist within subgroups. Each tribe consists of anywhere from 100–1500 members. One hundred and twenty-six tribes claim land in the Northern Territory, most of them in the densely populated Top End, near coastal waters.[1(px)] The remaining intact tribes live in the central regions, where the enormous land expanses protected them from annihilation. The Walpiri, a tribe with which I worked, claim 138,000 km of bush as their native territory. Within the tribe no strict hierarchical or leadership roles exist, although an older man usually exercises the greatest social and political influence.[2(p184)]

The relationships among the members of each tribe is amazingly complicated. Each member of the tribe is related to the others either by blood or through their "skin name." In the Walpiri tribe, there are sixteen skin names; eight women's names, each beginning with the letter "n," and eight men's, each beginning with the letter "j." For example, a girl with the name Nangala has a brother named Jangala. A Nangala may only marry a man named Jungarrayi, and a Jangala may only marry a woman named Nungarrayi. Generally, pairs of sisters marry pairs of brothers. A very close knit family structure results. The daughter of a Nangala and Jungarryi is named Napalgarryi, and a son, Japalgarryi. All women named Nagala are "mother" to all girls named Napalgarryi and all boys named Japalgarryi. No one has only one mother or grandmother, or one father. All members of the tribe are related through their skin names. The bonds are so strong that a Nangala mother will suckle another Nangala's child.

Child rearing is a team effort and only women's business.

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FIGURE 1. The author with a three-year-old child at Papunya.

Roughly, the mother is nurturer; the grandmother, caretaker; and the maternal aunts, disciplinarians. Each role can be played by the women of the tribe with the appropriate skin name, whether they are related or not. Men have nothing to do with children until the boys come of age.

Living arrangements follow three basic patterns. Sometimes old people camp together, women live in child-rearing groups, and extended families share a village. Usually 25–30 people live in a cluster. Living arrangements are constantly changing. At Papunya, the houses are identified by the oldest man in the house. For example, if we needed to find a child for an immunization, we would be told that "so and so" was staying at Long Jack's house. Very few people even live in houses. Most people set up makeshift corrugated aluminum structures called "humpies." Because mobility is a way of life, people do not amass belongings. Many people do not even own a second set of clothing. We did not dispense medication for longer than one week because when people move, it is spontaneous. They do have some modern conveniences such as televisions, guns, and cars.

Death alters the lives of every member of the tribe. When a person dies, their name is never mentioned. All members of the tribe with the same first name are called Culumjarryi, or no name. For example, if a woman named Nora dies, a woman named Nora Nangala will be called Culumjarryi Nangala. When a woman named Alice died, all the members of the tribe called Alice Springs, Culumjarryi Springs. One must be careful to ask "How is so and so's brother?" versus asking about him by name for fear that he died and you would upset his spirit by mentioning his name. The Aborigines still follow this tradition closely. Eventually, years later, the elders of the tribe will feel that the spirit has been put to rest and the name may be spoken again. Besides the name not being uttered, the person's dwelling must be abandoned. At Papunya, a shanty town of corrugated aluminum shacks and blankets thrown over tree branches lines the main road of the village. This is called the sorry camp, where families of the recently deceased live. Nearly half of the permanent houses in the settlement lie vacant as a result of death custom. Once the period of mourning is over, the permanent houses are re-inhabited.

ABORIGINAL HISTORY SINCE COLONIZATION

Aboriginal groups inhabited nearly all of Australia before the Europeans arrived. The coastal areas were the most densely populated, as well as the first to be overtaken by the new inhabitants. In the 1400s, the Dutch "discovered" Australia, but abandoned attempts at settlement because of the isolation of the island and their disastrous relations with the Aborigines.[1(p5)]

Before they left, the Dutch introduced the Macassans, a group of Indonesian people, to the Aborigines. The coast Aboriginal people and the Macassans began to trade. Perhaps it was this early contact with outsiders that has allowed Aborigines to sustain their existence through clashes and pressures by Europeans of the 19th and 20th centuries.

In 1788, Captain Cook and the British arrived on the eastern coast. Nearly all of the Aboriginal tribes that came into contact with the British were massacred, their spears no match for British bayonets.[3(p106)] The central Australian Aboriginal people did not come into contact with Europeans until 1855, when A. C. Gregory's land expedition found the Northern Territory to be "the most superior land to settle in all of central Australia."[1(pvii)]

In 1872, white settlement began en masse, when the overland telegraph was established, connecting central Australia with the rest of the country.[1(pvii)] Mining, gold digging, and cattle ranching were the major industries. Four thousand Chinese people were imported to begin building a North/South transcontinental railroad. Seventeen hundred kilometers from any coast, Alice Springs was established as a telegraph outpost. Probably because of the vastness of the land, the violence against the Aborigines on the east coast was not duplicated in central Australia. The greatest clashes occurred on cattle stations, from which Aborigines were banned. However, they were allowed to camp near the station homestead and trade their labor and women for food, tobacco, and clothes. They survived because their labor was desperately needed. By 1911, approximately 50,000 Aborigines inhabited the Northern Territory, while the non-aboriginal population numbered a little over 3,200.[4(p40)]

In 1932, gold, copper, bismuth, and silver were discovered at Tennant Creek, 500 km north of Alice Springs, changing the area profoundly. White people flocked to the area. With the Darwin to Alice Springs railroad completed by 1939, cattlemen could trade goods with the coast. The Aboriginal people maintained a completely separate existence, mixing only through labor. Aborigines had no rights to live on their own land and were not allowed to travel over these lands without permits. For nomadic people, this policy was quite an affront.[1(pviii)]

The government established its first assimilation policy in the 1920s. Part aboriginal children were taken from their mothers without consent and placed in "half-caste homes." The boys were trained for manual labor, and the girls were trained for domestic work. They were encouraged to mix with whites and breed out their color.[2(p182)] World War II heightened the Aboriginal people's awareness of the inequalities. Employed by the army, they lived in better conditions than ever before under white domination, and racism was less overt.[2(p182)] After World War II, the Stuart and Barkly highways were completed, improving communication and transportation throughout central Australia. Thus, began the modern era of Aboriginal/white conflict.

FIGURE 2. Performing audiometry at Mount Liebig, an outstation near Papunya.

THE SETTLEMENT MOVEMENT

The first settlement was established by Lutherans in 1877 at Hermannsberg, 70 kms west of Alice Springs.[5(pvi)] Obviously, the goal of the mission was to convert the Aborigines to Christianity. Anglicans, Catholics, and Methodists attempted to organize the Aborigines as well, but none of their attempts succeeded. Now under self-government, Hermannsberg still functions as one of the largest and most enduring of the communities. The next settlement to be opened was Haasts Bluff, at which missionaries supplied rations to women, children, and the elderly. Both Hermannsberg and Haasts Bluff served single tribal groups, whose members speak the same language and share a common ancestry. In 1953, the government assumed responsibility for operating Haasts Bluff, a settlement of 500. Six years later, its water supply became contaminated, necessitating a move to Papunya, 150 miles northwest of Alice Springs. It was the first settlement completely planned and built by the federal government.[1(px)] Staff houses, nurses quarters, a dining hall, a store, a power plant, an airstrip, an outpatient clinic, and two workshops were built. The lofty idea was to "bring Aboriginal people into Australian society, clothing, language, literacy, skills, and law."[1(pxi)] Members of at least five different tribes were herded together—the Arande, Walpiri, Pintubi, Loritja, and Pirjanjanjarri—each with their own language and customs. A white Australian superintendent operated the settlement and set policies.

By 1970, 20 government settlements were operating in the Northern Territory. They were designed to provide education, health services, and job training for Aborigines. By the mid-seventies, Papunya was determined to be a hopeless disaster. Aboriginal social discontent and disruption led to delinquency, vandalism, and drunkenness. Traditional relationships broke down and despair set in. Attempts at trying to assimilate Aborigines into white culture only led to further alienation.

ABORIGINAL HEALTH CARE

Aboriginal health care has been one of the most controversial and volatile problems on the settlements. Only within the last ten years have western health professionals come to recognize that an understanding of Aboriginal attitudes and beliefs about health must be incorporated into treatment.[6(p15)] For example, Aboriginal understanding of gastrointestinal disease is as follows. The Walpiri believe that each of us has a *kurrunpa* (spirit) that lives in our stomachs. When the wind blows or someone has an evil thought about the victim, the *kurrunpa* is displaced, resulting in diarrhea or vomiting. It may move around inside the body, or it may leave the body all together. For the patient to get better, his *kurrunpa* must return to its right place. The *ngungkarri* (medicine man) is the person who knows where the *kurrunpa* has moved, when it will return, and how to get it back. At Papunya, patients still consult the *ngungkarri* about their *kurrunpa.* To them, oral rehydration solution makes no sense. Yet, we are angry and shocked when it is not used.

FIGURE 3. Walpiri women performing a spiritual dance.

Each settlement is equipped with an outpatient clinic. The clinic at Papunya is housed in the administration building, the largest building on the settlement, located in the middle of the main compound. Spacious, air-conditioned, and with a functioning toilet, it is the social center of the community during the hot days. A treatment room, an examination room, and an office form a ring around the central area, which is used for breast feeding, observing patients, and communing. No microscope, EKG machine, X-ray equipment, or blood tests are available. The most sophisticated instrument is the centrifuge. All diagnoses are made on clinical grounds alone, with patients being flown into Alice Springs if tests are needed.

The outpatient clinics are operated by nurse practitioners, who are also certified midwives. Employed by the Commonwealth Department of Health, they oversee the daily activities of the clinic, dispense medication, and treat emergencies. Nurses also make weekly visits to the outstations, satellite communities settled by Aborigines after the Aboriginal Land Rights Act allowed them to return to their native lands. Employed by the Royal Flying Doctors Service, the doctors (District Medical Officers) visit each settlement fortnightly or monthly and conduct research projects on Aboriginal health. They are available by telephone for consultation when the nurses have management questions or need to evacuate patients for hospitalization.

Aboriginal health problems are overwhelming and complicated. Comprehensive data regarding morbidity and mortality are lacking, but a few studies done in the 1960s and followed up in the 1980s reveal gross inequalities in the health status between Aborigines and white Australians. In 1960, 150 infants died per 1,000 live Aboriginal births. This shocking statistic focused collective attention on improving prenatal care and nutrition for pregnant mothers. By 1981, the rate had decreased to 26.4 deaths per 1,000 live births, a vast improvement but still nearly three times that of the non-aboriginal population.[4(p40)] On the other end of the spectrum, the average life expectancy of an Aboriginal adult is 52.7 years (1978), with people dying most commonly of circulatory system ailments and pneumonia.[4(p42)]

Likewise, morbidity among Aborigines exceeds that of non-aborigines. The rate of hospitalization is twice that of non-aborigines and the length of stay longer, indicating that these patients are sicker on admission.[7(p940)] Their pattern of disease follows that of third world countries; most of their conditions are rare among white Australians.

The diseases seen most frequently among Aborigines at Papunya fall into three categories: infectious diseases, diseases of life-style, and psychiatric problems. Gastroenteritis is the number one cause of morbidity and mortality among the infants at Papunya. Poor personal hygiene and malnutrition predispose these infants to fecal-oral illnesses. People do not use toilets, and nearly everyone walks barefoot. Showering daily, washing one's hands after using the toilet, and changing clothes regularly are not practiced, nor is it understood that these habits will prevent the spread of disease. Babies are breast fed until they are two or three years old, not beginning solid food until breast feeding ceases. With malnutrition rampant throughout the adult community, mothers often cannot produce enough milk to sustain a two-year-old child. The resulting childhood protein malnutrition leaves these children unable to fight otherwise benign illnesses.

Other infectious diseases common among Aboriginal children at Papunya are *Chlamydia trachomata* and gonorrheal conjunctivitis. Trachoma persists in areas of poor hygiene and is the leading cause of blindness in the area.[8(p162)] I found trachoma to be the single most devastating ailment on the settlement because its prevention, so simple, merely washing the eyes daily, and its consequences, so devastating. Gonorrheal conjunctivitis spreads among families like wildfire. During my second week at the clinic, I went with one of the nurses to visit the home of two newborn twins who had gonorrheal conjunctivitis. The entire household of 30 people needed to be treated with penicillin.

Sexually transmitted diseases are a serious problem. As in the rest of the world, both gonorrhea and syphilis are on the rise. Fortunately, the gonococcus in this community is still sensitive to penicillin. Sexual promiscuity and polygamy contribute to the spread of these diseases. Pelvic inflammatory disease has been such a major problem that some epidemiologists believe that it has served as a contraceptive. I did not see any cases of acquired immunodeficiency syndrome at Papunya.

Life-style diseases, including malnutrition, diabetes mellitus, obesity, and hypertension plague the Aboriginal people. Except for malnutrition, I know less about these diseases because I had little contact with the doctor who visited the community and handled these problems. The problem with malnutrition stems from the change in eating habits experienced by Aborigines with the development of settlements. They no longer eat bush foods, the only diet they ever knew. They now obtain 90% of their food from the local store. They collect their government pension checks on the settlement and are expected to spend them at the local store, the only store between Papunya and Alice Springs. Expensive and poorly stocked, most people buy soda, potato chips, and ice cream. No nutritionists live at the community to teach well-balanced diets.

The most devastating psychiatric problem is alcoholism. In my estimation, 90% of adult Aboriginal people at Papunya suffer from alcoholism. *Grog* (alcohol) has had the greatest negative influence on their social condition since colonization. In Alice Springs, hundreds of Aborigines have abandoned their land and their families, living in the dry bed of the Todd River, drinking themselves to death. At Papunya, we flew numerous patients into town for tests and treatment, only to have them check out of the hospital against medical advice to drink. At the settlement, drunken fights serve as entertainment during the hot, listless days, the sad realities coming to light as children lose limbs and suffer severe lacerations in the melee.

Compulsive gambling wracks Papunya as well. Every other Thursday, when "sit down money" (pensions) is distributed, the community lies silent and the clinic lies empty. People cluster under trees for days and nights, gambling down to their last cent, while their children cry for food and the elderly sit neglected in the blazing sun. With the loss of their traditional lifestyle and roles, the people of Papunya have few interests with which to fill their lives. The women spend their days caring for the children and gossiping, while the men idle away their time fixing their cars and going for joy rides. Although compulsive gambling and alcoholism stem from complex social conditions, the lack of purpose in their lives and their despondence caused by their inability to resolve western influence with their tribal culture predispose them to these destructive behaviors.

The Aborigines of Central Australia remain one of the last intact tribal communities on earth. As I witnessed, they are quickly being absorbed into the modern, industrialized world. The new generations want the luxuries that I and my friends enjoy. Why throw a spear when a gun will kill more effectively and with less skill? Why walk if you can drive? Televisions and videos provide hours of collective pleasure and fantasy. In spite of the efforts of the older people to preserve their language and customs, the young and ambitious Aborigines are moving to the cities, where opportunity awaits them. The communities, isolated and barren, are being left to the disenfranchised: alcoholics, petrol sniffers, and the weak of spirit. Although the communities are run by Aboriginal governments, internal conflict and opportunism prevent their effective operation and the preservation of the traditional lifestyle. Within the next ten years, more and more communities will be abandoned. I believe that within my lifetime, the Aboriginal tribal lifestyle may only be preserved in history books.

Acknowledgments. I would like to thank the following for making this experience possible: the Alumni Fund of the College of Medicine of the State University of New York Health Science Center at Brooklyn for providing me with a grant to travel to Australia and work among the Aborigines, Dr Pascal James Imperato, Professor and Chairman, Department of Preventive Medicine and Community Health, State University of New York Health Science Center at Brooklyn, who organized and coordinated the elective with the rural health office in Alice Springs, and Dr Yoshi Inoue of the Royal Flying Doctors Service, who accepted me into his program and placed me at Papunya. Jannine Dicks and Fanou Burslem, the two nurses who worked with

me at Papunya, taught me never to underestimate the expertise of nurses. Finally, I want to thank Cheryl Moore and Lois Hahn, of the Department of Preventive Medicine and Community Health, State University of New York Health Science Center at Brooklyn, for faxing letters to Australia and helping me in any way they could.

REFERENCES

1. Powell A: History of the Northern Territory, in *The Northern Territory of Australia: Present Indicative and Future Conditional.* Alice Springs, Northern Territory University Planning Authority, 1981, pi–xx.
2. Myers F: *Pintubi Country, Pintubi Self* Washington, DC, Smithsonian Institution Press, 1981.
3. Hughes R: *The Fatal Shore*, New York, NY, Vintage Books, 1986.
4. Stack E: Human health and disease, in *The Northern Territory of Australia: Present Indicative and Future Conditional (A Selection of Papers on Northern Development).* Alice Springs, Northern Territory University Planning Authority, 1981, p40–45.
5. Lyappa Health Service: *Report of three rural community controlled Aboriginal health services in Central Australia.* Alice Springs, 1978–1982, vol 2, part C.
6. Franks C: Preventing petrol sniffing in Aboriginal communities. *Community Health Studies* 1989; 13:15–19.
7. Thompson N: Australian Aboriginal health and health care. *Soc Sci Med* 1985; 18:940–948.
8. *A National Aboriginal Health Strategy*. National Aboriginal Health Strategy Working Party. Alice Springs, March, 1989.

Leonardo da Vinci's anatomical drawings

Nora Krisch Shire

Leonardo da Vinci's precision and techniques in drawing the human body are unequalled, and many of his anatomical illustrations are accurate for use to this day. One of the truly interesting aspects of his drawings is the fine detail which is not visible in any form of photographs. Even in this day of high-quality reproduction, you must see the drawings with your own eyes to really appreciate Leonardo's talent, says Ron Philo, PhD, in an interview after returning from Windsor Castle where he studied Leonardo's original anatomical drawings. Philo is director of the Human Structure Facility and senior lecturer in the Department of Neurobiology and Anatomy at the University of Texas Medical School at Houston, one of six schools making up the UT Houston-Health Science Center.

Leonardo's drawing, *The Skull Sectioned* (Fig 1), heralds the precision that would later be required in anatomy and medicine. That early work (1489) expresses a 500-year-old technique originated by Leonardo and still popular: Half of the skull is cut away to reveal structures at a different depth. Another Leonardo innovation is seen in *The Spinal Column* (Fig 2), which displays the parts separately yet articulated with appropriate curvatures and the proper number of vertebrae.

Dr Philo went to Windsor at the request of Dr Peter Marzio, director of The Museum of Fine Arts in Houston, TX, in preparation for the exhibition: *Leonardo da Vinci—The Anatomy of Man. Drawings from the Collection of Her Majesty Queen Elizabeth II*. The installation, organized by the Houston museum, has been scheduled at three major medical communities: It closed September 6, 1992, at Houston, is currently at the Philadelphia Museum of Art, September 19–November 29, 1992, and will be seen at the Museum of Fine Arts, Boston, December 11, 1992–February 21, 1993.

As an anatomist, Philo was asked to view the original drawings, judge how accurate they are by today's standards, and then prepare comments for the exhibition's catalog written by Martin Clayton, assistant curator in the print room at Windsor castle.

Because of the national and international importance of this anatomical exhibition, Marzio was seeking an avenue to involve the medical communities where the exhibit would be on view. He decided to have the drawings critiqued by someone eminent in the field of anatomy, Dr Ron Philo. The Leonardo installation features 23 sheets, most double-sided, of 41 drawings of one of the greatest Italian Renaissance artists, who lived from 1452 to 1519.

The Renaissance

One of the fundamental achievements of the Renaissance was the elevation of painting, sculpture, and architecture to the status of the liberal arts. Before the 16th century, most scholars looked down on visual matters as purely mechanical, not a concern of the intellect, and they strongly resisted the corruption of a text by images. The revolution in pictorial representation, spreading outward from Florence in the early 15th century, thus had no immediate impact on anatomical illustration. Whereas the artist was primarily interested in superficial detail, as conveyed by a sensitive life-drawing, the anatomist concerned himself with internal systems. The union of the two, which held so much promise, could only come about either in a collaboration or in an individual skilled both as an artist and as an anatomist. These abilities were to be combined, incomparably, in Leonardo da Vinci.[1(p13)]

With a combination of artistic talent and superb powers of observation, Leonardo was an artist, sculptor, engineer, and architect in Italy during the Renaissance. His other talent, which few at the time knew about, was that of an anatomist. Little was known of his medical research in anatomy.

One of the intriguing facts about Leonardo, whom Clayton describes as one of the pivotal figures in the development of Western art and the father of the High Renaissance, is that he was not a recognized anatomist because he did not work as one professionally, never taught the subject, and most important, he never published any of his discoveries. A history of European anatomical knowledge may legitimately be written without once mentioning his name, Clayton observes. Seldom can a scientist have made so many advances while changing the course of his chosen subject so little. Of all the men who played a part in the Italian Renaissance, none is more remarkable than Leonardo. Master of any discipline to which he set his hand, he exemplified the spirit of inquiry about nature to which the vast corpus of modern scientific knowledge owes its origin. Leonardo was one of the most original and perceptive anatomists of his own or any other time, writes Clayton in the catalog, which by the way is a lavishly illustrated volume of coffee-table quality.[1(p11)]

Three Phases

The exhibit is structured around the three phases of anatomical investigation and illustration accomplished by Leonardo. The first period, from 1487 to 1495, witnessed an expression of Leonardo's belief in the scientific nature of painting. He probably dissected at the Ospedale del Brolo, a unit of the Ospedale Maggiore licensed to allow dissection.

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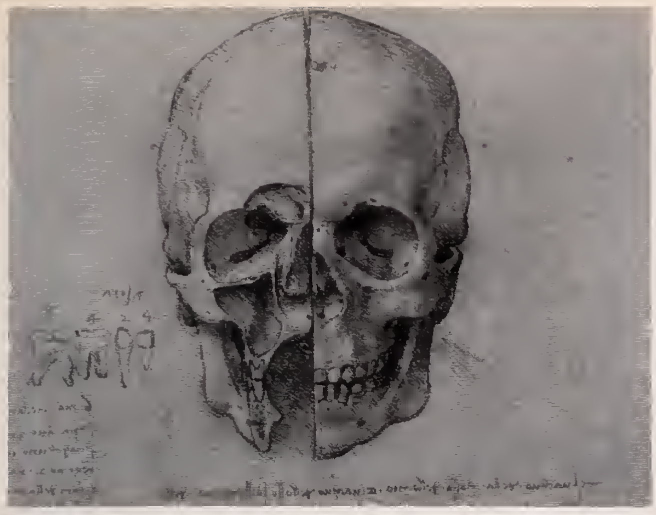

FIGURE 1. *The Skull Sectioned*, circa 1489, is the earliest work in the Leonardo da Vinci exhibition. It illustrates his precision which would later be required in anatomy. (courtesy Windsor Castle, Royal Library Copyright HM Queen Elizabeth II)

His insistence on the ultimate and absolute veracity of every element had its roots in the branch of Florentine objectivity as typified by Leon Battista Alberti, a 14th-century scholar, Clayton explains. However, Leonardo wished to go beyond the limits of material analysis, to an integrated understanding of every facet of the universe. The particle physicists and cosmologists of the late 20th century are still striving to uncover the Grand Unified Theory, the "theory of everything," and that is, in essence, the goal Leonardo set himself 500 years ago.[1(p15)]

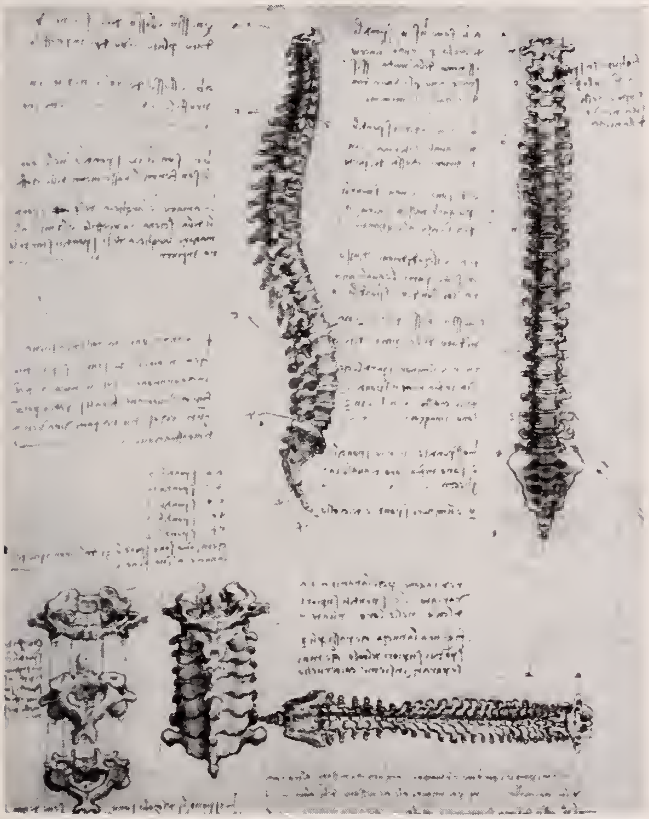

FIGURE 2. *The Spinal Column* displays the parts separately yet articulates the appropriate curvatures and proper number of vertebrae. (courtesy Windsor Castle, Royal Library Copyright HM Queen Elizabeth II)

From 1504 to 1509, Leonardo immersed himself in a second phase of investigation to aid in his important commission, *The Battle of Anghiari*, but the huge mural was never completed. *The Alimentary Tract and Subdiaphragmatic Organs* (Fig 3), circa 1508, illustrates this phase. Although the most prominent features of this sheet are continued views of the contents of the abdomen, the small figure on the bottom right is the most important anatomical representation, for here is presented the first illustration of the appendix, Philo observes.[1(p55)]

One is first tempted to criticize the caecum for being disproportionately large, but caeca of great size are routinely encountered in the laboratory, Philo continues.[1(p57)] Leonardo had already commented upon the empty and contracted nature of the gut, therefore the smallness of the bowels may relate to schematization. He never observed peristalsis. Only the process of gravity, action of body-wall musculature, and propulsion by gas were envisioned by Leonardo for movement of the gut contents, Philo explains.[1(p57)]

A last period of investigation, from 1510 until 1513, was in conjunction with Marcantino della Torre, an anatomist at the University of Pavia. It is fortunate that della Torre was a follower of Galen, for during this period Leonardo's drawings of muscles and bones reached its zenith, stimulated by Galenic methods of observation and deduction.

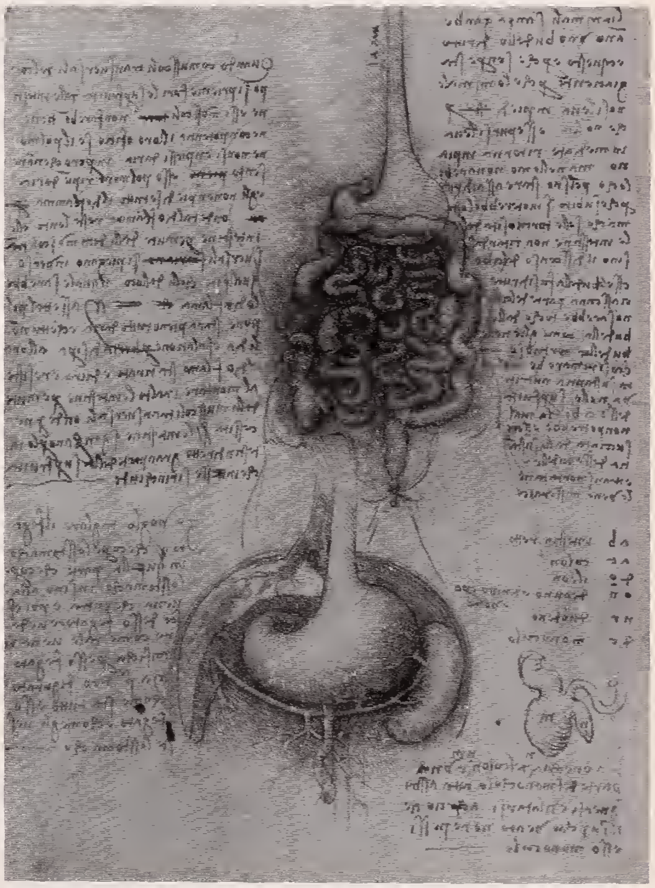

FIGURE 3. *The Alimentary Tract and Subdiaphragmatic Organs*, circa 1508, drawing is important because in the bottom right corner is the first illustration of the appendix. (courtesy Windsor Castle, Royal Library Copyright HM Queen Elizabeth II)

THE HEART

Another topic of enduring fascination to Leonardo during these years was a prolonged study of the heart. As a unit, the heart enabled Leonardo to integrate his observational skills, his understanding of mechanics, and his intuitive grasp of fluid dynamics, Clayton explains.[1(p20)]

In a brilliant investigation of the function of the valves, he constructed a glass model of the aortic valve and sinuses of Valsalva, by taking a cast from an ox's heart. Observing the vortices in the sinuses, he correctly deduced the mechanism of closure of the valves, reaching conclusions that are still being verified in the light of modern research.[1(p20)]

During this period, Leonardo perfected his surface anatomy technique, one of his many strengths. Many muscles are visible on the surface, especially in thin individuals. His pen-and-ink study of *The Muscles of the Arm, Shoulder and Neck* (Fig 4) would be welcomed in a modern-day anatomy text, Philo points out.[1(p99)]

One's attention is riveted to the depiction of musculature of the right upper extremity, chest, and neck, which flows across the two sheets, Philo observes. When viewed in sequence, they become animated, as if the body twists and the limbs move down and back. These drawings are alive, the skin is transparent, the pulse can be felt. Never before equalled and never surpassed, this is at once art, science, anatomy, kinesiology, physics, and architecture—demonstrating all of Leonardo's talents used together.[1(p101)]

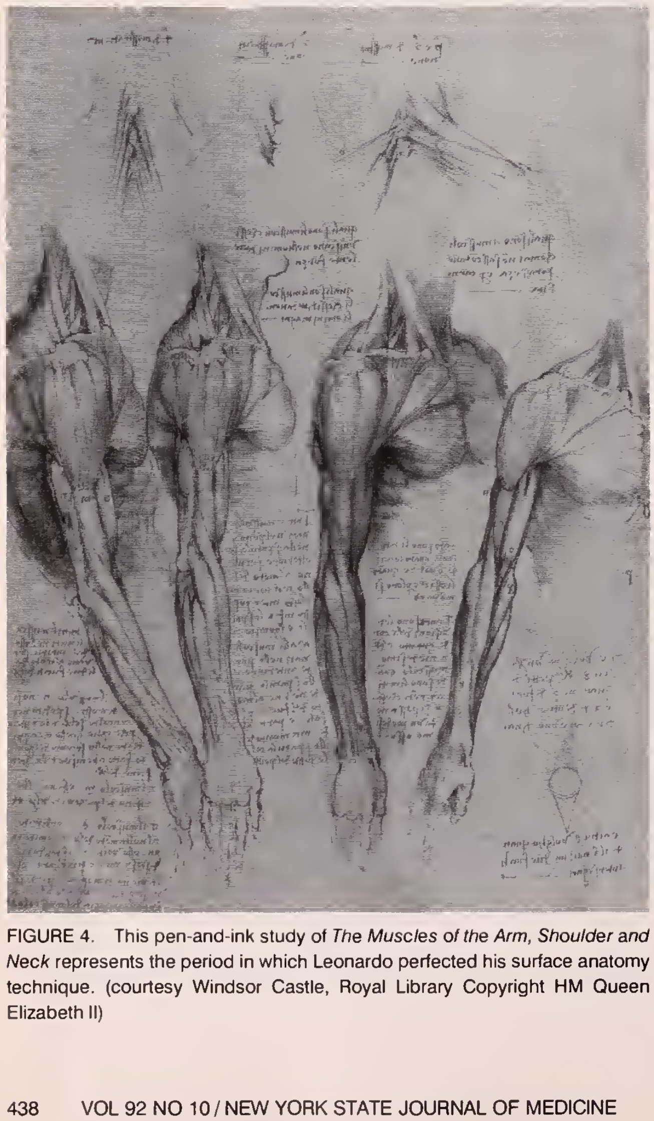

FIGURE 4. This pen-and-ink study of *The Muscles of the Arm, Shoulder and Neck* represents the period in which Leonardo perfected his surface anatomy technique. (courtesy Windsor Castle, Royal Library Copyright HM Queen Elizabeth II)

FINEST WORKS

Anatomically speaking, the finest works of Leonardo in this exhibition begin with the sheet showing *The Spinal Column* and end some eight sheets later, all created in 1510 and 1511. The drawing of the spine corrected a centuries-old confusion as to the number of vertebrae, their relative proportion, and the shape of the spine, Philo explains. Leonardo understood the importance of the curvature for weight-bearing and posture.[1(p87)] The subject matter of these eight sheets is primarily osteological, myological, and topographical. Leonardo's keen architectural mind and bioengineer's mentality could correctly interpret purpose, if not exact physiology, for the head and appendages far easier than for the viscera.[1(p87)] Few errors exist on these 16 pages (made up of the front and back of the eight sheets). The exhibit consists of sheets from his sketch pad. The artist used both sides of the paper because paper was hard to come by 500 years ago. Leonardo recycled every piece year after year.

One sees on the same sheet a drawing of an internal organ, sketches of a dead person, the architectural plans for a palace garden, perhaps a schematic of the eye, and then the blood flow to the heart. Drawings done possibly years apart are often on the same sheet.

COMPARATIVE ANATOMY

Because dissection of humans was not allowed except on criminals who had been executed, anatomists in Leonardo's lifetime depended on animal dissection to study internal structures and functions. Leonardo himself studied animals, and his drawings show what an excellent comparative anatomist he was. Animal dissections were of two basic types: those that were straightforward investigations of individual species, and those conducted to shed light on human anatomy. Of the latter type, Leonardo's favored subjects seem to have been the pig and the monkey.

His fame as a comparative anatomist could rest upon the drawing of *The Uterus of the Cow* (Fig 5), circa 1508, Philo points out. Humans have a single uterus, but cattle have two uterine horns, a condition termed bicornuate. Only one is gravid in this specimen. Uterine vessels are shown as well as ovaries. For further appreciation of this drawing, Philo continues, one must understand a small bit of comparative anatomy.[1(p70)] Cattle possess a cotyledonous form of placenta—these are the "rosettes" or "little sponges" seen through the uterine wall. Leonardo has not only correctly shown these structures, but he has also illustrated how they make contact with maternal tissue. What a tremendous observation and explanation, Philo emphasizes.[1(p70)]

NO EASY TASK

Dissecting and drawing are not as easy as they seem, comments Philo. One either dissects, stops and sketches, and repeats the process many times, or one dissects and draws from memory. Because of the accuracy of his work, Leonardo probably did a combination. Many of the drawings from his medical research were done for the purpose of producing a book with 120 chapters on the anatomy of man to be reproduced by copper engraving. Several outlines for the work survive.

Not all of Leonardo's drawings are anatomically correct, such as *The Fetus* and *Linings of the Uterus*. The images presented are derived from animal dissections and the artist's personal observation of the process of pregnancy. Although the parts are out of position and there are errors,

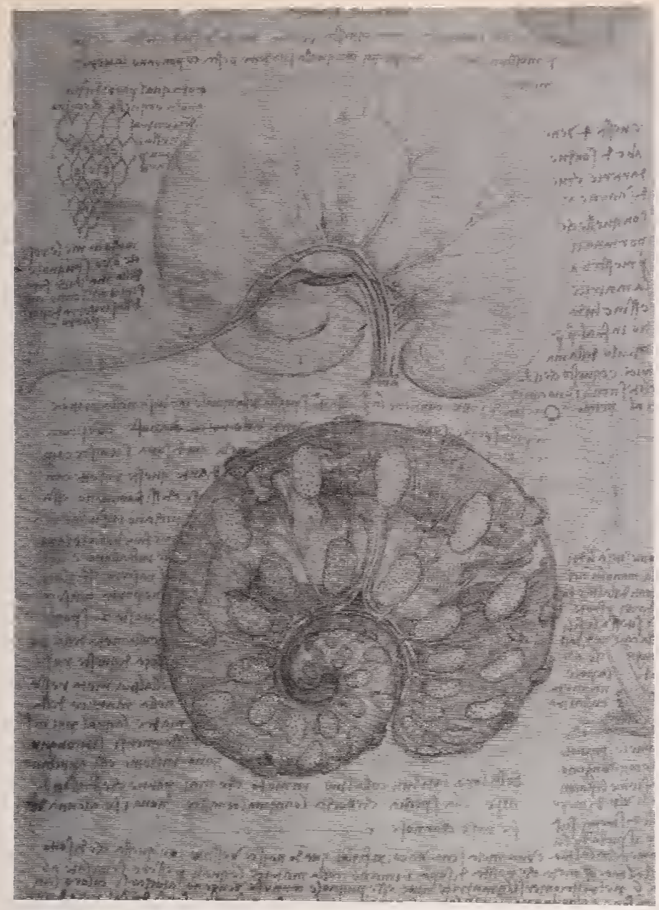

FIGURE 5. *The Uterus of the Cow*, circa 1508, shows the uterine vessels as well as ovaries. It is a excellent illustration of Leonardo's knowledge of comparative anatomy.

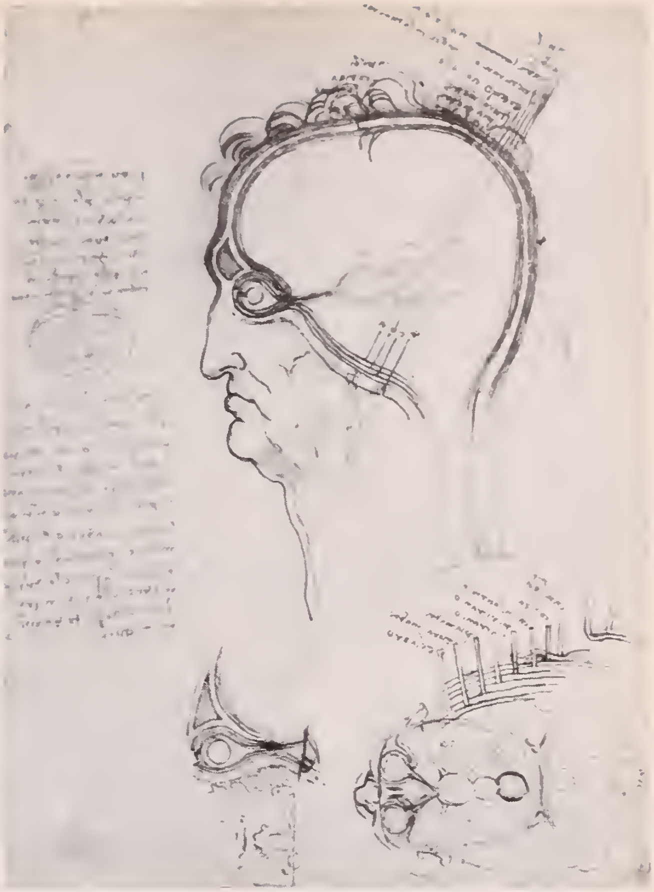

FIGURE 6. *The Cerebral Ventricles and the Layers of the Scalp*, circa 1489–1490, has numerous flaws yet correctly shows the layers of the scalp, the skull and the brain. (courtesy Windsor Castle, Royal Library Copyright HM Queen Elizabeth II)

Leonardo's work is greatly advanced over his predecessors and contemporaries, Philo stresses.1(p127)

The Cerebral Ventricles and *The Layers of the Scalp* (Fig 6), circa 1489–1490, has numerous flaws. The drawing contains an early, possibly the first, depiction of the frontal sinus above the orbit.1(p28) The layers of the scalp, the skull, and the brain are essentially correct and still relevant to today's students.

Another drawing, *The Skull Sectioned*, 1489, is accurate for use to this day. It is unprecedented in the history of anatomical illustration, Philo says.1(p35) Today's medical educators and imaging radiologists will note its prescient relevance of the sectional view to modern anatomical education. It includes a schematic drawing of the four types of teeth. Here Leonardo has set straight an old argument, possibly begun by Aristotle, that asserted women have fewer teeth than men.

On this matter, Leonardo may have quoted Galen through Avicenna, a Persian translator (980–1037) of the medical texts of Galen and Aristotle. Regardless, the artist obviously explored the problem and stated what he observed.

In another drawing, *The Skull: Interior View* (Fig 7), circa 1489, the work is unparalleled in its accuracy and concept. Here is the first accurate depiction of the meningeal arteries and their origination. Roots of the great cranial nerves can be seen converging upon the intersection of the axes Leonardo has drawn. One must remember that Leonardo's work predated the cell theory, the elaboration of circulation, the discovery of galvanic responses and electrophysiology. However, inaccurate his study of function or physiology, his depiction of structure or morphology is essentially correct. One can only wonder how many dissections are represented here, the length of each that was required and the conditions that must have been endured to depict accurately what we see here, Philo observes.1(p36)

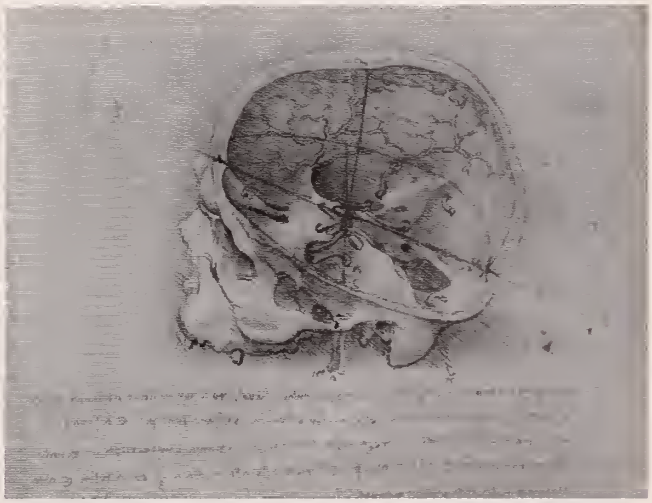

FIGURE 7. *The Accuracy and Concept of The Skull: Interior View* is unparalleled. Here is the first accurate depiction of the meningeal arteries and their origination.

MANY OBSTACLES

Let us not fault Leonardo too much; rather, we should look again at the problems facing him—the difficulty in obtaining human subject matter for dissection, no preservation for specimens, and no microscope, Philo says.[1(p135)]

His studies came to an abrupt end in 1515. Two years earlier, he moved with his pupils to Rome and conducted anatomical investigations in the Ospedale di Santo Spirito. He was slandered by Giovanni degli Specchi, a German mirror maker, who accused him of sacrilegious practices. Pope Leo X barred Leonardo from further dissection. When Leonardo died, the drawings were left to Francesco Melzi, bought by sculptor Pompeo Leoni, then acquired by Thomas Howard, the Earl of Arundel, who became artistic adviser to King Charles I. When the earl was forced to leave England during the Civil War, the collection may have been acquired by Charles I. This is one route through which the drawings may have come into the possession of the British Royal family. By whatever route it reached the Royal Collection, it is clearly recorded as being in the possession of Queen Mary II in 1690, a year after she and her husband King William III ascended the British throne as joint monarchs.[1(p6)]

The Royal Family has granted access to the artist's anatomical drawings since the early part of the 20th century. Since that time, the chronology of the collection's ownership has been elucidated.

In the 1970s, the *Drawings Restoration* team in the Royal Library, headed by Michael Warnes, applied a restoration technique, first to the anatomical drawings and subsequently to all the pages from Leonardo's sketch pad. The pages were encapsulated between two thin layers of acrylic sheeting containing an ultraviolet filter. This in turn was set into mountboard, thus providing a safe environment for the drawings and enabling both sides of each sheet to be seen. Leonardo's drawings are at once art, science, anatomy, kinesiology, physics, and architecture—demonstrating all of his talents used together.[1(p101)]

REFERENCE

1. Clayton M: *Leonardo da Vinci: The Anatomy of Man with Commentaries on Anatomy by Ron Philo*. Boston, Little Brown and Company, 1992.

The story of Lassa fever

Part III: The disease in the community

JOHN D. FRAME, MD

The outbreaks of Lassa fever (LF) in Lassa and Jos in 1969 and 1970 drew the attention of health care workers and the public to the virulence of the disease, the hazard it posed to African hospitals, and the risk of international transport of the virus. Although a serological survey had demonstrated that Lassa virus (LV) infections were endemic in Nigeria and in Guinea and that the disease might be milder than what was initially observed, little was understood of the public health significance of LF. Subsequent dramatic hospital outbreaks of LF eventually developed into markers of continued LV infections in the community. For a while, however, interest in LF remained dormant.

LASSA FEVER IMMUNE PLASMA COLLECTION

Not every one had forgotten the Nigerian outbreaks. The Lake Avenue Congregational Church of Pasadena, California, was aware that Dr Jeanette Troup might have survived LF if immune plasma had reached her in time. The congregation determined that mission hospitals in Nigeria should be prepared in case there was another outbreak of LF there. It donated funds for the purchase and transport of a freezer and plasmapheresis equipment and supplies to the Evangel Hospital of the Sudan Interior Mission (SIM) in Jos. Miss Lily Pinneo, the first to recover from this disease in 1969, donated her own plasma, and assisted in the collection of plasma units from survivors of the 1970 outbreak in the hospital. Immune plasma from Miss Pinneo and Dr Jordi Casals of the Yale Arbovirus Research Unit (YARU), the other survivor of the 1969 outbreak, was also stored at the US Centers for Disease Control (CDC) in Atlanta, Georgia.

In March 1970, I received a telephone call from Mrs Jordi Casals who was a volunteer worker in a Lutheran Church in New York. She had just heard that an outbreak of LF was suspected in the Lutheran mission hospital in Zorzor, Liberia. A seriously ill nurse, Miss Esther Bacon, had been flown by mission plane to the better equipped Phebe Hospital. The stir engendered by this phone call was reminiscent of that after the initial announcement of the newly discovered disease in 1970 and the coincidental second outbreak of LF in Nigeria. As before, the information available to us at Columbia University in New York was scanty. By telephone, we shared what we knew of LF with the office of the Firestone Tire and Rubber Company in Akron, Ohio, owners of "the world's largest rubber plantation" in Liberia. Firestone, too, had received word of the outbreak, as had other corporations with interests in West Africa. At the time, the early descriptions of LF I had prepared for mission groups were the only available concise summaries of the disease and were sent to those who wished to know more about LF and its possible impact on their operations. These telephone conversations were fruitful later in facilitating further research on the disease.

LF IN LIBERIA

Dr Paul Mertens came to the Curran Lutheran Hospital (CLH) in Zorzor, Liberia, in 1963, a year after graduating from the University of Minnesota Medical School. Zorzor is about 184 miles, by road, north of Monrovia, the capital.

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FIGURE 1. Esther Bacon, missionary nurse and midwife (courtesy of The Christopher Publishing House).

Dr Mertens acquired most of his knowledge of tropical medicine from Dr James Stull, Medical Director of CLH, and from veteran nurse, Miss Esther Bacon (Fig 1).

With her registered nurse degree from the Lutheran Hospital School of Nursing in Sioux City, Iowa, 25-year-old Esther Bacon first went to Liberia in 1941; for almost a year, she waited for the first Liberian to accept her service as midwife. Early, she found it necessary to visit patients in their homes, mounted on her horse Lizard, but eventually women came to her. On her first furlough, she studied midwifery at the Frontier Nursing Service in Hyden, Kentucky, then returned to begin instructing young women as midwives. For many years, most midwives in Liberia were either her pupils or her pupils' pupils. On subsequent furloughs, she continued her education, eventually earning a Master of Public Health degree from the Johns Hopkins University School of Public Health.[1]

In mid-1970, Dr Mertens received, through the office of the Lutheran Church in America, the description of LF I had distributed to mission agencies. That November, a student in midwifery at CLH became seriously ill. Her symptoms—fever, sore throat, dysphagia, hearing loss, and coma—suggested the diagnosis of LF to him, and he sent patient serum to the YARU for testing but never received an answer. The student recovered, and later joined the staff of the hospital associated with the SIM radio station ELWA 10 miles outside Monrovia. In 1974, her blood was among those tested by Alan Bloch,[2] a medical student from Yale taking a three-month overseas elective at the ELWA Hospital and was found positive for LF antibodies.

In 1971, four students in agriculture attending the Zorzor Training Institute near CLH were admitted to the hospital at one time; the laboratory tests revealed no cause for their severe fevers, and they did not respond to empiric treatment for malaria and typhoid fever. Again, Dr Mertens considered LF as a possible diagnosis. Later, when he attempted to obtain convalescent serum from the patients, they had left the Institute and could not be traced.

On March 2, 1972, a young woman four months pregnant entered CLH with a history of fever, vomiting, abdominal pain, and vaginal bleeding for several days. The next day, after she had aborted twin fetuses, Miss Bacon performed dilatation and curettage. The patient continued to have fever and complained of severe headache; findings included generalized lymphadenopathy, puffiness of the face and eyes, and oliguria. After two weeks, her health improved; she was discharged from the hospital on March 19.[3]

Between March 20 and 26, 1972, three other patients and seven hospital staff members became ill, and by March 28 Dr Mertens realized he was most likely dealing with an outbreak of LF. Thereafter, all cases were removed from the open ward. All sick staff members had had responsibilities on the obstetrical ward. One of the staff was the first to die. The patients had been in the ward with the index case. One had been treated for pyelonephritis as a complication of pregnancy; she had been in the bed next to the index case and shared food with her. Discharged as improved, she returned to CLH five days later because of fever and died there. Another young woman left the hospital after delivery, became ill at home and was admitted with fever to a small mission hospital in Foya, about 100 miles away. She recovered; but her infant died of fever; specimens could not be obtained to prove the cause of the baby's death.

The third patient, a young pregnant woman who had had a previous Caesarean section, was treated for pneumonia; she responded rapidly to antibiotic treatment and Caesarean section on March 14. Fever recurred suddenly six days after surgery. In spite of the administration of a unit of blood and general supportive care her condition deteriorated, and she died after vaginal hemorrhage on April 4. Her baby, too, died of fever three days later.

DEATH OF ESTHER BACON, MISSIONARY NURSE

Esther Bacon not only performed the dilatation and curettage on the index case, but also had frequent nursing contact with her until discharge from CLH March 19. On March 20, she developed chills and fever but continued to work. In four days, increasing fever, myalgia and headache, pleuritic, and epigastric pain, and sore throat developed. She could not swallow and was given intravenous fluids. Between the eighth and tenth days of her illness, she developed a maculopapular rash of the face, chest, and back, swelling of the face and bibasilar pulmonary rales. On March 30, she was transferred to Phebe Hospital. There she began to bleed from intravenous injection sites and from a lumbar puncture wound. She was infused with two units of plasma from the recently recovered patients in CLH, but her condition steadily grew worse and she died April 4.[3] She was buried about 50 feet from the obstetrical ward where she had served Liberian women (Fig 2).

As in the case of Dr Jeanette Troup in the 1970 outbreak in Nigeria, it was not yet known that in convalescents, neutralizing antibodies against LF do not reach adequately protective titers until three to six months after the onset of the infection.[4]

Three of the 11 LF patients developed hearing loss late in their illness; deafness persisted in two patients after

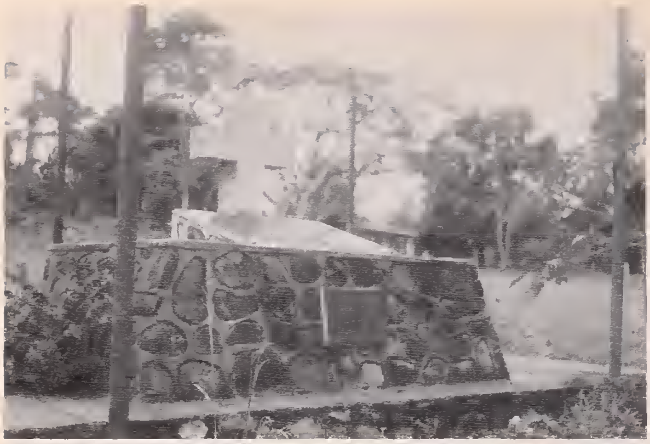

FIGURE 2. Grave of Esther Bacon, Zorzor, Liberia. The tablet reads in part: Esther Eleanor Bacon RN, BSH, MPH, mid-wife, nurse, true servant of Mercy. (John D. Frame).

recovery. One was found to have complete nerve deafness when tested audiometrically 10 years later. Unable to complete her training as midwife, she found no employment until she was accepted in the diet kitchen of the Firestone plantation hospital.

EPIDEMIOLOGICAL INVESTIGATIONS

Dr Thomas Monath of the US CDC on assignment at the Virus Research Laboratory in Ibadan, Nigeria, was scheduled to return to Atlanta. He was asked by the CDC to stop in Liberia to lead epidemiological investigations at Zorzor. At CLH, he was joined by Miss Lily Pinneo and Dr Jordi Casals, both presumably immune to the infection. They were considered to be relatively safe at a time when it was still not known what was the reservoir for LV, how it was transmitted, and what risks there might be in conducting hospital and field studies (Fig 3).

They and the staff of CLH reviewed hospital records and identified six patients with histories compatible with LF in the months preceding the illness of the index case. Only one could be found in her home village; she had been treated in the obstetrical ward from February 23 to March 4. She was positive for LV antibodies (LVA) by the complement fixation (CF) test. Five of the epidemic cases, including the first, had had no contact with her.

FIGURE 3. L to R, Lily (Penny) Pinneo, RN, Dr Paul Mertens, Dr Thomas Monath, and Dr Randy Mosher, investigating the epidemiology of LF in Liberia (courtesy of Lily Pinneo).

Fifty-nine patients in the hospital at the time of the outbreak who could be located in their homes were tested serologically; six had LVA. None had had fever lasting over a week during the period after hospital treatment. Of 17 workers in the ward, two had LVA, one with rising titers; neither had had recognized illness. None of the 40 workers in other parts of the hospital were positive on testing, and none of 22 family members of cases. In Zigida, the village from which the index case had come, four of 133 sera tested were positive as well.

It appeared, then, that cases of LV infection less severe than those identified in the outbreak had occurred at the time; in fact, some infections seemed to have been inapparent. The findings were compatible with what had been noted in the serological survey of missionaries in Guinea among whom there were no deaths and no apparent person-to-person transmission.[5,6]

The workers were unsuccessful in attempts to find what animal reservoir might be the source of infection. Incidentally, reports of their efforts to trap rodents were translated by the village populations as attempts to discover the spirits responsible for the outbreak.

SIERRA LEONE

In the summer of 1972, a Peace Corps physician reported to the CDC his suspicion that hospitals in Eastern Sierra Leone were dealing with an outbreak of LF. A team from the CDC, including Dr Monath, investigated this outbreak in September and October, 1972. The results of their study gave important new insights into the place of LF in the African community.[7]

The epidemic centered on the town of Panguma and in a group of neighboring towns collectively known as Tongo in the Kenema District. The investigation began at the Panguma Catholic Hospital (PCH) and the National Diamond Mining Company Hospital in Tongo. The team first developed clinical definitions of probable and possible LF. From hospital patient charts, the team identified 63 suspect cases treated in the two years after October 1, 1972, with a progressive increase in the number of cases observed monthly beginning in February, 1971. Twenty four had died. Only six had had direct contact with another LF case in the preceding month, and five of the six were hospital staff members. In contrast with previous LF outbreaks, only a minority of cases had been acquired in the hospital, and most had been infected in the community.

Among the 54 workers of PCH, 12 (22%) had CF antibodies to LV, but only four had a history of illness severe enough to require hospital care. This was in contrast with the outbreak in Liberia where only 2% of the staff were positive for LVA by CF tests.

The investigation was extended into the community. Compounds or households in which hospital cases had lived were identified; each was matched with a control compound or household chosen at random from the neighboring area. Serological testing demonstrated not only that LV infection was widespread in the community, but also that it was more common in the homes of cases than in homes with no illness severe enough to bring the patient to the hospital. It was also found that increased crowding within homes was associated with increased numbers of cases. The results were compatible with person-to-person spread of the infection, although transmission from an animal host was possible, at least in some instances.

Six percent of the residents of the two towns were sero-positive for LV infection whereas only 0.2% were recognized as having severe clinical illness. This, too, may be contrasted with the experience in Liberia, where only 3% of fellow villagers of the index case were found to have LVA. The results suggested that in contrast with the case-fatality rate of 38% in the hospital, the mortality rate of LV infections in the community was probably less than 5%.[6]

Clinical studies were conducted in 20 cases selected according to the case definition and seen by the team in September and October; one died. Twelve were proven to be LF by CF test and virus isolation. One case followed a course not seen in earlier patients; she improved and was discharged, then relapsed and was treated again at PCH.

Two patients who appeared seriously ill were given 250 mL of Pinneo plasma each; both improved subjectively and objectively in 12 hours. In one, LV was isolated from pleural fluid and blood before, but not after, infusion. Viremia persisted in only one patient after 15 days; the team concluded that quarantine for three weeks after infection was justified on the basis of the epidemiological evidence.[8]

THE ANIMAL HOST IS IDENTIFIED

Extensive attempts to isolate LV from small animals had been unsuccessful in both Nigeria and Liberia. Dr Monath and his colleagues knew that other viruses within the group of arenaviruses, among which LV was classified, normally infected rodents and a bat in one instance. In Sierra Leone, they trapped 641 small vertebrates in and near the homes of LF cases. LV was isolated from the organs of 14 of 82 multimammate rats, *Mastomys natalansis*, and from none of the other animals tested.[9]

Although the means of virus transmission from rodent to man was not yet certain, analogies seemed appropriate with what was known of the other arenavirus infections. In them, transmission depends on close association between rodent and man, and contamination of the environment with infected rodent urine and other excreta. At least some of the agents in the group induce a chronic virus carrier state in their natural hosts with persistent excretion of the virus in their urine.

M natalensis is widely distributed in sub-Saharan Africa, and is adapted to life both in fields and in houses. Its main rodent competitor is the more aggressive *Rattus rattus*; where the latter is present in homes, *M natalensis* is generally absent. Dr Monath and his colleagues pointed out that though rodent-proofing of food stores might be effective in limiting LV infections, indiscriminate anti-rodent control measures such as poisoning might favor *M natalensis* by eliminating *R rattus,* its natural antagonist.

SEROLOGICAL SURVEYS FOR THE DISEASE

When we at Columbia University first learned of the outbreak of LF at CLH, we called the Firestone Tire and Rubber Co and sent their office the information regarding LF I had earlier prepared. About two weeks later, I suggested to Dr Michael Katz, who was now chairman of the Division of Tropical Medicine, that we ask Firestone for research funds. Dr Katz was quite anxious that I not ask for too great an amount, perhaps just enough to pay for materials, postage, and the like.

I had already discussed a plan for a serosurvey for LV and arboviral infections with Dr Casals of YARU. I thought of two populations whose testing might be useful in determining the distribution of LV infections in Africa: missionaries whom I examined on their return from Africa, and patients in mission hospitals in Africa. I prepared a budget for the survey; Dr Casals told me that YARU would not charge for the testing. I wrote to Firestone outlining my plan with a budget of $4,000 and soon received the funds. Later, Dr Katz was able to obtain an additional $1,000 from the World Health Organization. With this sum, between 1972 and 1978, we found evidence of LV infection in six countries where it had not yet been recognized and studied two outbreaks of LF in Nigeria.

In 1972, only the CF test was generally available for the serodiagnosis of LF. The plaque reduction neutralization test (PRNT) measuring neutralizing antibodies required the use of active LV and could only be performed in special laboratories with elaborate precautions against infection. It became apparent that CF responses to LV began in the second week of illness and were not detectable in most patients until the end of the third week. Titers tended to drop rather rapidly, some to negative responses by the end of a year, and most within four years. Surveys using CF would discover most, but not all, relatively recent infections. We believed, however, that even with these limitations the survey would provide useful information.

From the serological testing of missionaries on furlough from Africa, Dr Casals found CF antibodies for LV from workers in Mali, Upper Volta (now Burkino Faso), and Ivory Coast. One missionary was living in Zaire when she became ill; she had recently returned from the Central African Republic where she packed household goods the family had left in their previous home, so it was not certain where she had become infected.[10] A missionary child from south-central Zaire was positive as well.

Twelve mission hospitals in East, Central, and West Africa joined in this cooperative program to test febrile patients for LV and selected arboviruses. They were sent kits for drawing blood into vacuum tubes, and equipment for transferring serum to vials to be mailed to New York. Unfortunately, not all the specimens reached us in good condition; many were anticomplementary and unfit for CF testing. A pair of specimens from Cameroon showed rising titers to LVA, proving the presence of infection. A number of arbovirus infections were diagnosed as well, and reports of the findings were fed back to the hospitals.

The SIM medical department in Nigeria in particular asked its hospitals to cooperate in this survey. The Evangel Hospital in Jos, the site of the second recognized outbreak of LF in 1970, sent patient specimens, and a number were found to have had LF on the basis of rising titers of CF antibody; the patients, both missionary and African, had come from various parts of Nigeria.[10,11]

I was particularly impressed by the contribution of a small hospital in Kagoro, Nigeria, which was under the SIM-associated Evangelical Church of West Africa. A Nigerian physician's assistant, Mr C. Damian, was in charge. Out of this small institution came 26 serum pairs from which we were able to diagnose two cases of LF from rising antibody titers, and instances of other arboviral infections as well. Kagoro was second only to Evangel Hospital in the number of definite diagnoses of arboviral and LV infections.

The program of testing for LV and selected arboviruses had benefits beyond what we had anticipated. For one, feed-back of results to the hospitals led to an increased ability of hospital staffs to diagnose arbovirus infections and distinguish them clinically from LF. Then, the routine testing of patients with fever resulted in the unexpected diagnosis of LF in some instances. Evangel hospital sent serum from a Swiss missionary laboratory technologist transferred from the SIM hospital in Bembereke, Benin, because of fulminant hepatitis. When I realized it was an early specimen from a severely ill patient, I recognized that although it might be too early to find CF antibodies, virus isolation might be successful. I forwarded the specimen to the CDC, packaged and doubly wrapped against spillage. LV was indeed isolated. We learned that autopsy had led to the diagnosis of fulminant viral hepatitis. However, neither the CDC nor I was able to obtain the pathological slides from the University Hospital of Ibadan where the necropsy was done. (unpublished data, J. D. Frame, 1976).

During this period, we identified two outbreaks of LF at the Vom Christian Hospital located a few miles from Evangel and where some of the patients in the 1970 outbreak had been treated.[11] In December 1974, a British physician who was pregnant was treated for a febrile illness. Three Nigerian student nurses who cared for her became ill and one died; the initial patient delivered a macerated fetus six weeks after the onset of her illness. After this experience, the mission hospitals in Nigeria agreed that in case of another suspected outbreak of LF workers who had recovered from the disease, whatever their assignment, would be released to work in the affected hospital.

The second outbreak at Vom in 1976 was community based. A pregnant woman with fever and vaginal bleeding died two weeks after admission to the hospital; LV was isolated from her serum. A Nigerian nursing student who shared in her care became ill; she was administered two units of LF convalescent plasma on the 18th day of her illness, seemed to improve initially, but died in kidney failure nine days later. With her illness and a sudden influx of fever patients, medical, nursing, and laboratory workers who were presumably immune were assembled from other mission hospitals; there were no more cases among staff members.

LV was also isolated from a 10-month-old infant and another pregnant woman, both of whom died, and from a woman who survived; several fever patients admitted at the time had LVA by CF, but in the absence of paired sera, the diagnosis of active LF could not be confirmed.

It was obvious by this time that LF was a continuing health problem in a wide area of West Africa. In Europe and North America, public interest in the disease was maintained by occasional reports of the illness in expatriates. One was an Irish nun from Sierra Leone in 1972, who was convalescent by the time she reached home.[13] One of two German missionary physicians in Onitsha, Nigeria, died of LF in 1974, and the other was evacuated to Germany by air; he survived.[14] A Scottish missionary physician became ill in 1974; he may not have known that convalescent plasma was available in Jos, a few miles from his hospital in Zonkwa, and flew home only to die there. Radio warnings to those who had traveled with him were broadcast in Western Europe;[15] warnings were aired again when an engineer working in Nigeria was admitted to a British hospital in 1976.[16] In early 1976, a Peace Corps worker with an undiagnosed illness returned to the United States with a four-hour stopover in Heathrow Airport in England and a week-end in a Washington hotel. LV was recovered from her urine, although not from blood after her admission to the hospital.[17] A mysterious case in Toronto was reported in wire service dispatches across the country.[18] The woman had not been in Africa, but had met many other tourists while on a European vacation.[19] The mystery was finally explained by the false positive result of a newly developed diagnostic test.

It was no longer appropriate to await chance outbreaks of LF to study the disease. It was evident that systematic research was needed to elucidate what was recognized as an important public health problem, in the international arena as well as in Africa.

REFERENCES

1. Dibble JB: *Outlaw for God: The Esther Bacon Story.* Hanover, MA, The Christopher Publishing House, 1992.
2. Bloch A: A serological survey of Lassa fever in Liberia. *Bull WHO* 1978; 56:811–813.
3. Mertens PE, Patton R, Baum JJ, et al: Clinical presentation of Lassa fever cases during the hospital epidemic at Zorzor, Liberia, March–April, 1972. *Am J Trop Med Hyg* 1973; 22:780–784.
4. Jahrling PB, Frame JD, Smith SB, et al: Endemic Lassa fever in Liberia. IV. Selection of optimally effective plasma for treatment by passive immunization. *Trans Roy Soc Trop Med Hyg* 1985; 79:380–384.
5. Monath TP, Mertens PE, Patton R, et al: A hospital epidemic of Lassa fever in Zorzor, Liberia, March–April, 1972. *Am J Trop Med Hyg* 1973; 22:773–779.
6. Henderson BE, Hazry GW Jr, Kissling RE, et al: Lassa fever: Virological and serological studies. *Trans Roy Soc Trop Med Hyg* 1972; 66:409–416.
7. Fraser DW, Campbell CC, Monath TP, et al: Lassa fever in the Eastern Province of Sierra Leone, 1970–1972. I. Epidemiologic studies. *Am J Trop Med Hyg* 1974; 23:1131–1138.
8. Monath TP, Maher M, Casals J, et al: Lassa fever in the Eastern Province of Sierra Leone, 1970–1972. II. Clinical observations and virologic studies on selected hospital cases. *Am J Trop Med Hyg* 1974; 23:1140–1149.
9. Monath TP, Kemp GE, Setzer HW, et al: Lassa virus isolation from Mastomys natalensis rodents during an epidemic in Sierra Leone. *Science* 1974; 185:263–265.
10. Frame JD: Surveillance of Lassa fever in missionaries stationed in West Africa. *Bull WHO* 1975; 52:593–598.
11. Frame JD, Casals J: Serological testing for selected African viruses in the clinical ambience: Results of complement-fixation testing. Annual Meeting, American Society of Tropical Medicine and Hygiene, Nov 3–5, 1976. Philadelphia, PA.
12. Frame JD, Thomas IL, Evans S, et al: Outbreaks of Lassa fever in the Vom Christian Hospital, 1974–75 and 1976. Annual Meeting, American Society of Tropical Medicine and Hygiene, Nov 3–5, 1976. Philadelphia, PA.
13. Woodruff AW, Monath TP, Mahmoud AF, et al: Lassa fever in Britain: An imported case. *Br Med J* 1973; 3:616–617.
14. Bowen GS, Tomori O, Wulff H, et al: Lassa fever in Onitsha, East Central State, Nigeria in 1974. *Bull WHO* 1975; 52:599–604.
15. How a deadly virus slipped into Britain: *The Sunday Times (London)* Jan 16, 1975; 7911:1.
16. Gillie O: Lassa suspect refuses to go to hospital. *The Sunday Times (London)* Aug 1, 1976; 7990 p1.
17. Zweighaft RM, Fraser DW, Hattwick MAW, et al: Lassa fever: Response to an imported case. *New Eng J Med* 1977; 297:803–807.
18. Deadly african fever suspected in Canada. *Casper (Wyoming) Star Tribune* 1976; 85 (227) p2.
19. Best EWR: The Lassa fever episode, Metro Toronto, August, 1976. *Canad J Publ Hlth* 1976; 67:361–366.

Acute tricuspid valve incompetence secondary to blunt trauma

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Chronic tricuspid valve insufficiency usually occurs as a result of pulmonary hypertension and/or annular dilatation secondary to diseases of the mitral valve or cardiomyopathy. Isolated valvular regurgitation caused by blunt trauma is a rare event.[1] Fewer than 60 cases have been described in which traumatic ruptures of papillary muscles, chordae tendineae, or valve leaflets resulted in chronic tricuspid valve insufficiency.[2] In the majority of cases, the diagnosis is established during cardiac catheterization, at surgery, or at autopsy, many years after injury. Transthoracic echocardiography has proved useful in the evaluation of valvular pathology. However, transthoracic echocardiography in victims of multiple trauma may not be appropriate due to chest configuration, lung tissue, or other external factors that would impede such a study. More recently, transesophageal echocardiography has improved cardiac imaging.[3] In this report, we present the case of a young man in whom the diagnosis of isolated tricuspid incompetence after blunt chest trauma was established early and accurately using transesophageal echocardiography.

Case Report

A 33-year-old man was admitted to the hospital after being involved in an automobile accident in which he sustained a closed head injury and lacerations of the spleen and liver. Immediate surgical exploration resulted in repair of the splenic and hepatic injuries. Five days after the accident, prominent jugular venous pulsations were noted on physical examination. An electrocardiogram revealed right bundle branch block. Before the accident, there was no history of heart disease.

Physical examination revealed a temperature of 37.9°C; pulse rate, 120 beats/min; respiration, 18/min; and blood pressure, 120/60 mm Hg. The chest was clear to auscultation. There was a grade III/VI pansystolic heart murmur over the right parasternal border. Peripheral edema, hepatomegaly, and ascites were not present. Laboratory studies revealed a hemoglobin level of 11.7 g/dL and a white blood cell count of 13.6/mL. The chemistry profile was normal. A roentgenogram of the chest revealed moderate cardiomegaly with clear lung fields. Two-dimensional transthoracic echocardiography showed enlargement of the right atrium and right ventricle with poor visualization of the tricuspid valve apparatus. However, transesophageal echocardiography equipped with Doppler and color flow demonstrated the presence of significant tricuspid insufficiency with a flail anterior leaflet of the tricuspid valve (Fig 1).

The patient had recovered satisfactorily from the head and abdominal injuries, and three weeks after the injury he was discharged home. At the five-week follow-up, right bundle branch block was still present on an electrocardiogram. Because of dyspnea and fatigue, he could not attain more than 5 METS on a treadmill test with a peak heart rate of 153/min and blood pressure of 138/60 mm Hg. Cardiac catheterization revealed a right atrial pressure of 6 mm Hg with a V-wave of 13 mm Hg and right ventricular pressure of 22/4 mm Hg. The pulmonary arterial pressure was 23/12 mm Hg, and the pulmonary capillary wedge pressure was 8 mm Hg. The cardiac index was 2.1 L/min/m^2. Right ventriculography demonstrated severe tricuspid insufficiency. Since the patient was symptomatic in the presence of severe tricuspid regurgitation, tricuspid valve repair was recommended. The patient declined surgery, and one year later, he continues to work with minimal functional limitation.

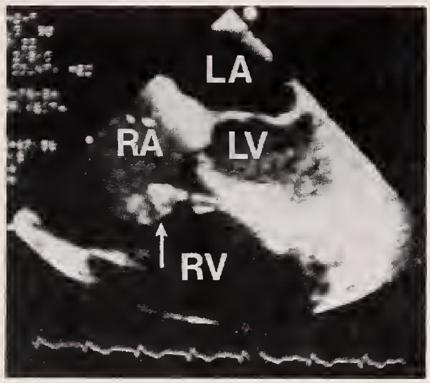

FIGURE 1. Transesophageal echocardiogram showing incompetence of the tricuspid valve. The ruptured papillary muscle is shown by the arrow. (LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle).

Discussion

Symptoms and physical manifestations of tricuspid regurgitation are usually sufficient to establish the diagnosis of tricuspid incompetence. The jugular venous pulse may show a dominant V-wave. The murmur is pansystolic, often high-pitched, and increases on inspiration. The enlarged liver may show systolic pulsation. In advanced cases, peripheral edema and ascites may occur.

Valve repair or replacement for isolated insufficiency of the tricuspid valve is rarely performed in the general practice of cardiac surgery. When required, it is usually due to concomitant left-sided disease or severe rheumatic tricuspid valve disease and consequent severe functional tricuspid regurgitation. Rarely, tricuspid incompetence occurs as a result of severe nonpenetrating chest injury. It is usually due to rupture of papillary muscles, chordae disruption or detachment, rupture of the valve leaflets, or complete valve destruction.[4]

Making a definite diagnosis of tricuspid regurgitation immediately after blunt trauma may be difficult because the usual symptoms and manifestations may not be apparent. In the setting of trauma, patients are usually in critical care units and in need of mechanical ventilatory support. Contrast ventriculography is associated with increased risk, and the presence of a catheter through the tricuspid valve may simulate regurgitation. Two-dimensional echocardiography has been particularly useful in the recognition of abnormalities of intracardiac structures.[5] Applied transthoracically, the technique has a sensitivity of 77%–92% and a specificity of 84%–94%, but an adequate study can be obtained in only 75% of patients.[6] Transesophageal echocardiography, with its superior resolution, may be a more useful diagnostic tool in traumatized individuals. By placing the transducer within the esophagus, the amount of tissue intervening between the transducer and the heart is greatly reduced, particularly in the supine position. Transesophageal images of the tricuspid valve apparatus may be superior because of

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the proximity of the structures to the transducer, and this technique may delineate the cause of tricuspid insufficiency better than the transthoracic approach. Although transesophageal echocardiography is a semi-invasive procedure, the risk is small.

In patients who survive the initial trauma, tricuspid incompetence may be tolerated for many months or years. This probably reflects the much slower development of the deleterious effects of ventricular volume overload on the right of the heart compared with the left. Surgery is recommended promptly in the presence of chronic right-sided cardiac failure not relieved by medical management.[7] However, some cases of traumatic tricuspid insufficiency caused by ruptured papillary muscle may have a relatively benign course, with long-term survival and no surgery, as in this case.[8]

Transesophageal echocardiography may add a new dimension in our ability to identify patients with traumatic tricuspid incompetence and could provide important information to the surgeons enabling them to repair the tricuspid valve injury at the time of emergency surgery, or to delay until life-threatening problems have been solved.

References

1. Shabeta R, Adolph R, Spencer FC: Successful replacement of the tricuspid valve 10 years after traumatic incompetence. *Am J Cardio* 1966; 18:916–920.
2. Gayet C, Pierre B, Delahoye JP, et al: Traumatic tricuspid insufficiency: An underdiagnosed disease. *Chest* 1987; 92:429–432.
3. Hilton T, Mezei L, Pearson AC: Delayed rupture of tricuspid papillary muscle following blunt chest trauma. *Am Heart J* 1990: 119:1410–1412.
4. Katz NM, Pallas RS: Traumatic rupture of the tricuspid valve: Repair by chordal replacements and annuloplasty. *J Thorac Cardiovasc Surg* 1986: 91:310–314.
5. Child JS: Improved guides to tricuspid valve repair: Two-dimensional echocardiographic analysis of tricuspid annulus function and color flow imaging of severity of tricuspid regurgitation. *J Am Coll Cardiol* 1989; 14:1275–1277.
6. Ezekowitz MD, Wilson DA, Smith EO, et al: Comparison of indium-111 platelet scintigraphy and two-dimensional echocardiography in the diagnosis of left ventricular thrombi. *N Engl J Med* 1982; 306: 1506–1512.
7. Noera G, Sanguinetti M, Pensa P, et al: Tricuspid valve incompetence caused by nonpenetrating thoracic trauma. *Ann Thorac Surg* 1991; 51: 320–322.
8. Osborn JR, Jones RC, Jahnke EJ: Traumatic tricuspid insufficiency. *Am J Cardiol* 1970; 26:200–204.

Cervical esophageal perforation—A unique presentation

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Esophageal perforation constitutes a true emergency that demands immediate diagnosis. Spontaneous esophageal rupture, or Boerhaave's syndrome, was first reported in 1724. Boerhaave described the case of Baron van Wassenauer who took an emetic agent after overeating and ruptured his esophagus on vomiting.[1] The use of the word "spontaneous" is actually misleading since some initiating factor is usually identified; examples include emesis, coughing, defecation, lifting, sneezing, yelling, and childbirth. The perforation is usually in the distal one third of the esophagus.[2–5] Typical symptoms and signs of esophageal rupture are lower thoracic pain and subcutaneous emphysema.

We describe a patient who presented after single episode of vomiting, accompanied by shortness of breath, pain on swallowing, and inspiratory stridor. We believe she had a perforation of the cervical esophagus.

Case Report

A 22-year-old woman was brought to the emergency department by ambulance for acute shortness of breath and pain on swallowing. Her general health was excellent; she had a medical history significant only for a left salivary gland biopsy seven years previously which demonstrated malignancy. This was never followed up.

Late on the evening of presentation, she felt something stick in her throat while she was swallowing a piece of cookie. She vomited once and afterwards noted mucus coming from her mouth that was initially clear but which then became pink. This was quickly followed by shortness of breath.

On arrival, at the emergency department, her only complaints were shortness of breath and pain on swallowing. She denied chest pain, nausea, back pain, or abdominal pain. There was no history of drug or alcohol abuse.

Physical examination revealed mild-to-moderate respiratory distress with a respiratory rate of 30 breaths per minute. Her other vital signs were as follows: blood pressure, 138/62 mm Hg; pulse, 66 beats/min; and oral temperature, 37.5°C. Inspiratory stridor was present, and there was tenderness on palpation in the left neck without crepitus. The oropharynx was mildly inflamed. The lungs were clear, cardiac rhythm was regular, and heart sounds were normal with no murmur or rub. The remainder of the physical examination was normal.

The patient was immediately treated with oxygen at 2 L/min by nasal canula and an aerosolized nebulizer with racemic epinephrine. This resulted in a marked improvement in symptoms and in the inspiratory stridor.

An arterial blood gas determination demonstrated a pH of 7.48; $\mathrm{Pco_2}$, 28 mm Hg; and $\mathrm{Po_2}$, 112 mm Hg with 99% oxygen saturation. The white blood cell count was 11,800/mm^3, with 63% segmented neutrophils, 32% lymphocytes, and 5% monocytes; the hematocrit was 38%. Electrolytes, blood urea nitrogen, glucose, and amylase levels were within normal limits.

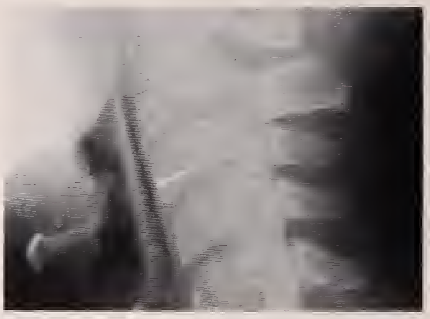

FIGURE 1. Lateral neck film demonstrating air in the soft tissues posterior to the pharynx and trachea and surrounding the esophagus.

The chest radiograph was normal, but lateral neck films revealed air in the soft tissues posterior to the pharynx and trachea, surrounding the esophagus and extending into the superior mediastinum (Fig 1). A computed tomographic (CT) scan of the neck with intravenous contrast was obtained with 5 mm cuts extending from the foramen magnum to the thoracic inlet, followed by 1 cm cuts through the mid-thoracic region. There was gas in the soft tissues and retropharyngeal region extending laterally to the left, as well as gas adjacent to the esophagus and great vessels at the thoracic inlet on the left side (Fig 2). Mediastinal gas extended inferiorly to the carina. No evidence of a foreign body or mass was identified. A diatrizoate meglumine

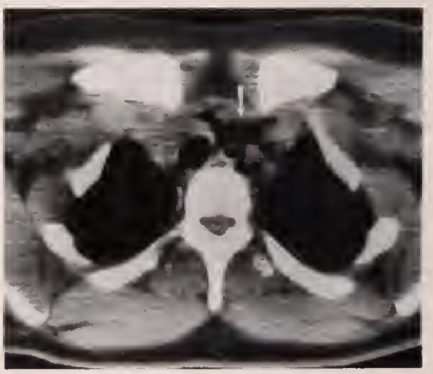

FIGURE 2. CT scan at the level of the clavicles demonstrating gas adjacent to the esophagus and great vessels on the left.

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swallow test revealed air in the neck and superior mediastinum but no evidence of an esophageal leak.

An otolaryngology consult was obtained. The patient was placed on intravenous antibiotics as follows: cefazolin, 1 g every eight hours; metronidazole, 500 mg every eight hours; and gentamicin, 100 mg initial dose and 80 mg every eight hours thereafter. She was taken to the operating room the day of presentation and underwent intraoperative laryngoscopy, endoscopy, left neck exploration, and tracheotomy. Laryngoscopy revealed no abnormalities. Endoscopy was performed to the distal portion of the esophagus; examination of the entire circumference of the mucosa did not reveal any lesions. An incision was made at approximately two fingerbreadths above the left clavicular head, and dissection was carried out to the carotid sheath with bubbles of air noted during dissection. Drains were placed, and a no. 7 Portex tracheostomy tube was inserted. The patient made an uneventful recovery and was discharged home seven days later.

DISCUSSION

Esophageal rupture can be fatal. Delay in diagnosis and treatment is a major factor contributing to morbidity and mortality. Prompt diagnosis of esophageal perforation requires both a high degree of clinical suspicion and familiarity with the full range of precipitating circumstances and resulting signs and symptoms.

This case demonstrates a unique presentation of this condition. The patient had neither lower thoracic pain nor subcutaneous emphysema which, along with vomiting, form Mackler's classic triad of findings in Boerhaave's syndrome. Furthermore, shortness of breath was the dominant symptom and inspiratory stridor was a prominent presenting manifestation. Our review of the literature failed to disclose a previous case with inspiratory stridor.

We believe that this patient's symptoms resulted from the unusual location of the perforation in the cervical esophagus. Most esophageal perforations occur in the distal third, attributable in some cases to a lack of muscularis mucosa in the distal esophageal wall.[6] Cervical esophageal perforation has been described after endoscopy.[7] Although a cervical esophageal tear was never visualized in this patient, radiographic studies clearly demonstrated that a perforation had occurred.

The etiology was most likely the episode of emesis in conjunction with swallowing a piece of cookie. Possibly a sharp edge of the cookie pierced the esophagus, and the large intraesophageal pressures produced by the patient's emesis forced air into the surrounding tissues. Although the patient did have a history of a malignant salivary gland tumor seven years previously, no evidence of recurrence was found at the time of surgery, and we believe that this event was unrelated.

It is possible that this patient's perforation was actually in the pharynx rather than the cervical esophagus. The soft tissue air seen on both the lateral neck film and the CT scan extended from the level of the pharynx all the way down to the mediastinum and could have resulted from a pharyngeal tear. Clinical distinction between these two entities is impossible unless the perforation is actually identified; in such cases the term pharyngoesophageal tear might be more appropriate.

The usual treatment of esophageal rupture is surgical intervention and repair of the esophageal leak. This patient underwent prompt surgical intervention; however, repair was impossible since no esophageal tear was visualized. She did well clinically in spite of this. Perhaps initial treatment with close observation, nasogastric drainage, intravenous fluids, and antibiotics is reasonable, reserving surgical intervention for patients whose condition does not promptly improve.

This case demonstrates that perforation of the cervical esophagus should be considered in the differential diagnosis of a patient presenting with shortness of breath and inspiratory stridor, especially if vomiting preceded the onset of these symptoms. Lateral neck films should be obtained and carefully examined for the presence of soft tissue air; this can provide the clue to esophageal rupture.

REFERENCES

1. Boerhaave H: Atrocis, nec descripti prius, morbi historia. Secundum medicae artis leges conscripta. *Lugduni Batavorum Boutesteniana* 1724. Translated by Derbes VJ and Mitchell RE. *Bull Med Libr Ass* 1955; 43:217–240.
2. Wilson DR, Cote RW, Bailey LL: Boerhaave syndrome: Lifter's liability. *West J Med* 1980; 133: 517–519.
3. Kennard HH: Rupture of oesophagus during childbirth. *Br Med J* 1950; 1:417.
4. Bank JG, Bancemicz J: Perforation of esophagus: Experience in general hospital. *Br J Surg* 1981; 68:580–584.
5. Tidman MK, John HT: Spontaneous rupture of oesophagus. *Br J Surg* 1967; 54:286–292.
6. Kuwano H, Matsumata T, Adachi E, et al: Lack of muscularis mucosa and the occurrence of Boerhaave's syndrome. *Am J Surg* 1989; 158:420–422.
7. Graeber G, Niezgoda J, Albus R, et al: A comparison of patients with endoscopic esophageal perforations and patients with Boerhaave's syndrome. *Chest* 1987; 92:995–998.

Diltiazem-induced myoclonus

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Calcium channel blockers are being used for an increasing number of medical and neurologic conditions, with a concomitant increase in reports of their side effects. Myoclonus has been reported as a rare side effect of the calcium channel blockers nifedipine[1,2] and verapamil.[3] To our knowledge, myoclonus has never been reported in the literature as a side effect of diltiazem. We report two patients in whom myoclonus developed as a result of sustained-release diltiazem (Cardizem-SR) use.

CASE REPORTS

Patient 1. A 75-year-old woman with hypertension and chronic obstructive pulmonary disease (COPD) presented to Kings County Hospital Center on August 2, 1990, with involuntary episodic jerks involving all her extremities and progressive somnolence. Her medications included transdermal nitroglycerin, triamterene-hydrochlorothiazide, pentoxyphylline, salsalate, ipratropium bromide inhaler, albuterol inhaler, and sustained-release diltiazem, 180 mg/day. She had initiated diltiazem therapy more than two years previously at a dose of 30 mg four times per day and was changed to the sustained-release preparation several months before this admission.

Examination showed mild dementia and multifocal myoclonus. Laboratory studies revealed a normal complete blood cell count and electrolyte levels, including magnesium and calcium. The arterial blood gas study showed the following: pH 7.35; Po_2, 45 mm Hg, Pco_2, 62 mm Hg; bicarbonate, 37.3; O_2 saturation, 77%. An awake electroencephalogram (EEG) showed no epileptiform activity.

On delving further into her history, she admitted to medicating herself with diltiazem at a dose exceeding 1,500 mg/day. After discontinuing the medication, the myoclonus gradually disappeared, and her mental status returned to normal.

Several weeks later, when she resumed self-medicating with high doses of diltiazem, her myoclonus returned. Discontinuing the medication again resulted in disappearance of the myoclonus and confusion. No other changes in her medications were necessary.

Patient 2. A 60-year-old woman with hypertension reported awakening with epi-

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sodic involuntary jerks involving all extremities, particularly her shoulders, two days after initiating therapy with 90 mg/day of sustained-release diltiazem. She was taking no other medications. The myoclonus was exacerbated by stress, anxiety, and emotional turmoil.

Physical examination revealed multifocal myoclonus particularly affecting the proximal muscles. Laboratory studies, including a complete blood cell count, electrolytes, magnesium, calcium, and arterial blood gas measurements were all within normal limits. An awake EEG was normal; back averaging the EEG showed no time-locked cortical events that corresponded to the jerky movements.

After discontinuing diltiazem, her myoclonus gradually diminished. One evening, when her blood pressure was transiently elevated, she took nifedipine, 10 mg orally. This resulted in temporary exacerbation of the myoclonus. Her symptoms disappeared approximately two weeks after discontinuing therapy with calcium channel blockers.

DISCUSSION

Myoclonus has been reported as a rare side effect of the calcium channel blockers nifedipine[1,2] and verapamil.[3] To our knowledge, it has never been reported in the literature as a side effect of dilitiazem. One of the manufacturers of diltiazem has received only one report of myoclonus, which involved "a 71-year-old male who experienced myoclonic jerks of the arms while receiving diltiazem 180 mg/day (duration of therapy was not reported). The event was reported to abate after discontinuation of diltiazem and recurred after rechallenge. Additional information such as concomitant medications and medical history was not provided." (Personal communication, Deborah Wilson Tady, Marion Merrell Dow, Inc, Kansas City, MO, 1991).

The patients described here developed myoclonus caused by diltiazem. The dose and duration of therapy differed significantly. In both cases, myoclonus abated when the offending medication was discontinued. In the first patient, rechallenge resulted in recurrence. In the second patient, rechallenge with another calcium channel blocker resulted in recurrence.

Diltiazem, like other calcium antagonists, can cause myoclonus. This may (as with the first patient) or may not (as with the second patient) be a dose-dependent effect. In our second patient, myoclonus was also not selective for a particular calcium channel blocker, since she proved sensitive to both diltiazem and nifedipine. In one previous report, diltiazem challenge of a patient with verapamil-induced myoclonus did not result in symptom recurrence.[3] However, the dosages of these calcium antagonists may have differed, and the patient may have been sensitive only to higher dosages.

Although a central effect of calcium channel blockers at the level of the basal ganglia has been postulated in myoclonus[2,3] proof of the mechanism is lacking. Three types of neuronal calcium channels have been described, and their role in the regulation of neurotransmitter release has been postulated.[4,5] Although a thorough understanding of the pathophysiology is lacking, we draw attention to this phenomenon as a reversible cause of myoclonus and encephalopathy.

REFERENCES

1. De Medina A, Biasini O, Rivera A, et al: Nifedipine and myoclonic dystonia. *Ann Intern Med* 1986; 104:125.
2. Pedro-Botet ML, Bonal J, Caralps A: Nifedipine and myoclonic disorders. *Nephron* 1989; 51: 281.
3. Hicks CB, Abraham K: Verapamil and myoclonic dystonia. *Ann Intern Med* 1985; 103:155.
4. Miller RJ: Multiple calcium channels and neuronal function. *Science* 1987; 235:46–52.
5. Tsien RW, Lipscombe D, Madison DV, et al: Multiple types of neuronal calcium channels and their selective modulation. *Trends Neurosci* 1988; 11: 431–438.

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

The *New York State Journal of Medicine*

TO THE EDITOR: I want to compliment Dr Milton Gordon on his excellent article entitled "An update on the *New York State Journal of Medicine*" that appeared in the July 1992 issue.[1]

The *Journal* plays an important role as an outreach to young physicians and medical students. Through the *Journal*, they become linked to the Medical Society of the State of New York (MSSNY) and are able to find a respected peer-review publication for their scientific articles. My first article was in the *Journal* many years ago, and I have been actively involved with medical society affairs since then.

While MSSNY has a critical role to play with regard to socio-economic and political issues, it must retain a scientific and educational link if it is to survive and grow. For this purpose, the *Journal* is indispensable.

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[1] Gordon M: An update on the *New York State Journal of Medicine. NY State J Med* 1992; 92:294–296.

TO THE EDITOR: I have always enjoyed reading the *New York State Journal of Medicine*. As a former New Yorker, I frequently peruse the *Journal*. Fiscal restraints preclude my subscribing, but my medical center's library subscribes. The report by the Committee on Bioethical Issues, "Ethical considerations concerning the HIV-positive physician," that appeared in the April issue was particularly valuable to me.[1] The Arizona Infectious Disease Society recently sent written recommendations to the Governor of Arizona on this issue. It was reassuring to me to note that the Committee's well-reasoned report had the same conclusions as those of the Arizona Infectious Disease Society.

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[1] Rosner F: Ethical considerations concerning the HIV-positive physician. *NY State J Med* 1992; 92:151–155.

The role of third party payors in medical care

TO THE EDITOR: I wish to sympathize with John D. Krafchuk, MD, but hasten to add that "managed care," is precisely an effort at "influencing patient care decision-making through case by case assessment."[1,2] It is described by Sharfstein as the "most explicit and direct effort to regulate and control physicians by modifying their decisions."[3] He further states that "It is therefore a direct intrusion on professional autonomy and judgement."[3]

Goodman, Brown, and Deitz made the following observations: "In 1988, only five million people with indemnity insurance had their mental health care reviewed by case managers. It is estimated that by 1993 that number will increase to 50 million, and less than 20% of the population will have traditional indemnity insurance without any element of managed care."[4]

I believe that "managed care" will destroy the medical profession, as we have known it, unless we stop it, now.

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[1] Krafchuk JD: The role of third party payors in medical care. *NY State J Med* 1992; 92:323-324.
[2] Field MJ, Gray BH (eds): *Institute of Medicine (US) Committee on Utilization Management by Third Parties: Controlling Cost and Changing Patient Care? The Role of Utilization Management.* Washington, DC, National Academy Press, 1989.
[3] Sharfstein SS: Utilization management: Managed or mangled psychiatric care? *Amer J Psych* 1990; 147:965.
[4] Goodman M, Brown J, Deitz P: *Managing Managed Care, Washington*, DC, American Psychiatric Press, 1992, p 19.

The changing winds

TO THE EDITOR: We are currently witnessing a remarkable change in the direction of our society. For the first time since women won the right to vote, we are an audience to the rising power of women. In health care, it has become very clear that the numbers of women who are medical students and residents is markedly increasing. They are no longer willing to be assigned solely to nursing and social work. Organized medicine has also begun to reflect this shift in constituency.

The phenomenon, however, that is more striking is their forceful entry into the political arena. I watched part of the Anita Hill proceedings with a very astute observer of the scene. A prescient remark of hers stuck with me. She predicted that the behavior of the senators toward Ms Hill and her response to them would galvanize women in the same way that Rosa Parks helped set motion the civil rights struggle.

History has proved her to be right on target. The number of women who are running for office, including the US Senate, has dramatically increased. One has to look no further than New York, Pennsylvania, Illinois, and California to appreciate what is happening.

As we reflect on the leadership role of women in Israel, India, Great Britain, and elsewhere, it would seem to be only a matter of time before we see the same. If a third-party, male candidate can so intrigue the voters of this country, can a women president be far behind?

Given our need as a medical society to sense the changing winds in politics, this ground swell must guide us as we proceed to make things better for our patients and ourselves.

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Pseudoleukemia in a patient with Hodgkin's disease

TO THE EDITOR: Pseudoleukemia is a recognized entity that, on rare occasions, can mislead even the most experienced hematologist. The benign nature of this diagnosis contrasts sharply with the alternative diagnosis of acute promyelocytic leukemia (APL). A case of pseudoleukemia is described in this report, and in the discussion an attempt is made to review the literature and to focus on subtle morphological findings that may help the clinician arrive at the appropriate diagnosis as early as possible.

Case Report. A 38-year-old male with a past medical history of Hodgkin's disease was in good health until developing fevers, malaise, abdominal pain, and jaundice in mid-1991. His Stage IIIB nodular sclerosing Hodgkin's disease had been initially diagnosed in August 1980 after presenting with cervical, mediastinal, and abdominal lymphadenopathy. He achieved a remission after six cycles of nitrogen mustard, vincristine, prednisone, and procarbozine (MOPP), had remained in complete remission after completing eight cycles of MOPP chemotherapy, but relapsed less than one year later and was restarted with alternating MOPP/ABVD (adriamycin, bleomycin, vinblastine, and dacarbazine), followed by radiation therapy to the mantle area. He remained in complete remission for the next nine years.

On admission to a local hospital in mid-1991, an exploratory laparotomy revealed extensive tumor growth involving the liver and biliary tree, and biopsies confirmed nodular sclerosing Hodgkin's disease. He was discharged after placement of a Groshong subclavian catheter and received MOPP chemotherapy as an outpatient at the end of the month through the catheter. He developed pleuritic chest pain that evening, which gradually resolved. He returned to the office the following week, where yellow fluid was aspirated from the catheter. X-rays localized the catheter tip to the posterior mediastinum. He was readmitted seven days after his chemotherapy for catheter removal and replacement. His hemoglobin was 11.6 g/dL, hematocrit was 35%, platelet count was 336,000/µL, and white blood cell (WBC) count was 5,500/mm^3 with a differential of 19% segmented neutrophils, 3% bands, 60% lymphocytes, 1% atypical lymphocytes, 17% monocytes for an absolute neutrophil count (ANC) of 1,210/µL. Post-operatively, he developed a temperature to 102.6°F. He defervesced on ceftriaxone and was later switched to vancomycin (500 mg every six hours) when blood cultures grew *Staphylococcus epidermitis.* Fevers recurred after 11 days of antibiotic therapy, accompanied by nausea, vomiting, and night sweats. Cultures were unrevealing. At that time, his WBC count was 2,800/mm^3 with a differential of 17% segmented neutrophils, 74% lymphocytes anhocytes, 25% monocytes (ANC = 280). His platelet count was 219,000/µL, and hematocrit was 33% mL/dL. Bone marrow aspirate and biopsy showed myeloid proliferation with a presumptive diagnosis of APL. Twenty-three days after chemotherapy, he was transferred to Strong Memorial Hospital, Rochester, NY, for further evaluation. Medications at that time included ceftriaxone, clindamycin, solumedrol, and mycostatin; vancomycin had been discontinued on the previous day.

Physical examination showed a thin, ill-appearing man with an oral temperature of 97.8°F, blood pressure of 110/80, heart rate of 72/min, and a respiratory rate 18/min. His skin was pale without rashes or ecchymoses. No lymphadenopathy was palpable. The Groshong catheter exit site was without erythemata or drainage. His abdomen had normal active bowel sounds and discomfort on palpation. Laboratory values included WBC count 3,300/mm^3, differential of 17% segmented neutrophils, 1% bands, 43% lymphocytes, 55% monocytes, and two nucleated RBCs per 100 WBCs (ANC = 33), a platelet count of 144,000/µL, and a hematocrit of 26%. Right hilar adenopathy was seen on chest films. A peripheral blood smear showed granulocytopenia with rare promyelocytes. Bone marrow examination revealed intense myeloid proliferation with predominant promyelocytes and occasional myelocytes on smear. Blast cells and Auer rods were not visualized. Cell marker studies did not show a monoclonal population. On the day after admission, laboratory results included the following: WBC, 7,400/mm^3 with a differential of 22% segmented neutrophils, 12% bands, 33% lymphocytes, and 31% myelocytes (ANC = 2,500). Dohle bodies were seen on the peripheral smear. A repeat bone marrow biopsy on five days after admission was hypercellular with normal appearing maturation of all three cell lines, and myeloid hyperplasia with increased numbers of mature neutrophils.

Discussion. The course of events in this patient are consistent with the diagnosis of "pseudoleukemia" and provide an excellent example of this condition. In this entity, the bone marrow has the appearance of APL with maturation arrest and a predominance of promyelocytes. This is felt to represent a recovery phase from agranulocytosis. Rare case reports have documented an association with certain medications including dapsone [1] and methotrexate.[2] Other reports note an association with certain infections,[3,4] including two cases of *Pseudomonas aeruginosa* infection.[5] Promyelocytes in all cases seem to share the characteristics of a prominent paranuclear Golgi zone, as opposed to the diffuse granulation seen in malignant promyelocytes.[6] The promyelocytic granules in APL are also typically larger than in normal promyelocytes. The promyelocytes of pseudoleukemia also lack Auer rods, a common feature of APL. The exact cause of the leukemia-like appearance of the bone marrow has not been determined. Some have theorized that recovery of myeloid cells after damage to multipotential stem cells could give the appearance of maturation arrest. Experimental data support infection as a possible cause of pseudoleukemia. Endotoxin from gram-negative organisms causes release of neutrophils from the bone marrow resulting in a marked depletion of mature granulocytes.[7] Endotoxin also stimulates increased myeloid-colony-forming unit production which could explain the myeloid proliferation seen in bone marrows in pseudoleukemia.[8]

There are many possible contributing factors to this patient's "pseudoleukemia." He had documented staph epidermitis bacteremia, as well as a second febrile illness. In addition, he was treated with multiple antibiotics. He also had received MOPP chemotherapy 23 days earlier. This chemotherapy should normally have lowered his counts by ten days post-treatment, but his nadir may have been delayed due to the route of administration. The patient received his MOPP chemotherapy through his initial catheter into his mediastinum. Thus, delayed absorption may have contributed to the acute leukemia-like picture. The characteristics that helped distinguish this case from APL include the lack of Auer rods, absence of blasts, the granulation pattern of the promyelocytes, and the rapid spontaneous recovery of mature myeloid proliferation.

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1. Levine PH, Weintraub LR: Pseudoleukemia during recovery from dapsone-induced agranulocytosis. *Ann Int Med* 1968; 68:1060–65.
2. Gailani SD: Pseudoleukemia during recovery from methotrexate toxicity in psoriasis. *NY State J Med* 1972; 72:1054–1056.
3. Dreskin SC, Iberti TJ, Watson-Williams EJ: Pseudoleukemia due to infection. *J Med* 1983; 14: 147–155.
4. Sanal SM, Campbell EW, Bowdler AJ, et al: Pseudoleukemia. *Postgrad Med* 1979; 65(6):143–147.
5. Lanham GR, Dahl GV, Billings FT, et al: Pseudomonas aeruginosa infection with marrow suppression simulating acute promyelocytic leukemia. *Am J Clin Pathol* 1983; 80:404–408.
6. Innes DJ, Hess CE, Bertholf MF, et al: Promyelocyte morphology. *Am J Clin Pathol* 1987; 88:725–729.
7. Christensen RD, Rothstein G: Exhaustion of mature marrow neutrophils in neonates with sepsis. *J Pediatr* 1980; 96:316–318.
8. Golde DW, Cline MJ: Endotoxin-induced release of colony-stimulating activity in man. *Proc Soc Exp Biol Med* 1975; 149:845–148.

ERRATA

In the May 1992 issue of the *Journal,* on page 182, there is an error in Table I. Under Group 1, the headings AIDS and Advanced Cancer were reversed. The lines should read AIDS-144-97-20 and Advanced Cancer-36-246-124. On page 184 of the same issue, the 29th line should read "However, we have no reason to believe that the law would have affected the use of DNR differently in such patients than in those patients who actually died, and in our study of deaths, the use of CPR and DNR did not appear to change regardless of disease severity.

Rationing America's medical care

Rationing America's Medical Care: The Oregon Plan and Beyond. Edited by Martin A. Strosberg, Joshua M. Wiener, Robert Baker with I. Alan Fein. 238 pp, Washington, DC, The Brookings Institution, 1992. $12.95 (softcover)

Access to health care was the dominant theme during the 1960s and 1970s. Subsequently, quality of care and cost containment became the health policy issues. As costs soared in the 1980s, people could not afford needed medical care. Rationing of health care erupted into a public debate before and after the passage of the Oregon Basic Health Services Act in 1989.

Discussing how Oregon dealt with limited resources, Welch and Larsen[1] quoted an articulate Oregonian:

> If Oregon truly has limited resources, it would make sense to allocate those resources to the individuals who would benefit the most. At the same time, perhaps we should withhold resources from those who would survive without our assistance, and we should also deny resources to those who may soon die even with our help.

Kevin Concannon,[2] director of the Oregon Department of Human Resources, commented: "Sure, people will die because of this decision .... This is a striking example of the difficult health choices officials have to make with limited health resources."

Oregon's Act made rationing a legislative reality as professional and public representatives created a priority ranking list. A wave of controversy ensued and the Brookings Institution convened a conference "Rationing America's Medical Care: Opening Pandora's Box?" on May 20, 1991. This book contains the 18 presentations from that conference in five sections: Introduction and Summary; Policy Questions; Political Perspective; Physician Perspectives; and Legal and Philosophical Reflections. Almost every article has a conclusion and a list of pertinent references. Alternatives, severe criticisms, and faint praise oscillate throughout the book. Ethicist Robert M. Veatch concluded: "The experiment is well worth trying—indeed, morally imperative—but certain elements of the approach will do serious moral harm if they are not corrected." By contrast, Sara Rosenbaum of the Children's Defense Fund, contends: "The Oregon Plan is not an experiment that can be justified legally, scientifically, programmatically, or ethically." Recently, Fisher, Welch, and Wennberg[3] analyzed Oregon's statistical data, and theoretically identified and then eliminated excess health care capacity. These researchers estimated savings of $47 million if ceilings were placed on discretionary hospital medical admissions. That saving would obviate the need for Oregon's priority list.

Oregon's law expanded Medicaid coverage, mandated universal employer health care coverage, and established a high risk pool for medically uninsured individuals. In addition, a publicly appointed Oregon Health Services Commission (OHSC) was authorized. OHSC Commissioners, with community participation, created 17 priority categories in three groupings ranging as follows: no. 1) Acute, fatal conditions for which treatment prevents death and provides full recovery (myocarditis, appendectomy); no. 10) Acute, nonfatal conditions for which treatment is likely to bring a return to previous health (thyroiditis, vaginitis); no. 17) Fatal or nonfatal conditions for which treatment provides minimal or no improvement in quality of life (viral warts, repair fingertip avulsion that does not include fingernail). Pairing 709 health conditions with one or more treatments and fitting all into the 17 categories, the priority list materialized by means of lexicographic ordering, according to economists.[4] Oregon's 1991 legislature budgeted Medicaid reimbursement through health condition number 587; the remaining 122 were unfunded. Funds paid for the following percentages in each of the three groups in the 17 item priority list: 98% (essential); 82% (very important); and 7% (minimal gain and high cost).

Interestingly, former Colorado Governor Richard D. Lamm[5] declared that "Every country and system rations health care. We pretend we don't. . . . We find it distasteful to admit the obvious: That we can't give presidential health care to every citizen." In this book, Joshua M. Weiner points out that "Rationing, mostly in the form of limiting services by ability to pay, is already widely practiced in the United States." Grumet[6] found rationing that existed via the third party insurer's secret weapon, inconvenience. Eight administrative actions did the job: procedural complexity; use of exotic terms; slowdowns; shifting of procedures; fail-safe payment systems; overlapping coverage; fragmentation of transactions; and uncertainty of coverage. Gail Wilensky,[7] now President George Bush's health advisor, was surprised by the crescendo of the discussion about rationing. She had taken it for granted that rationing was an economic fact of life.

Jones[8] defined rationing as "the limiting of potentially beneficial resources." No longer can health resources be discussed without taking into account the concept of rationing. There simply are fewer resources to go around to provide an equitable distribution of health care services to all who want them. Health economist, Victor Fuchs,[9] commented that "trade-offs must be made." Individual needs must be weighed against societal determinations.

Rationing is a critical strategic health planning issue closely connected to the concepts of cost containment and the allocation of limited resources.[10] Of course, rationing applies equally to four major types of resources: personnel, facilities, equipment and supplies, and funding.

Personnel include professionals, ancillary staff, management, clerical, and even volunteers. Facilities refer to hospitals, nursing homes, satellite centers, or any other space used for activity. Equipment/supplies include the standard and high technology apparatus as well as the usual papers and clips needed for any program. Funding relates to public and private moneys and even in terms of exchange or bartering.

Obviously, some population groups will agonize most from rationing. As it turned out, low income and unemployed persons were the ones who suffered from implicit rationing.[11] This implicit and unequal rationing of health care appears even more paradoxical in light of the alleged surplus of physicians and hospital beds in the country.

In the past, physicians abided by the maximalist principle: Use whatever services are necessary to obtain the best medical results for the patient. However, resource allocation decisions shifted from the physician to the health care institution. This shift was spurred by national cost containment policies creating programs such as Certificate of Need (CON), Quality Assurance (QA), Peer Review Organizations (PROs), Managed Care entities, and a Prospective Payment System (PPS), using Diagnosis Related Groups (DRGs) for reimbursement.[12] Institutional administrators "educated" staff physicians to consider how individual medical service decisions affected not only the patients, but also the institution and society as a whole.

Health care has been rationed for some time in Great Britain. There, health policy analysts developed guidelines to ensure rational choices rather than having a state legislative body respond emotionally to community sentiment. Clinicians are expected to go through a three step process in determining the quantity and quality of medical services to be provided:[13] 1) Consideration of how much benefit is likely and the probabilities of achieving that benefit; 2) Evaluation of the financial and personal costs to the patient in discomfort, hazard, or indignity; 3) Finally, clinicians weigh these two considerations against each other in making a decision.

In addition, British physicians consider the following five "U's" which are generally defined as inappropriate activities:

1. Unsuccessful: when the patient is too far gone to benefit from medical attention.
2. Unnecessary: when the patient's condition is not serious enough to justify evaluation and monitoring.
3. Unkind: when care prolongs what is a poor quality of life or imposes potential hurt such as mutilating surgery or toxic chemotherapy for an advanced cancer patient.
4. Unwise: when medical resources are diverted from an area where they could bring greater benefit.
5. Unsafe: when the risk of complication outweighs the desired benefits.

Rationing guidelines may remove the onus from one set of actors of deciding a patient's welfare. A significant deliberation in generating guidelines is the application of cost-benefit, cost-effectiveness, and cost-utility analyses to the community as well as to the individual. Logically, health care administrators can serve as the advocate of the silent, larger community. Without advanced planning specifying who gets what and how resources are allocated, newly formed Committees may make decisions on "who shall live when not all can live."[14]

Are we getting our money's worth? A comparison of 1989 international data on infant mortality[15] and per capita health care expenditures[16] is startling. With an infant mortality death rate of 9.80, the United States ranked 22nd and spent $2,354 per capita. Japan spent $1,035 and ranked first with 4.59. With 8.42, the United Kingdom ranked 17 but spent only $836. Even Italy in 20th place with 8.80 spent less than half as much as the United States with $1,050. Since infant mortality is alleged to be a measure of effectiveness of a nation's health care services, it is apparent that the United States is spending more and getting less. These better results elsewhere are attributed to lower health care prices, lower administrative costs, and to less available high technology. In view of this comparison, reallocation of our resources could certainly be a viable contemplation, but public attitudes in the United States may require a metamorphosis.

Ethicist Daniel Callahan[17] identified three major obstacles to rationing: 1) the individual's belief in his/her right to unlimited medical care; 2) the physician's maximalist approach to medical care; and 3) health care insurance that insulates the individual from feeling the impact of the cost of treatment. A combination of those three factors results in a mass denial by society of the necessity of rationing health care services. Seemingly, people believe that medical and health resources will always be available when needed. Public attitudes need to acknowledge that health care resources are finite. Yet, people surmise that rationing can be evaded through cutting the defense budget, efficiency, technological innovation, or by outproducing our needs. If a majority of society continues to believe that the health care system can be fine-tuned to overcome the need to ration resources, national beliefs will not change. Callahan[17] commented:

> A view of reality that has optimistically declared limits to be unnecessary and even to think about them as dangerous, and that has self-righteously declared any denial of a capacity to meet some needs in an affluent society to be morally wrong, is one at war with any realistic notion of human community. That notion should encompass mutual help, mutual sacrifice, and mutual limits. The present system of focusing on individual needs leaves no room for the broader needs of community as a whole.

This book exhaustively reviews all sides of the rationing dilemma. Anyone with any concern about rationing will comprehend and benefit from the diversity and intensity of the opinions expressed. About the only thing missing is an index that would allow for finding similar subject matter in each of the presentations.

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1. Welch HG, Larson EB: Dealing with limited resources: The Oregon decision to curtail funding for organ transplantation. *N Eng J Med* 1988; 319: 171–173.
2. Lund DS: Oregon to fund Medicaid transplants again. *Am Medical News* 1988; 31:11.
3. Fisher ES, Welch HG, Wennberg JE: Prioritizing Oregon's hospital resources: An example based on variations in discretionary medical utilization. *JAMA* 1992; 267:1925–1931.
4. Grannemann TW: Priority setting: A sensible approach to Medicaid policy? *Inquiry* 1991; 28:300–305.
5. Perrone J: As needs, costs rise, states face problems of health care rationing. *Am Medical News* 1988; 31:2,33.
6. Grumet GW: Health care rationing through inconvenience: The third party's secret weapon. *N Eng J Med* 1989; 321:607–611.
7. Wilensky GR: Making decisions on rationing. *Business and Health* 1985; 3:36–38.
8. Jones L: Brookings report addresses rationing of care. *Am Medical News* 1989; 32:9.

9. The rationing of medical care. *N Eng J Med* 1984; 311:1572–2573.

10. Spiegel AD, Hyman HH: Resource means manpower, materials and money. *Strategic Health Planning, Methods and Techniques Applied to Marketing and Management.* Norwood, New Jersey, ABLEX Publishing Co, 1991, pp 103–158.

11. Gross J: What medical care the poor can have: Lists are drawn up. *N Y Times* March 27, 1989, p18.

12. Spiegel Ad, Kavaler F: *Cost Containment and DRGs: A Guide to Prospective Payment.* Baltimore, MD, National Health Publications, 1986.

13. Jennett B: Balancing benefits. *Health Serv J* 1986; 96:6–7.

14. Benjamin M: Medical ethics and economics of organ transplantation. *Health Prog* 1988; 69:47–53.

15. Improved US infant mortality picture still poor. *Am Med News* 1992; 35:9.

16. Scheiber GJ, Poullier JP, Greenwald LM: Health care systems in twenty-four hour countries. *Health Affairs* Fall 1991; 10:22–38.

17. Callahan D: Allocating health resources. *Hast Cent Reports* 1988; 18: 14–20.

FROM THE LIBRARY

CO-OPERATION

If a business man saw his business going to a competitor he would study the situation, find the reason, and remedy the conditions causing it. What are we doing to prevent our business going to the drugless healers and faddists who apply practically one treatment to all diseases and ailments? It is said on good authority that there are in this country 28,300 drugless healers with a following of 17,600,000 people.

The term "Charity" covers a "multitude of sins" in the medical profession. I would not complain of true need that receives our willing service, but of unnecessary charity and of that so-called charity that allows well-to-do people to take advantage of dispensary practice in the big cities. Do you know that the charity abuse in Chicago alone amounts to an average of $2,500 per year for every physician in the city? On the other hand I am told by a physician connected with the circulation department of the *Journal of the American Medical Association* that a canvass of the physicians of Chicago showed an average income of about $725 per year. Why should this condition prevail?

"If physicians would organize and co-operate with each other they could increase their efficiency in many things. Every member of a co-operative firm should be a general practitioner with some specialty. All should co-operate with each member of the firm in diagnosis and do it cheerfully. The firm could maintain one or more laboratories which would be under the direction of some member of the combination."

Co-operation seems to be the basis of most successful ventures. It is true all co-operative ventures have not been brilliant successes but the grocers and coal dealers in this city appear to have very effective co-operative associations and some of the druggists had a co-operative buying club that seemed to work well.

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(*NY State J Med* 1912; 12:335–336)

SET NO LIMITS: A REBUTTAL TO DANIEL CALLAHAN'S PROPOSAL TO LIMIT HEALTH CARE FOR THE ELDERLY

Edited by Robert L. Barry, OP, and Gerard V. Bradley. 134 pp. University of Illinois Press, 1991. $22.95 (hardcover)

This volume of six essays, a prolog, and an epilog is based on papers presented at a symposium held at the University of Illinois College of Law in 1989. The focus is analysis and discussion of Daniel Callahan's work, particularly his views on medical goals in an aging society. Callahan, a medical ethicist, is Director of the Hastings Center.

To comprehend this book we must first understand what Callahan has stated in *Setting Limits* (1987), his more recent *What Kind of Life* (1990), and related papers.

Callahan calls for limits to treatment for the aged in a medically technological world. He forces us to acknowledge, at the extremes, two nightmare images. One is of arbitrary death by chronology, elderly men and women forced onto ice flows and shoved off into arctic waters without consideration of their humanity, right to existence or sanctity of life. The other is of aged bodies in serried ranks kept from the grave indefinitely by a vast array of tubes, solutions, and silicon chips.

His key points are the following:

After a person has lived out a natural life span, medical care should no longer be oriented to resisting death.

Provision of medical care for those who have lived out this span will be limited to the relief of suffering.

The existence of medical technologies capable of extending the lives of these elderly persons creates no presumptions whatsoever that the technologies must be used for that purpose.

The approaches of the authors of the work under review here range from the merely polemical to significant analysis. Marshall Kapp, in his chapter "Rationing Health Care: Legal Issues and Alternatives to Age-based Rationing," argues that an overall revamping of the health care system must take place before age-based rationing would be fair. The paper is a careful analysis of constitutional law as it may in future test cases apply to Callahan's concepts. As such, Kapp's view is legitimately argued, substantial, and interesting.

Lawrence DeBrock, in considering efficient allocation of care to the elderly, provides us a clear and valuable lesson in economics as applied to health expenditures. He recognizes that "From an economic standpoint . . . some form of rationing of resources *is* a necessity," without offering specific interpretation of Callahan's views.

Hadley V. Arkes gives his chapter the provocative title "When Bungling Practice is Joined to Absurd Theory: Doctor Philosophers and the Right to Die." He is concerned with the definition of life and offers an elegant, instructive statement. He establishes that we cannot claim a right to do a wrong; thus, there exists no right to a "substituted judgement" permitting withdrawal of treatment. However, since the subject of withdrawal of treatment is not primarily related to Callahan's work, Arkes seems off beam as one of Callahan's opponents.

Robert A. Destro, in "Target—the Elderly: A Nondiscrimination Perspective on . . . Setting Limits," gives a useful review of non-discrimination and civil rights law. However, in attacking Callahan he states that "How much is enough" is an impossible question to answer at the societal level. This statement is a dodge, a refusal to grapple fairly with Callahan's major point regarding technological excess. Destro's unwillingness to help solve such questions would allow a heart transplant on a brain dead patient by default, simply because the procedure is technically feasible.

The most noteworthy chapter is "Life as an Evil, Death as a Good, and Callahan's Inversion" by Robert P. George. Here the raw distinction between the pragmatists (such as Callahan) and those who argue from moral grounds alone is clear. Although George's discussion of the moral issue is fascinating, he does not respond to Callahan's view that technological advances have blurred the distinction between life and death. George appears to advocate no limits on the use of resources to ward off death. This reduction to absurdity conjures the image of an ancient skin stuffed with transplanted organs.

The papers by Robert L. Barry, Gerard V. Bradley, and Nat Hentoff are marked by gross inaccuracies and name-calling (references to Nazi genocide). The most tawdry of debating tactics is used: crafting false statements of Callahan's views and then attacking them. The other essays are the heart of the work. Although not rebuttals of Callahan, they are thoughtful and make the book worth reading.

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THE GUIDE TO MEDICAL PROFESSIONAL LIABILITY INSURANCE

By Kirk B. Johnson, JD, Martin J. Hatlie, JD, Illene Davidson Johnson, JD. 146 pp. American Medical Association, 1991. $24.00 (softcover)

This book falls into the "how to" category of publications, as it seeks to establish itself as a single reference source for the physician, on the often confusing and intimidating subject of professional liability insurance coverage. Historically, physicians like many other insurance consumers have been woefully ignorant of the details of malpractice coverage in general. This is somewhat surprising in view of the costly nature of professional liability coverage and the emotional burden of malpractice lawsuits. This book does an excellent job in describing the potentially complex matters concerning professional liability insurance in an easily understood and helpful fashion. Overall, the book helps to clarify the subject for the busy physician, and I cannot recall a publication resembling this one that presents this information in such a concise manner.

I believe that the authors are uniquely qualified to write on the subject matter given their background additionally the acknowledgments reflect contributions from well-recognized experts.

The guide introduces the reader to the basics of professional liability insurance and then takes the reader on a logically arranged, clear review of the types of coverage available. Subjects such as "Claims Made and Occurrence Policies" are discussed in detail. In addition, the different types of insurance carriers and how the practitioner may evaluate their performance and financial stability is covered in easily understood fashion. The authors also discuss how a physician may determine what constitutes sufficient insurance coverage and how special circumstances such as group practice, channeling programs, and employment by HMO or PPOs may effect the practitioner in the crucial area of insurance coverage. I found the book provided excellent information for the practitioner concerning what action should be taken if in fact a claim is made and how the physician may recognize and deal with potential conflicts of interest. The authors effectively point out the significance of certain types of policies and policy provisions and at times employ a cost benefit type of analysis that is generally helpful. Especially worthwhile is the occasional use of the question and answer technique by the authors. In my estimation, they have appreciated certain fundamental issues involving liability insurance most confusing to the practitioner and most often asked. The overall format is good with annotations in the margin and page references that are likely to be very helpful in understanding the material.

Considering that insurance department regulations vary widely on a state-by-state basis, the authors have done a good job in addressing generic insurance problems for the practitioner. I do feel that issues surrounding "claims made policies and prior acts coverages" could have been discussed in more detail. I found the "examples" on page 26 and 27 potentially confusing since they tended to group certain scenarios affecting coverages. In my experience, the issues and pitfalls surrounding claims-made insurance have been the most difficult for the practitioner and the least understood. This is the only area of the book that I felt could have benefited from additional discussion.

The material is certainly presented in unbiased fashion with the emphasis on giving the physician the necessary background detail and outlining ramifications of insurance coverage issues. Given the cost of insurance and the number of sleepless nights that may result from a uninformed decision, I would think that any practicing physician would find this publication a worthwhile addition to his or her library.

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HANDBOOK OF PRESCRIBING MEDICATIONS FOR GERIATRIC PATIENTS

By Judith C. Ahronheim, MD, 465 pp. Boston, Mass. Little, Brown and Co, 1992. $26.50 (softcover)

This book is a valuable aid for caring for older adults in all medical care settings. Because of the many chronic and acute conditions that afflict our geriatric patients, it is often a pharmacological nightmare attempting to treat all problems in a practical and logical fashion while not creating a polypharmacy catastrophe. This book is very sensitive, to this issue and the many other issues that confront the clinician who treats this population of people.

Dr Ahronheim has used many resources in compiling a reference text for commonly prescribed drugs. She did not attempt to list every known complication for each medication, but rather highlights common side effects or problems seen in the clinical setting. For some classes of medications, she also lists therapeutic problems that either increase the toxicity potential or decrease the effectiveness of the drug. Additionally, she gives numerous warnings about potential drug interactions.

This book is organized in a fashion that can be used in many health care settings. Unlike most reference books that usually gather dust in a bookcase, this handbook is small and compact, well referenced and organized, and indexed for quick references. Although the author, I am sure, attempted to keep the narrative to a minimum, it is not as austere as a "Harriet Lane"-type pocket reference. An aspect that I felt was very valuable was a brief reminder of the pharmacologic and physiologic effects of that class of drug.

It is obvious to me that Dr Ahronheim and the consulting staff she used have had a great deal of practical and clinical experience. Her discussions include practical suggestions and warnings about the appropriate methods of prescribing certain drugs, and suggestions on starting dosages and schedules for increasing the amount of the drug. Some of these recommendations are not listed in the *Physicians' Desk Reference* (PDR) but are rather found only by practical experience.

Dr Judith Ahronheim is well qualified for her role as an author. She is Associate Professor of Geriatrics and Adult Development and Medicine at Mount Sinai School of Medicine of the City University of New York. Her other clinical activities include being a medical attendant in the Department of Geriatrics at the same institution. She also used a number of different consultants from a wide range of medical subspecialties to review the accuracy of her text.

I strongly recommend *Handbook of Prescribing Medications for Geriatric Patients* for any clinician who treats and prescribes medications for the geriatric age group. I believe that this text will come to be regarded as standard reference for residents and practicing physicians. The fact that it is small enough to fit in a pocket allows it to be very portable, especially when one makes hospital rounds and in an ambulatory care setting. It is easy to use and very practical in its approach to this important and complex topic.

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FINAL EXIT: THE PRACTICALITIES OF SELF-DELIVERANCE AND ASSISTED SUICIDE FOR THE DYING

By Derek Humphry. 192 pp. New York, Carol Publishing Group, 1991. $16.95 (hardcover)

For those who have managed to avoid the publicity surrounding this book, it is the ultimate how-to manual. It covers the nuts and bolts of committing suicide, as well as how to assist someone else. Substantively, the topics range from the pros and cons of various methods to the insurance implications. The book provides very specific information about various drugs, including information about lethal dosages.

Author Derek Humphry, the former Executive Director and founder of the Hemlock Society, assumes that the reader accepts the concept that suicide for the terminally ill is an appropriate option. In a matter of fact approach, Humphry simply states that the actual decision is solely for the individual: "This book will help, but in the final analysis, whether you bring your life to an abrupt end, and how you achieve this, is entirely your responsibility, ethically, and legally." He barely mentions depression as a factor in decision making by the terminally ill, a criticism that apparently will be addressed in the revised paperback edition.

Part I is the guide to "self-deliverance." It addresses the illegality of assisting a suicide but notes the infrequency of detection. After surveying the various possible ways of committing suicide, Humphry takes a strong position on the advisability of the use of a plastic bag over the head, in addition to an overdose of drugs, to avoid the 10% chance that the individual might have an unusual tolerance or reaction. Some parts read like a suicide etiquette manual. There are pointers about how to avoid detection, such as don't turn off your answering machine. There are also guidelines about how to make it easier on others. One should warn one's friends, tell those closest to you, and leave a clear note taking full responsibility. It seems to be a matter of taste as to whether one wants one's family present. Consideration for others is paramount: "If you are unfortunately obliged to end your life in a hospital or motel, it is gracious to leave a note apologizing for the shock and inconvenience to the staff. I have also heard of an individual leaving a generous tip to a motel staff."

The much shorter Part II covers "Euthanasia Involving Doctors and Nurses" and has a less jarring tone, perhaps because we are more used to health care providers having control over the timing of death. It addresses the quirky Dr Jack Kevorkian, the proponent of his own "suicide machine." Although Humphry and Hemlock do not agree with all of Kevorkian's ideas, he is hailed as having forced the medical profession to confront the issues. Humphry surveys the arguments in favor of physician-assisted suicide but only acknowledges that there might be reasons why an *individual* physician might not want to participate. The wisdom of a social policy permitting doctors to assist is taken for granted.

Final Exit is actually more interesting as a phenomenon than as a book. It is awkwardly written, and the tone is strange. Its overly negative assessment of the utility of advance directives and pain control is a disservice to readers. Yet, it has sold over 500,000 copies and has been on the *New York Times* bestseller list for many months. It also has been found at the site of a number of suicides, not all of which involved terminally ill individuals.

The book clearly reflects the strong public interest in the topic of suicide for the terminally ill. People clearly want this information; it is unclear how many of the purchasers of this book ever will actually act on it. Nevertheless, modern medicine is seen as dehumanizing, expensive, and incapable of controlling pain and suffering at the end of life. The public's attitude is reflected not only in the sales of this book, but in the increased popularity of legislative initiatives to legalize physician-assisted suicide in some circumstances. One can dismiss Dr Kevorkian and Derek Humphry as mavericks, but they are striking a very strong chord in the patient community.

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CASARETT AND DOULL'S TOXICOLOGY: THE BASIC SCIENCE OF POISONS

Fourth Edition. Edited by Mary O. Amdur, PhD; John Doull, MD, PhD; and Curtis D. Klaassen, MD, PhD. New York, Pergamon Press. (hardcover)

Fifteen years ago, the first edition of this now classic text was published. It was used by medical and graduate students learning about environmental medicine before there was such a field of study. As a classic reference book and toxicology textbook, this edition continues the tradition of excellence and utility.

This book is organized so that even those among us with such well-defined interest like the "hypothetical" psychopharmacologist interested in the toxicity associated with illicit drugs and medications can quickly understand and use it. The first selection reviews the basic science and methodology of toxicology. I thoroughly enjoyed the short but very well written history and scope of toxicology chapter by Gallo and Doull. The second section reviews the systemic actions of known toxins. Naturally, I benefited a great deal by reading "Toxic Responses of the Nervous System" by Anthony and Graham. I especially appreciated the description of MPTP toxicity which came to the attention of addiction specialists when a chemists error gave street users of a meperidine derivative "designer drug" the contaminant MPTP as well. This neurotoxicant had exceptional specificity for the *substantia nigra* therapy producing irreversible Parkonsonism and to a lesser extent the noradrenergic locus coeruleus which would presumably produce alertness, alarm, and other deficits.

The third section takes specific toxins by chemical class or other logical groupings. Environmental toxicology chapters entitled "Food Additives" by Miller, "Air Pollutants" by Amdur, and "Water and Soil Pollutants" by Menzer are excellent reviews and exceedingly readable for the non-toxicologist. The final section's chapter entitled "Clinical Toxicology" by Rumack and Lovejoy reviews the toxicology that virtually all interns, primary care physicians, and medical practitioners manage. Identification of the poison, treatment of the poisoned patient, and methods to enhance elimination in the emergency room or office are concisely reviewed. Commonly prescribed medications such as salicylates, anticholinergics, antipsychotics, hypnotics, antidepressants, and benzodiazepines as well as drugs of abuse such as the stimulants, ethanol, opiates, PCP are reviewed from the clinical toxicologist's point of view. The volume ends with a superb overview of the issues and methods of risk assessment. Scala's comprehensive and timely chapter on risk assessment is made exceedingly reader friendly by nine well-conceived tables and figures.

I found *The Basic Science of Poisons* to be a comprehensive text with the added benefit of having stand-alone chapters which can be read and appreciated at anytime by almost any physician.

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CURRENT PRACTICE OF INTERVENTIONAL RADIOLOGY

Edited by Saadoon Kadir, MD. 779 pp, illustrated. Philadelphia, BC Decker Inc., 1991. $149.00 (hardcover)

This is an ambitious attempt to define the state of the art in vascular and nonvascular radiologic interventions. The text uses contributions by 180 authors to address vascular topics from angioplasty to Z-stents, as well as biliary and renal interventions, abscess drainage, biopsies, tubal recanalization, and a host of other therapeutic interventions.

An initial section on principles of interventional techniques includes appropriate material on organization of an interventional practice, with its requirements for dedicated space and equipment, support personnel, and hospital in-patient services. Basic techniques are well described. The remainder of the text organizes vascular and nonvascular interventions by anatomic region, a structure that initially seems cumbersome but ultimately works well.

Doctor Kadir is remarkably successful in dealing with such a broad and diverse field. The numerous topics are dealt with in crisp and concise chapters written by a group of national and international authorities. The emphasis is on "how I do it," with the inclusion of specific procedural details not often provided in textbooks. This is certainly to the reader's benefit, although alternative procedural approaches are not always discussed. Traditional referencing is replaced by a list of suggested readings at the end of each chapter.

In spite of the number of contributors, there is little duplication. Excellent editorial work has made for a smooth and coherent text. There is good cross referencing to related topics by other contributors. Illustrations are plentiful, and radiographic reproduction is excellent. The variety of specialized equipment is readily identified by manufacturer.

Dr Kadir has produced the best current text in this broad and dynamic field. It will be useful to the practicing interventionist, the radiologist-in-training, and the nonradiologic clinician who uses these services.

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AMBULATORY PERITONEAL DIALYSIS

Edited by Morrell M. Avram, MD, and Carmelo Giordano, MD. 340 pp, illustrated. New York, Plenum Publishing Corp, 1990. $69.50 (hardcover)

Chronic ambulatory peritoneal dialysis (CAPD) has become a common, accepted and effective method of treatment for patients with chronic renal failure. There is ample evidence in the literature, and this is further documented in this current volume, of the effectiveness of CAPD in reversing or controlling many of the chronic problems of end-stage renal disease. CAPD has been found to be especially useful for patients with diabetes mellitus and those with severe ischemic disease, as well as for individuals who wish a type of treatment that will free them from the regimentation of a hemodialysis unit. However, major problems with the technique continue. Many of these involve problems with the peritoneal dialysis catheters, largely related to infection. In addition, the peritoneal membrane appears to change over time, rendering ultrafiltration less effective in some patients. The quantification of peritoneal dialysis is in its infancy and methods are just now becoming established to individualize prescriptions for peritoneal dialysis.

This volume, edited by Drs Avram and Giordano, is a compilation of papers presented at the Fourth International Congress of Peritoneal Dialysis that took place from June 29 to July 2, 1987. The papers are divided into several subgroups and span the gamut of scientific and clinical issues in the use of this type of therapy. Several of the papers have been updated from this symposium and data up to 1990 are presented in a state of the art section. There are articles on the physiology, pharmacology, and morphology of the peritoneal membrane, on nutrition and metabolism in patients with peritoneal dialysis, on the emerging and constantly improving technology involved and on the most important issues of peritonitis. Clinical experience in adults, infants, and children is also well reviewed.

These papers offer a fascinating review of the state of the art of peritoneal dialysis.

The state of the art section is especially rewarding. Two authors with extensive experience in the field, Drs Maher and Oreopoulos, present their thoughts about where we are and where we are going with CAPD. Twenty-four articles address problems of the peritoneal membrane. Pharmacologic methods to increase ultrafiltration are presented as well as changes in the function of the peritoneal membrane in altered metabolic states. Most of the articles are clearly written, well presented, and undoubtedly reflect the care with which the editors reviewed and selected this material. This book is certainly useful for all nephrologists involved with the care of patients with chronic renal failure and should prove a stimulating and fascinating source of information for renal fellows to dip into during their training period.

KURT STENZEL, MD
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Cornell University Medical College
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New York, NY 10021

NEW FEDERAL LAW ON IMMUNIZATIONS

On April 15, 1992, a new Federal law took effect which requires that health care providers who administer diphtheria-tetanus-pertussis combined (DTP), measles-mumps-rubella combined (MMR) and polio vaccines inform patients, parents, or guardians of the potential side effects and benefits of these three immunizations before they are given. The law requires that the health care provider ensure that the patient, parent, or guardian has read the brochure, understands its content, and has had the opportunity to have questions answered before the immunization is administered. This procedure must be carried out each time a DTP, MMR, or polio immunization is administered.

The Centers for Disease Control (CDC) have developed three pamphlets which can be used to inform patients, parents, and guardians. Each brochure has a perforated consent form for patient records.

The Medical Society of the State of New York (MSSNY) is making these pamphlets available in bulk to members at a substantial discount. Vaccination brochure order forms may be obtained from the MSSNY which will insert a free immunization record form repro sheet for office use.

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OBITUARY LISTINGS

Manuel A. Auli, MD, Brooklyn. Died May 26, 1992; age 42. Dr Auli was a 1978 graduate of Boston University School of Medicine, Boston. His memberships included the Queens County Medical Society and the Medical Society of the State of New York.

Russell L. Battaglia, MD, Williamsville. Died May 10, 1992; age 78. Dr Battaglia was a 1939 graduate of the University of Buffalo School of Medicine, Buffalo. His memberships included the Erie County Medical Society and the Medical Society of the State of New York.

Duncan Leonard Best, MD, Middleburgh. Died July 18, 1991; age 78. Dr Best was a 1930 graduate of Albany Medical College of Union University, Albany. His memberships included the Schoharie County Medical Society and the Medical Society of the State of New York.

Earle W. Brauer, MD, New York City. Died September 5, 1991; age 74. Dr Brauer was a 1943 graduate of New York University College of Medicine, New York City. He was a Diplomate of the American Board of Dermatology. His memberships included the Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Society for Investigative Dermatology, New York County Medical Society, and the Medical Society of the State of New York.

Andrew DeRoetth, MD, New York City. Died February 22, 1992; age 71. Dr DeRoetth was a 1946 graduate of Northwestern University Medical School, Chicago. He was a Diplomate of the American Board of Ophthalmology. His memberships included the Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, the New York County Medical Society, and the Medical Society of the State of New York.

Sanford Miller Farrer, MD, Brooklyn. Died May 5, 1992; age 61. Dr Farrer was a 1956 graduate of Columbia University College of Physicians and Surgeons, New York City. He was a Diplomate of the American Board of Pathology. He was a Fellow of the American College of Pathologists. His memberships included the New York State Society of Pathologists, the American Public Health Association, the American Society of Clinical Pathologists, the Kings County Medical Society, and the Medical Society of the State of New York.

Milton Felix Gitlin, MD, Brooklyn. Died May 9, 1992; age 80. Dr Gitlin was a 1938 graduate of State University of New York Health Science Center, New York City. He was a Fellow of the American Academy of Family Practice and the American College of Allergists. His memberships included the Academy of Psychosomatic Medicine, the Kings County Medical Society, and the Medical Society of the State of New York.

David V. Habif, MD, New York City. Died January 17, 1992; age 78. Dr Habif was a 1939 graduate of Columbia University College of Physicians and Surgeons, New York City. He was a Diplomate of the American Board of Surgery. He was a Fellow of the American College of Surgeons. His memberships included the New York State Surgical Society, the American Surgical Association, the Society for Experimental Biology and Medicine, the New York County Medical Society, and the Medical Society of the State of New York.

Bernard Charles Hecht, MD, Vero Beach, FL. Died January 19, 1992; age 87. Dr Hecht was a 1929 graduate of New York University College of Medicine, New York City. His memberships included the Clinical Society, New York Diabetes Association, the Westchester County Medical Society, and the Medical Society of the State of New York.

John A. Heinlein, MD, Great Neck. Died May 14, 1992; age 84. Dr Heinlein was a 1935 graduate of State University of New York Health Science Center, New York City. He was a Diplomate of the American Board of Dermatology. His memberships included the New York State Dermatological Society, the Nassau County Medical Society, and the Medical Society of the State of New York.

Harrison Myron Karp, MD, Niskayuna. Died November 19, 1991; age 76. Dr Karp was a 1942 graduate of University of Buffalo School of Medicine, Buffalo. He was a Diplomate of the American Board of Otolaryngology. His memberships included the Schenectady County Medical Society and the Medical Society of the State of New York.

Joseph F. LaFlare, MD, Massapequa Park. Died May 24, 1992; age 73. Dr LaFlare was a 1945 graduate of the State University of New York Health Science Center, New York City. His memberships included the Nassau County Medical Society and the Medical Society of the State of New York.

Henry Leonard, MD, Kew Gardens. Died September 30, 1991; age 76. Dr Leonard was a 1944 graduate of State University of New York Health Science Center, New York City. He was a Diplomate of the American Board of Radiology and the American Board of Pediatrics. He was a Fellow of the American Academy of Pediatrics, and the American College of Radiology. His memberships included the New York Roentegen Society, the Radiological Society of North America, the Queens County Medical Society, and the Medical Society of the State of New York.

Erwin Lindenfeld, MD, North Syracuse. Died March 26, 1992; age 88. Dr Lindenfeld was a 1930 graduate of Medizinische Fakultat Der Universitat, Wien. His memberships included the Academy of Medicine, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Samuel Mortimor Peck, MD, New York City. Died December 30, 1991; age 92. Dr Peck was a 1924 graduate of Columbia University College of Physicians and Surgeons, New York City. He was a Diplomate of the American Board of Dermatology. He was a Fellow of the American College of Allergists. His memberships included the Academy of Medicine, the New York Pathological Society, the American Dermatological Association, the New York State Dermatological Society, the American Academy of Dermatology, the American Academy of Allergy, the American Society for Legal and Industrial Medicine, the American Public Health Association, the Society for Investigative Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Howard Norman Scal, MD, New York City. Died May 1, 1991; age 74. His memberships included the American Psychiatric Association, the American Psychoanalytic Association, the New York County Medical Society, and the Medical Society of the State of New York.

William B. Siegel, MD, Jewett. Died May 17, 1992; age 83. Dr Siegel was a 1939 graduate of Wayne State University College of Medicine, Detroit. His memberships included the Queens County Medical Society and the Medical Society of the State of New York.

Joseph Spanbock, MD, Fort Lee. Died March 29, 1992; age 87. Dr Spanbock was a 1932 graduate of University of Edinburgh Faculty of Medicine. His memberships included the Bronx County Medical Society and the Medical Society of the State of New York.

Irving Swartz, MD, Syracuse. Died April 30, 1992; age 88. Dr Swartz was a 1928 graduate of Boston University School of Medicine. He was a Diplomate of the American Board of Dermatology. His memberships included the Academy of Medicine, the New York State Dermatological Society, the American Academy of Dermatology, the Onondaga County Medical Society, and the Medical Society of the State of New York.

Harry Newman Taylor, MD, Kenmore. Died May 15, 1992; age 82. Dr Taylor was a 1935 graduate of the University of Buffalo School of Medicine. His memberships included the Erie County Medical Society and the Medical Society of the State of New York.

Harry Moses Throne, MD, New York City. Died March 12, 1992; age 84. Dr Throne was a 1933 graduate of St. Louis University School of Medicine. His memberships included the American Rheumatism Association, the Bronx County Medical Society, and the Medical Society of the State of New York.

Russell J. Van Coevering II, MD, Buffalo. Died May 8, 1992; age 41. Dr Van Coevering was a 1977 graduate of the State University of New York at Buffalo. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists. His memberships included the American Fertility Society, the Erie County Medical Society, and the Medical Society of the State of New York.

Robert Warner, MD, Buffalo. Died May 17, 1992; age 80. Dr Warner was a 1939 graduate of the University of Chicago School of Medicine. He was a Diplomate of the American Board of Pediatrics. He was a Fellow of the American Academy of Pediatrics. His memberships included the Academy of Medicine, the American Academy of Neurology, the Erie County Medical Society, and the Medical Society of the State of New York.

Wendy Louise Winsted, MD, New York City. Died August 21, 1991; age 45. Dr Winsted was a 1986 graduate of the University of Cincinnati College of Medicine. She was a Fellow of the American Academy of Pediatrics. Her memberships included the New York County Medical Society and the Medical Society of the State of New York.

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The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1992; 92:122–125). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

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An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

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Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

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Samples references:

1. Bowen J: Autoimmune paraneoplastic diseases of the nervous system. *NY State J Med* 1991; 91:2–3.
2. Cotran RS, Kumar V, Robbins SL: *Robbins Pathologic Basis of Disease,* ed 4. Philadelphia, WB Saunders Co, 1989, pp 842–845.

REVIEW

All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

Address correspondence to Pascal James Imperato, MD, Editor, *New York State Journal of Medicine,* PO Box 5404, 420 Lakeville Road, Lake Success, NY 11042.

(*continued from p 3A*)

Nov 19–21. **Aesthetic and Reconstructive Surgery of the Aging Face.** Waldorf Astoria Hotel. Contact: Francine Leinhardt, Course Coordinator, Manhattan Eye, Ear & Throat Hospital, 210 E 64th St, New York, NY 10021. Tel: (212) 831-9200.

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CALIFORNIA

Nov 5–7. **The UCSF Otolaryngology Update: 1992.** Ritz-Carlton Hotel, San Francisco. *Nov 13–15.* **39th Annual Group Therapy Symposium.** Fisherman's Wharf. San Francisco. Contact: Extended Programs in Medical Education, Room LS-105, University of California, San Francisco, CA 94143-0742. Tel: (415) 476-4251.

Nov 9–13. **A Practical Approach to Breast Imaging.** Ritz-Carlton, Rancho Mirage. Contact: Radiology Postgraduate Education, UCSF School of Medicine, Box 0628, Rm C-324, San Francisco, CA 94143-0628. Tel: (415) 476-5731.

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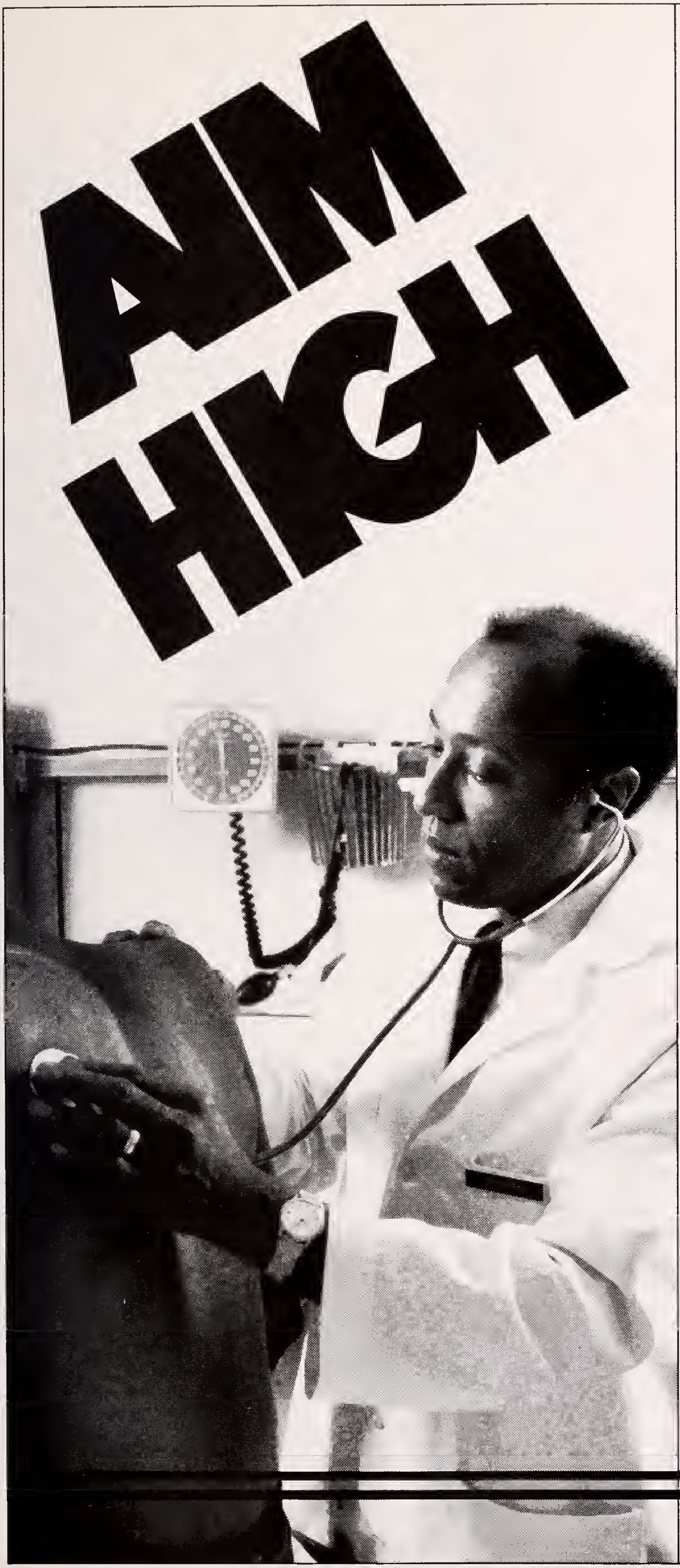

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NO 11
NOVEMBER
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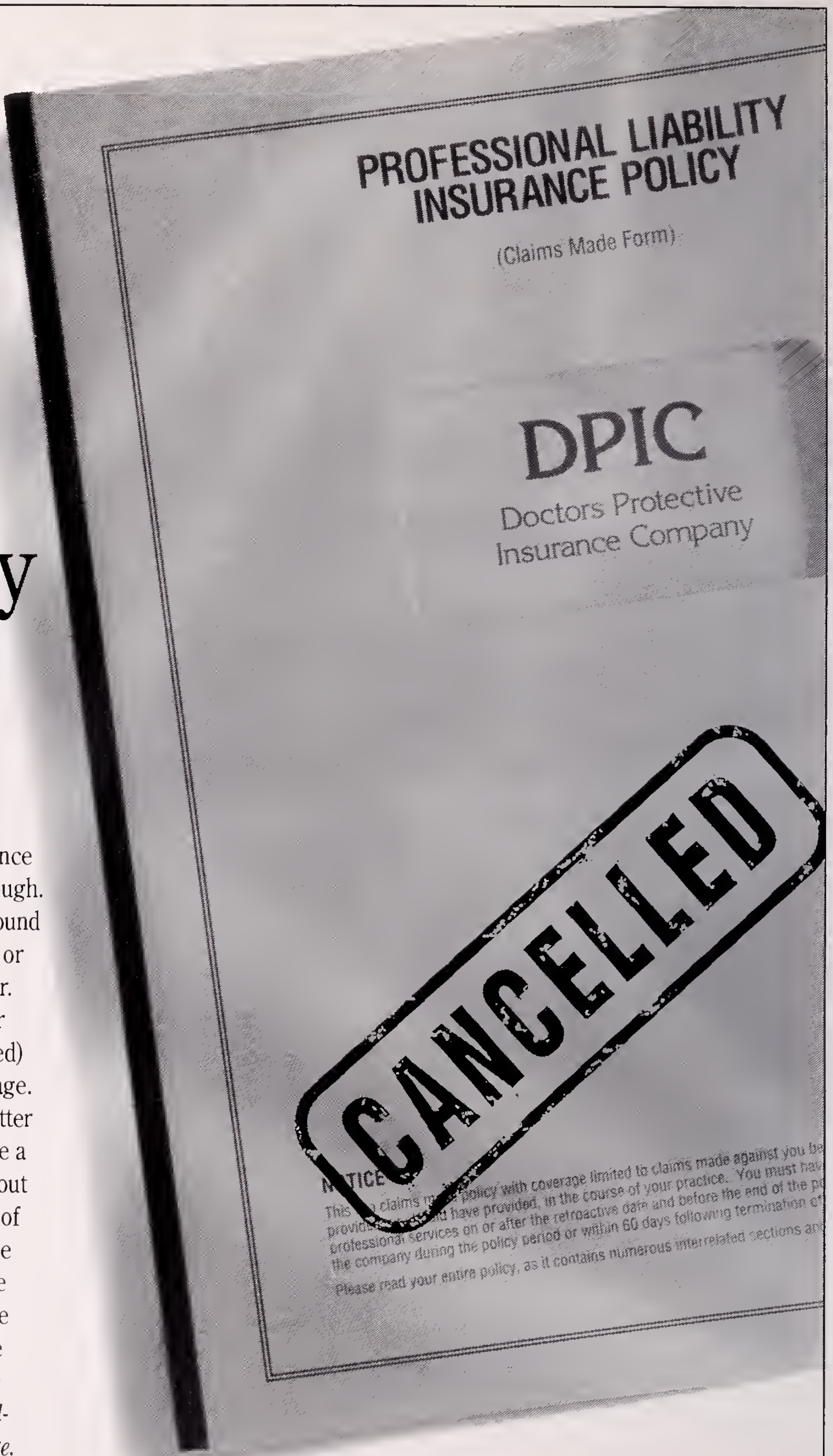
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New York State Journal of Medicine

NOVEMBER 1992 / Volume 92, Number 11

Contents

COMMENTARIES

461 **Health and humanitarian assistance in conflicts and disasters**
PASCAL JAMES IMPERATO, MD

462 **The hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis**
WALLY GORDON, MD

463 **Diseases associated with monoclonal immunoglobulins**
MEHDI FARHANGI, MD

RESEARCH PAPERS

465 **Access to health care in rural western New York State**
CHERYL A. LUCAS, MS, THOMAS C. ROSENTHAL, MD

469 **Health and economic impact of cigarette smoking in New York State, 1987–1989**
K. MICHAEL CUMMINGS, PhD; JEFFREY STILES; MARTIN C. MAHONEY, PhD; RUSSELL SCIANDRA, MS

MEDICAL CONFERENCE

474 **Widespread complications of lupus erythematosus**

HISTORY

480 **Smallpox in an 18th-century Italian princess**
SAUL JARCHO, MD

COMMITTEE REPORT

485 **Medical futility**
FRED ROSNER, MD; PIETER R. KARK, MD; ALLEN J. BENNETT, MD; ANTHONY BUSCAGLIA, MD; ERIC J. CASSELL, MD; PETER B. FARNSWORTH, MD; ABRAHAM L. HALPERN, MD; JOHN B. HENRY, MD; ALLISON B. LANDOLT, MD; LAURENCE LOEB, MD; ROBERT LOWENSTEIN, MD, PhD; PATRICIA J. NUMANN, MD; FERNANDO V. ONA, MD; HERMAN M. RISEMBERG, MD; PETER ROGATZ, MD; PHILIP H. SECHZER, MD; PETER P. SORDILLO, MD; PhD; JONATHAN R. WOLPAW, MD

(continued on p 6A)

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SPECIAL ARTICLES

489 **The Patient Self-Determination Act and patient-physician collaboration in New York State**
JOSEPH J. FINS, MD

494 **Total body photography for melanoma surveillance**
WILLIAM E. SLUE, JR

CASE REPORTS

496 **Hyperlipidemia in monoclonal gammopathy**
ZAEEM ANSARI, MD

497 **Primary sarcomas of the breast in women under 20 years of age**
ALBERTA R. LUNA VEGA, MD; JOHN T. VETTO, MD; DAVID W. KINNE, MD

499 **Morgagni hernia mimicking a lipomatous tumor**
PERRY S. GERARD, MD; ERIC WILCK, MD; ELLIOT SENDEROFF, MD; STUART GOLBEY, MD

500 **Transitional cell carcinoma of the renal pelvis in an incompletely duplicating collecting system**
DANILO ASASE, MD; RONALD G. FRANK, MD; PERRY S. GERARD, MD; KRIS LINDSAY, MD; GILBERT J. WISE, MD

LETTERS TO THE EDITOR

502 **Proper obstetric care for women in New York State**
DON SLOAN, MD, RICHARD H. SCHWARZ, MD

502 **Cigarette advertising in news magazines**
PETER S. LIEBERT, MD

503 **Idiopathic segmental infarction of the greater omentum**
MAZEN A. ABDALLA, MD; THAYYULLATHIL BHARATHAN, MD; GERALD POSNER, MD

503 **Mucinous cystic neoplasm of the pancreas**
VELLORE S. PARITHIVEL, MD; VIRENDRA A. PARIKH, MD; VIBHU R. KSHETTRY, MD; PAUL H. GERST, MD; SOVI JOSEPH, MD

505 BOOK REVIEWS

509 BOOKS RECEIVED

510 OBITUARY LISTINGS

511 IN MEMORIAM

512 AUTHOR GUIDELINES

9A MEDICAL MEETINGS AND LECTURES

THE NEWLY APPROVED MEDICAL ASSOCIATION TRUSTS
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Dec 1. **Antibiotic Therapy.** *Dec 8.* **Prenatal Diagnosis and Karyotype.** *Dec 15.* **Fluid and Electrolytes.** Cat I Credits. Schneider Children's Hospital Grand Rounds at Huntington Hospital. Tuesdays, 8:30–9:30 AM. Contact: Arthur J. Schwager, MD, 164 E Main Street, Huntington, NY 11743. Tel: (516) 427-0630.

Dec 22. **Pulmonary Hypertension in the Newborn: New Modalities of Treatment.** 8:30–9:30 AM at Huntington Hospital. Cat I Credits. Contact: Arthur J. Schwager, MD, Director, Continuing Medical Pediatric Education, SUNY at Stony Brook, Huntington Hospital, Huntington, NY 11743. Tel: (516) 427-0530.

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Dec 4–5. **Cochlear Implantation: Principles and Techniques.** 12.25 Cat I Credits. NYU Medical Center. *Dec 7–10.* **Neurosurgery NYC 1992.** 24 Cat I Credits. Grand Hyatt Hotel. *Dec 9–11.* **Managing Clinical Problems in the Elderly.** 19 Cat I Credits. NYU Medical Center. *Dec 14–19.* **11th Annual CT/MRI Head to Toe.** Grand Hyatt. *Dec 18–20.* **Interventional Laparoscopy and Stapling Techniques in Urology.** Contact: NYU Medical Center Post-Graduate Medical School, 550 Fifth Avenue, New York, NY 10016.

Dec 4–5. **HIV, AIDS, and the Brain. 72nd Annual Meeting of the Association for Research in Nervous and Mental Disease.** Holiday Inn Crowne Plaza Hotel. Contact: Association for Research in Nervous and Mental Disease, Inc., Mt. Sinai School of Medicine, 1 Gustave Levy Place, Box 1052, New York, NY 10029. Tel: (212) 348-8133.

Dec 4–7. **25th Annual New York Cardiovascular Symposium.** 21.5 Cat I Credits. New York Hilton and Towers. Contact: Registration Secretary, Extramural Programs Dept, American College of Cardi-

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical Letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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New York State
Journal of Medicine

COMMENTARIES

Health and humanitarian assistance in conflicts and disasters

Two major armed conflicts and one natural disaster have recently heightened public awareness that political and logistical determinants often shape the timing and character of humanitarian assistance. The dimensions of the disaster caused by Hurricane Andrew in southern Florida and Louisiana were apparent within hours after the storm had passed. Yet, large-scale Federal government humanitarian assistance was slow in coming and initially inadequate in scope until the United States Army was mobilized, as much by political concerns as by humanitarian ones.

A civil war has been decimating Somalia since early 1991. It has resulted in the almost total physical ruin of towns and cities, a complete breakdown of government, the destruction of agriculture, famine, and hundreds of thousands of refugees. This unprecedented level of devastation in Somalia has been fueled by clan rivalries and an enormous armory of modern weapons. The latter are the legacy of cold war politics in which Somalia's shifting alliances gave it access to arms, first from the former Soviet Union and later from the United States.

By mid-1992, the civil war driven famine in Somalia had become so severe that thousands were dying from starvation every day. Food became a source of power to rival clans that resorted to extreme violence in order to hijack humanitarian shipments from foreign donors.

The civil war and famine in Somalia received scant attention in Europe and the United States until mid-1992 when the situation had grown desperate. Even then, the provision of assistance by the United States was minimal and driven by political considerations. The first plane-loads of American food arrived in a safe area of Somalia at the time of the Republican National Convention as President George Bush came under increasing criticism for doing too little.

There are many parallels between the civil war in Somalia and that in former Yugoslavia. The provision of humanitarian aid to the people of Bosnia-Herzegovina is rendered difficult because of the ongoing conflict and the great risks to relief workers. Unlike in Somalia, however, United Nations forces established control of the Sarajevo airport so as to permit the delivery of relief supplies. That the same effort was not made in Mogadishu, Somalia's capital, has understandably led to accusations of preferential treatment of a European crisis. These differing responses have their roots in complex historical, security, and political issues. They demonstrate the powerful character of these issues in shaping both the timing and character of humanitarian assistance. Somalia is in the Horn of Africa, very distant from the heart of Europe where the conflict in Bosnia-Herzegovina has immediate political, security, and refugee implications for adjacent countries which are usually donors in times of disaster. Unlike the Somalis, the peoples of former Yugoslavia benefit from the lobbying efforts of well-organized political action groups in both Europe and the United States, primarily established by immigrants.

From another perspective, protracted civil wars and famines in the Sudan, Mozambique, and Ethiopia during the 1980s created what many relief organizations term "compassion fatigue." According to this view, the people of donor countries have become wearied by disasters created by what often strikes them as senseless human conflicts. They are less inclined than before either to rapid responses or to sustained ones because they perceive the underlying causes as defying solutions. They see the provision of humanitarian assistance as not only helping the victims of these conflicts, but also those responsible for them. Defining humanitarian conflict relief in terms of only the civilian victims, usually children, women, and the elderly, is a difficult task in such circumstances. However, it is one that is crucial for mustering broad public support.

In spite of these problems, there is ample evidence that policy initiatives that foster health and humanitarian assistance can not only heal the wounds of war, but

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The opinions expressed in this editorial are those of the author alone and do not constitute an expression of official opinion of the Medical Society of the State of New York.

also help promote peaceful solutions to what at first appear to be intractable conflicts. This was the general theme at a recent symposium in New York City entitled *A Framework for Survival: Health, Human Rights, and Humanitarian Assistance in Conflicts and Disasters.* This two-day symposium was co-sponsored by the Center for International Health and Cooperation and the Council on Foreign Relations. Over 20 distinguished speakers addressed a broad range of issues dealing with providing humanitarian assistance to civilian populations affected by war and natural disasters. Although the emphasis was on medical assistance; legal, operational, economic, and foreign policy questions were also covered in depth. The opening remarks were delivered by Peter Tarnoff of the Council on Foreign Relations and Kevin M. Cahill, MD, President and Director of the Center for International Health and Cooperation. Former United States Secretary of State, Cyrus R. Vance, now a Special Envoy of the United Nations who is co-chairing the peace conference on the conflict in former Yugoslavia, set the tone for the symposium with his address "Health in the New World Order."

This symposium assembled some of the most distinguished and experienced experts in all fields related to humanitarian assistance. Among them were Lord David Owen, who is co-chairing the peace conference on the conflict in former Yugoslavia, and Kofi Awoonor, Ambassador of Ghana to the United Nations, who expressed the concerns of recipient nations. Also in attendence were diplomats, academicians, representatives of the International Red Cross, CARE, CONCERN, the Centers For Disease Control, and Medécins Sans Frontières, the Director of the United Nations Childrens Fund, the United Nations High Commissioner for Refugees, and others. The assembling of so many distinguished speakers from a broad array of organizations and institutions concerned with disaster relief not only provided for a sharing of expertise but more importantly spoke of a strong commitment to helping the victims of conflicts and disasters. The symposium brought forth humanity's nobler instincts of helping the victims of war no matter the cause and to be there when disaster strikes.

The magnitude of the health and other problems caused by disasters and conflicts is often so great that only donor governments and the international organizations they support can effectively intervene. It is an inescapable fact that the rapidity and scope of responses are often shaped by both foreign and domestic policies. However, these policies in the United States and western Europe are sensitive to both the will of the people and political reassessments as evidenced by recent relief efforts. Although the Federal government's reaction to the disaster created by Hurricane Andrew was slow, it eventually intensified in speed and magnitude in response to both political and public pressures. Even though voluntary, church and other groups were quick to respond, their means were inadequate to meet the enormous needs, again underscoring the essential role of government in such massive relief efforts.

It is easy to lose heart in the face of what appear to be intractable conflicts and overwhelming disasters. However, human beings have an enormous ability to renew and rebuild, given a few resources and some hope. Health and humanitarian assistance not only rebuild broken bodies and heal wounds. They also heal the spirit and provide people with hope for the future.

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The hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis

Large outbreaks of drug-resistant tuberculosis have been reported in Florida and New York,[1] and a survey by the Centers for Disease Control for the first quarter of 1991 found *Mycobacterium tuberculosis* isolates resistant to isoniazid and rifampin in 11 other states.[2] The primary cause of resistance to drugs is probably the failure of patients to complete the course of treatment prescribed. This results in the selection of drug-resistant organisms. About 90% of cases of drug-resistant tuberculosis have occurred in persons infected with human immunodeficiency virus (HIV). Drug-resistant tuberculosis among HIV-infected patients has been reported also from outside the United States,[3] and global spread of drug-resistant tuberculosis is a real source of concern.

The treatment of drug-resistant tuberculosis may require a return to pre-antibiotic programs. If cavitation develops in a tuberculous lung, thus changing a relatively benign disease into a potentially fatal one, collapse or removal of the cavitated area is indicated. Most modern, young thoracic surgeons have little or no experience of presently outdated collapse techniques, and few older surgeons remain to teach them, if this training becomes necessary. Therefore, it seems appropriate to inform those who will be responsible in the future, but who may be unaware of the problem because of a lack of exposure. Perhaps, they will learn to perform induction of artificial pneumothorax and thoracoplasty from books and articles. If so, they should know that failure to close a cavity and the serious complications of both collapse therapy and pulmonary excision are likely to be due to the presence of bronchial tuberculosis.

Tuberculosis of major bronchi is very common in patients with pulmonary tuberculosis. The British Tuberculosis Association[4] reported that of 94 operation specimens, histological evidence of tuberculosis was found at the point of bronchial transection in 95%. Gordon found this evidence in 87% of 31 specimens.[5] He also reported that bronchoscopy revealed narrowing of the bronchial lumen by swollen and often red tuberculous mucosa in 51 of 100 hospital patients with pulmonary tuberculosis.[6] Severe cough, more than 60 mL of sputum daily, wheeze, diminished air entry, rhonchi, collapse of lung tissue, and a cavity with a fluid level appear to be reliable guides to the presence of tuberculosis of the larger bronchi. This lesion was seen in 71% of patients with any one of these clinical features.[6]

In the presence of an artificial pneumothorax, the narrowing of a bronchus by swollen mucosa may cause a cavity to persist and perhaps even rupture. After thoracoplasty, the bronchial lesion appears to favor collapse of the lower lobe and spread of disease.[6] Even when bronchoscopy is normal, the microscopic lesion is an important factor in the development of bronchopleural fistula after pulmonary excision for tuberculosis.[5] The incidence of gross and microscopical lesions and postoperative complications are reduced significantly by antibiotic treatment before thoracoplasty and pulmonary excision.[5,6,7] This benefit is denied patients with multidrug-resistant tuberculosis.

Reports of pulmonary excision in patients with HIV infection have not appeared so far. This operation may be impractical in patients with HIV infection, and facilities for pulmonary excision may not be available in some less developed countries. Pneumothorax or thoracoplasty may be effective alternative treatments. If bronchoscopy performed on a patient with a drug-resistant infection does not show evidence of bronchial tuberculosis, there is no contraindication to collapse therapy.

Since tuberculous lesions are very common in the central end of the divided bronchus of specimens removed from patients who receive little or no preoperative antibiotic therapy, it is very likely that these lesions will be present in the bronchi of patients with drug-resistant infection. Should such patients undergo excision of a lung or all or part of an upper lobe, a postoperative fistula could be expected in 50% of cases. This is the incidence found in patients who were not treated with antibiotics.[5] This high incidence was observed in 1954, and it is not certain that the same expectation is justified when applied to the relatively recent problem of drug-resistant tuberculosis. Only one relevant report, that by Pomerantz et al, has appeared.[8] These investigators applied a muscle flap to the site of bronchial closure after pneumonectomy or lobectomy in 14 patients without HIV infection but with sputum positive for drug-resistant *M tuberculosis*. Only one fistula developed. However, all these patients had received antibiotics for at least three months and the drugs may have had some beneficial effect on any bronchial lesion. The view that such an effect occurred is supported by the report of Pomerantz et al that preoperative bronchoscopy was normal in all but one patient with a non-specific deformity.

If bronchoscopy reveals swollen, red bronchial mucosa, the surgeon faces a difficult choice—to run the risk of serious postoperative complications or else the risk of extension of disease if conservative management is continued. No information has been published regarding the results of excisions performed on patients with multidrug-resistant tuberculosis when bronchoscopy is abnormal. If bronchial lesions are visible, pulmonary excision, in spite of its complications, may be the only alternative to medical therapy.

Since the incidence of tuberculosis is increasing, especially among patients with the acquired immunodeficiency syndrome, which itself is spreading, an increase is likely in the demand for the management of disease resistant to drug therapy. Surgeons should become aware that treatment may be required under circumstances which they have not experienced. They should become familiar with the surgical options available to the unfortunate patient with a pulmonary infection resistant to antibiotics.

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1. Nosocomial transmission of multidrug-resistant tuberculosis among HIV-infected persons. Florida and New York, 1988–1991. *MMWR* 1991; 40:585–591.
2. Snider DE Jr, Roper W: The new tuberculosis. *N Engl J Med* 1992; 326: 703–705.
3. Monno L, Angarano G, Carbonara S, et al: Emergence of drug-resistant Mycobacterium tuberculosis in HIV-infected patients. *Lancet* 1991; 337:852.
4. British Tuberculosis Association: Report by research committee on tuberculous tracheobronchitis. *Tubercle* 1953; 34:74–80.
5. Gordon W: The effect of preoperative streptomycin on the incidence of bronchopleural fistula after pulmonary excision for tuberculosis. *J Thorac Surg* 1954; 28:1–3.
6. Gordon W: Bronchial tuberculosis. *Brit J Tuberc Dis Chest* 1955; 49:115–118.
7. Gordon W: The effect of preoperative chemotherapy on the incidence of tuberculous lesions in the bronchi. *J Thorac Surg* 1957; 33:399–400.
8. Pomerantz M, Madsen L, Goble M, et al: Surgical management of resistant mycobacterial tuberculosis and other mycobacterial pulmonary infections. *Ann Th Surg* 1991; 52:1108–1112.

Diseases associated with monoclonal immunoglobulins

Monoclonal immunoglobulins (MIgs), also known as myeloma proteins, paraproteins, and M-components are the products of expanded and terminally differentiated B cell clones. They occur in malignancies (multiple myeloma, Waldenström macroglobulinemia, heavy chain diseases) and benign or apparently benign conditions

TABLE I. Clinical Syndromes Caused by the Antibody Activity of MIgs

Clinical Syndrome	MIg	Antigen	B-cell Disorder*
Cold agglutinen-associated hemolysis	IgM IgG IgA	Red-cell surface antigens	CCHD WM MM MGUS CLL NHL
Type II cryoglobulinemia	IgM IgG IgA	IgG	WM MM MGUS
Vasculites	IgG	Albumin	MGUS
Bleeding disorder	IgG	Factor VIII-C Factor VIII-R	NHL MM
Polyneuropathy	IgM IgG	Myelin-associated Glycoprotein chondroitin sulfate	MGUS MM WM
Xanthoderma	IgG	Riboflavin	MM
Xanthoma and hyperlipidemia	IgA IgG IgM	APo B APo E Lipoproteins Heparin	MGUS MM
Hemochromatosis	IgG	Transferin	MGUS
Hypoglycemia	IgG	Insulin	MM MGUS
Hypothyroidism	IgM	Thyroxine	WM

*CCHD: Chronic cold hemagglutinin disease; CLL: Chronic lymphocytic leukemia; MGUS: Monoclonal gammopathy of unknown significance; MM: Multiple myeloma; NHL: Non-Hodgkins lymphoma; WM: Waldenström macroglobulinemia

(monoclonal gammopathy of unknown significance). The presence of large quantities of such proteins in vascular and extra-vascular compartments is not without consequences. Aside from distinct clinical syndromes, derived from the physico-chemical properties of MIgs (eg, the hyper-viscosity syndrome), uncommonly, the antibody function of these proteins have been found to cause a variety of fascinating and intriguing clinical situations. Yellow coloration of the skin and the hair in an elderly woman with multiple myeloma led us to the discovery of a riboflavin-binding MIg.[1] The syndrome of chronic cold hemagglutinin disease (CCHD) is almost entirely attributable to the affinity of certain MIgs for the red-cell surface antigens. The rheumatoid factor activity of some M-components causes immune complex formation, and type II cryoglobulinemia with dermal, articular, and renal morbidity. Recently, MIgs with insulin binding property have been reported as causing hypoglycemia.[2] Table I summarizes an incomplete list of clinical syndromes that are closely related to the antibody activity of such M-components.

In this issue of the *Journal*, Ansari[3] describes a patient whose diagnosis of multiple myeloma was made in the course of pursuing an incidental finding of hyperlipidemia. Although the binding of paraprotein to the lipid moiety was not demonstrated in vitro, the investigator is quite justified in postulating that such a biologic property of MIg may have in fact been responsible for the hyperlipidemia. Most lipid-binding MIgs are IgAs and IgGs, and only rarely are IgMs.[4] The binding site, lipid structure or apoproteins, have been elucidated in few instances. The resultant immune complex formation leads to a retardation of lipid catabolism. Consequently, hyperlipidemia with or without xanthoma may ensue. As noted by Ansari, hyperlipidemia caused by heparin-binding myeloma proteins has also been reported.[3]

A good number of MIgs with identified antibody activity are autoantibodies. These antibodies tend to be polyreactive (binding to several diverse ligands) with a low-affinity constant and at times exhibit a shared idiotype. Thus, in some ways, they resemble naturally occurring autoantibodies.[5] Are MIgs the product of specific antigenic stimulation (antigen driven) or simply the product of random clonal expansion? The answer is not known. The polyreactivity and shared idiotype reported among several MIgs with rheumatoid factor activity[4] tend to support the latter. On the other hand, some MIgs have been shown to be monospecific with high affinity for their ligand, suggesting an antigen driven mechanism. In this regard, it is worth noting that some mouse myeloma proteins with antibody activity react against antigens of microbial origin which might have served as antigenic stimuli.[6] Regardless of the pathophysiologic mechanism involved, the spectrum of clinical conditions in which MIgs are found to be the culprit continues to expand. It is hoped that the physician's familiarity with them may help to unravel some although rare, challenging diagnostic and clinical dilemmas.

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1. Farhangi M, Osserman EF: Myeloma with xanthoderma due to an IgGλ monoclonal antiflavin antibody. *New Eng J Med* 1976; 294:177–183.
2. Redmon B, Pyzdrowski KL, Elson MK, et al: Hypoglycemia due to a monoclonal insulin binding antibody in multiple myeloma. *New Eng J Med* 1992; 326:994–998.
3. Ansari A: Hyperlipidemia in monoclonal gammopathy. *NY State J Med* 1992; 11:496–497.
4. Merlini G, Farhangi M, Osserman EF: Monoclonal immunoglobulins with antibody activity in myeloma, macroglobulinemia and related plasma cell dyscrasias. *Semin Oncol* 1986; 13:350–365.
5. Viard JP, Bach JF: Clonality in autoimmune diseases. *Semin Hematol* 1991; 28:57–65.
6. Potter M: Antigen binding M-components in Man and Mouse, in Azar HA, Potter M (eds): *Multiple Myeloma and Related Disorders*, vol 1, Harper and Row Publishers, New York, 1973, pp 195–246.
7. Trimarchi F, Benvenga S, Fenzi G, et al: Immunoglobulin binding of thyroid hormones in a case of Woldenström's macroglobulinemia. *J Clin Endocrinol Metab* 1982; 54:1045–1050.

Access to health care in rural western New York State

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ABSTRACT. Persons living in rural counties often have limited access to health care. From a random sample, 1,332 households in six rural western New York counties were interviewed by telephone to identify what health care facilities are used when seeking professional health care what factors affect the preference for a health care facility and whether individuals perceive a shortage of health services in their communities. Seventy-nine percent of rural western New York individuals surveyed visited a physician between June 1988 and January 1990. The majority (83%) of individuals visited their regular health care source, which in most cases (81%) was a physician's private office. Factors related to a preference for some other health care source for the last visit included having traveled 20 or more miles to the visit ($p < 0.01$) and lack of overall satisfaction with the last visit ($p < 0.001$). Factors that varied by county of residency and were significantly associated with satisfaction included the type of health source used ($p < 0.001$), and the distance traveled to the last health care visit ($p < 0.01$). The majority of the respondents did not perceive a shortage of health care services in their communities probably because they have a lower expectation of health care accessibility.

(NY State J Med 1992; 92:465–469)

Population characteristics such as minority race and ethnic group may limit health care access; whereas, increases in education level, age, and income have been associated with increased access to health care.[1–5] Geographic proximity to a health care provider and satisfaction with a provider have also been found to improve access to health care.[6–8] All these factors are greatly influenced by an individual's proclivity to seek care.[9] It is not well understood how residence in a rural region, such as western New York State, affects access to health care.

Many factors linked to health care access differ across the nation between Standard Metropolitan Statistical Areas (SMSAs), cities, and rural areas (non-SMSAs). There is a higher proportion of elderly persons and of patients with poor health status in rural counties; and the economic decline of agribusiness during the 1980s further strained the resources of these regions.[6,10] More than 40 rural New York State hospitals closed during the past decade, as many of the characteristics associated with them (ie, small size, low occupancy rate, small medical staff, and high dependence on Medicare), challenged their economic viability.[6,11,12] In western New York alone, two of 16 rural hospitals closed in 1988.[13,14]

A regional survey of residents in six rural western New York counties was undertaken to assess the perception of health care access. The purpose of the survey was to determine the type of facilities that rural individuals choose when they need professional health care define factors that affect the preference for a health care facility, and to determine health care needs as perceived by rural community members. The authors hypothesized that a significant number of rural residents would not have a regular health care source, and that when they sought professional care they would rely on hospital-related facilities, would need urban hospitals for a portion of their health care and would perceive a shortage of health care resources in their communities.

METHODS

The six rural counties included Genesee, Orleans, Cattaraugus, Chautauqua, Wyoming, and Allegany. These rural counties have a total population of approximately 422,000 and a density of 150 persons or less per square mile.[15] We obtained a random sample of 3,300 household telephone numbers from an independent market research agency (Survey Sampling Inc, Connecticut). The sample was selected using the random-digit-dialing probability sampling method and was stratified proportional to the population of each county.[16] Interviews were obtained by telephone using a modified version of the 1982 National Access Survey, with approval of the State University of New York-Buffalo Human Subjects Committee.[5] The telephone surveyors were college students hired to work on a full-time basis during winter break. An instructor from the US Bureau of the Census conducted a six hour training session including didactic instruction, role playing, and supervision of actual phone interviews. Telephone interviews were conducted during daytime, evening, and weekend hours in January of 1990.

An initial screening question eliminated individuals who had not lived in the area during the entire 18 months before January 1990. Only individuals aged 18 years or older were interviewed. The individual whose health care was discussed in the survey was termed the "index" individual. Each interviewer alternated the gender and age of the index individual by rotating requests for information about an adult male, an adult female, a male child, or a female child.[16] This ensured that all age and gender groups were represented in the sample at the expense of a truly random collection of index individuals. Adult index individuals provided information about themselves. Information on children was obtained by interviewing the household adult most likely to know about the child's health care. In single households or households without children, an adult was interviewed and information on a child was sought on the next call.

Questions focused on household and individual demographic information, the index individual's use of specific health facilities (hospital, clinic), factors that affected this utilization (travel time, illness, finances) and satisfaction with general aspects of health care (quality of care, access to

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TABLE I. Comparison of the Proportion of Completed Interviews with the Proportion of Households in Original Sample

County	Total Completed Interviews	Proportion of Total	Total Households in Sample	Proportion of Households
Genessee	176	13.2	443	13.4
Orleans	141	10.6	311	9.4
Cattaraugus	310	23.3	753	22.8
Chautauqua	450	33.8	1156	35.0
Allegany	153	11.5	383	11.6
Wyoming	102	7.7	254	7.7
Total	1,332	100.0	3,300	100.0

providers). Questions about travel mode, travel time to the health facility, personal preference for a facility, health insurance coverage, and information about illness and quality of care for the most recent visit were asked. Information concerning dental and medical health care was also elicited. The survey ended with questions about perceived unmet needs for health care services. Individuals were told that they could stop the interview at any time and that responses were confidential.

Analysis was done using the Statistical Package for the Social Sciences (SPSSX) mainframe version 3.0.[17] In addition to determining frequencies and mean values for each variable, χ^2 analysis was used to determine statistical association between variables.

RESULTS

Completed responses were obtained from 1,332 households for an adjusted overall response rate of 41%. The response rate was adjusted by the proportion of disconnected numbers, wrong numbers, business numbers, and out of western New York numbers found in the sample. Interviewers were able to contact 1,968 households. Five-hundred and sixty refused to complete the interview, 59 hung up, and none could not be interviewed due to language barriers. Therefore, of this 1,968, 1,332 responses represent 68% of the telephone numbers actually contacted. The county distribution of the completed responses were proportioned to the populations of the six target western New York rural counties (Table I).

The rural households were inhabited mainly by nuclear families (1,052 or 79%), or single residents (229 or 27%). Most heads of household were employed full time (750 or 59%), whereas 383 (30%) were retired, 54 (4%) were employed part-time, and 79 (6%) were temporarily out of work. The majority of household heads (687 or 57%) held blue collar employment. The mean 1988 household income was between $20,000 and $24,999.

Using 1980 US Census data, the number of adult females (42%) is over-represented by 7% in the responding households, adult males (22%) are under-represented by 9%, and the sample of children is representative of the population (Table II). The large majority of the index individuals identified themselves as white (1,271 or 97%), and the mean age was 39.5 year (SD = 25.9).[15] The majority (94%) of the individuals were born in New York State.

Household Health Care Source. Most individuals (1,162 or 87%) we were able to identify one source of health care for the household. Reasons given for not having a regular health care source for the entire household were that household members did not deem it important it was difficult finding a doctor or hospital, lack of money, their regular doctor retired, or that they have no need for a doctor.

Between June 1988 and January 1990, 1,046 (79%) of the individuals visited a physician, and of those, 863 (83%) reported that they went to their regular source of health care. During the same period, 134 (10%) individuals had been admitted to a hospital for at least one night. Only 19 persons (14% of admissions) sought treatment at a facility not located in one of the rural counties (Table III).

One hundred and eighty-two (18%) individuals who sought health care in the previous 18 months defined their health care visits as emergencies. Of the individuals who had an emergency visit, 65 (36%) had an emergency involving a new illness, and 84 (46%) involving a new injury; 29 (16%) had an emergency relating to an existing condition; and 1 (1%) for a pregnancy. Emergency visits for infants and children (53%) exceeded that proportion expected by the population ratio (Cramer's $V = 0.16, p < 0.001$).[17]

Eighty-one percent (837) of individuals reported visiting a physician's private office for the last health care visit, 106 (10%) visited a hospital, 75 (75%) went to a clinic, 9 (1%) were not specific in their answer, and the rest visited either a medical group, a school, or company health facility, or a doctor came to their home.

Between June 1988 and January 1990, the majority of index individuals (886 or 65%) saw a dentist for a check-up. Only fifty-three (4%) of responders reported visiting a psychiatrist or a mental health specialist during the 18-month period.

Barriers. Thirty-six (4%) individuals had some difficulty communicating with the caregiver at their last visit. In 15 (45%) of these cases, the communication problem was due to the individual's own inability to speak fluent English, and in 18 cases (55%) individuals reported that the physician did not speak fluent English. Fourteen percent (146) of the individuals traveled more than 20 miles to their last health care visit, but

TABLE II. Demographic Characteristics of Index Individuals

	Age (yr)				
	(18+) Male	(18+) Female	(<18) Proxy Male Child	(<18) Proxy Female Child	Total
Gender (N = 1,332)	300 (22%)	558 (42%)	252 (19%)	222 (17%)	1,332
Race (N = 1,332)					
White	285 (97%)	531 (98%)	242 (97%)	213 (96%)	1,271
Age (yr, N = 1,332)					
<19	0	0	252 (100%)	222 (100%)	474 (36%)
9–34	79 (26%)	74 (13%)	0	0	153 (11%)
35–64	116 (39%)	225 (40%)	0	0	341 (26%)
65+	105 (35%)	259 (46%)	0	0	364 (27%)
	300 (100%)	558 (100%)	252 (100%)	222 (100%)	1,332 (100%)
Mean age (yr)	39.5 (SD, 25.9)				

TABLE III. Type of Health Care Visit Made by Index Individuals (June 1988–January 1990)

Factor	Yes	%	No	%	Not Applicable*	Total
Had regular health care source	951	95	54	5	327	1,332
Visited a physician	1046	79	277	21	9	1,332
Visited regular source	863	83	132	13	51	1,046
Last visit an emergency	182	18	854	82	296	1,332
Overnight hospital stay	134	13	912	87	286	1,332
Went to a rural hospital	115	86	19	14	—	134

*Not asked because individual had not visited a physician during June 1988–January 1989.

only 31 individuals (3%) reported that transportation was a problem.

Only 76 individuals (7%) had Medicaid coverage for some part of their outpatient visit, 220 individuals (21%) had Medicare, and 49 individuals (5%) reported that a portion of their last visit was covered by another type of government insurance. Seventy-two percent (736) of the individuals or their family members paid for at least a portion of the cost of the last visit. Forty-nine percent (512) stated that their insurance was an employer-provided benefit.

Seventy-eight percent (812) of individuals were completely satisfied with their last health care visit. Twenty percent (205) were somewhat satisfied, and 2% (22) were not satisfied. Seven percent (70) of the individuals reported that they would have preferred to go to another health care source. Of those willing to give a reason why they would have preferred another provider, 32% (15) would have preferred a closer facility. Only 2% (16) of the individuals actually sought care at another site.

No statistically significant associations were found between communication barriers, travel problems, or third party coverage and the preference for a health care facility. However, a significant association was identified between preference for another facility and the distance traveled to the last visit. Of those individuals who traveled 20 miles or less to their last visit, 47 (5%) individuals would have preferred to go to another source, and 26 (36%) of those who traveled more than 20 miles or more to their last visit would have preferred to have gone elsewhere (Cramer's $V = 0.17, p < 0.001$).

Individual preference was also associated with an overall satisfaction with the first visit. Forty-one (57%) of those who reported preference for another source reported not being completely satisfied with their last visit for health care, and 175 (18%) of those who did not prefer another source reported not being completely satisfied with their last visit (Cramer's $V = 0.60, p < 0.001$).

County Comparisons. There was a statistically significant association between the health care source that individuals went to for their last physician visit and their county of residence (Table IV). Orleans county had the highest proportion of residents who visited a physician's office last (94%, Cramer's $V = 0.11, p < 0.01$). Eighty-one percent or more of the individuals from Genesee and Chautauqua visited a physician's private office for their last visit, whereas the remainder visited hospitals or clinics. In comparison, between 75% and 79% of individuals in Cattaraugus, Wyoming, and Allegany counties visited a physician's office, whereas the remainder visited hospitals or clinics. Cattaraugus County had the highest proportion of individuals (13%) who reported visiting a clinic. Another significant association was found between the county of residence and the distance that individuals traveled to the last visit. Between 19% and 22% of the individuals from Genesee, Allegany, and Wyoming counties traveled more than 20 miles to their last physician visit, while between 10% and 13% of the responders from Cattaraugus, Chautauqua, and Orleans counties traveled more than 20 miles to the last visit (Cramer's $V = 0.12, p < 0.01$). This association is illustrated in Table V.

When individuals were asked whether they felt there was a health care shortage in their rural community, 716 (54%) individuals reported that there was no shortage of health care services in their communities. Specific needs that were mentioned included physicians and hospitals.

DISCUSSION

Sample selection, telephone methodology, and retrospective recall may bias results. Although the sample phone list was scientifically generated, only 41% of the sample numbers actually resulted in a completed survey. An inherent bias in the telephone survey method is possible in that 5% of the rural western New York population do not have telephones.[18,19] This bias may seriously affect estimates of health insurance coverage because the population without telephones is more likely to be indigent and uninsured.[18] The retrospective methodology of a survey also exposes the results to recall bias. As noted, adult females and individuals over 65 years of age are over-represented in our survey. This is the likely result of the daytime prevalence for telephone surveying. Twenty-one percent of the individuals surveyed had some Medicare coverage, exceeding the

TABLE IV. Association Between Types of Health Care Sources Used and County of Residence*

County	Doctor's Office (%)	Clinic (%)	Other (%)	Total
Genesse	113 (81)	19 (14)	7 (5)	139
Orleans	103 (94)	4 (4)	3 (2)	110
Cattaraugus	181 (75)	21 (9)	38 (16)	240
Chautauqua	288 (82)	38 (11)	24 (7)	350
Allegany	91 (79)	15 (13)	9 (8)	115
Wyoming	61 (78)	9 (12)	8 (10)	78
Total	837 (81)	106 (10)	89 (9)	1,032

*Association between individual's county of residence and the source that they used for their last health care visit between June 1988 and January 1990 (Cramer's $V = .11, p < .01$).

TABLE V. Association Between Distance Traveled to Last Visit and County of Residence*

County	Distance Traveled in Miles (%)		
	≤20	≥20	Total
Genesee	114 (81)	26 (19)	140
Orleans	99 (90)	11 (10)	110
Cattaraugus	208 (87)	32 (13)	240
Chautauqua	315 (89)	37 (11)	352
Allegany	90 (78)	26 (22)	116
Wyoming	62 (81)	15 (19)	77
Total	888 (86)	147 (14)	1,035

*Association between individuals' county of residence and the distance that they traveled to their last health care visit between January 1988 and June 1990 (Cramer's $V = .12, p < .01$).

national rate by 3%, probably because of the overrepresentation of elderly individuals in our survey.[19]

Most households were able to identify a single regular source of health care. If not, the responder usually indicated that a single regular source was not perceived to be important, or the individual was seeing specialists. In the 1982 National Access Survey, individuals in higher income brackets tended to have regular health care sources, whereas low income individuals did not.[5] The majority of individuals reported seeking health care during the past 18 months in a physician's private office rather than a hospital or a clinic, and the physician tended to be their regular health care source. Although an inverse relationship between income and having a hospital as the regular source of health care has been found by national surveys, no relationship was determined in our data.[5] There are three explanations offered for the lack of income influence on health care access in the rural population: the rural population tends to be more financially homogeneous, factors such as transportation or attitude toward health care may have a greater influence than income; and because rural institutions have a less developed outpatient clinic system, non-physician office sources of health care are not as available.[19] If included, the lowest income person, inherently not well represented in a phone survey, may alter this observation. Other studies have suggested that rural residents have shorter travel distances to health care facilities than is commonly thought. However, although only 14% of these responders traveled more than 20 miles, this subgroup was significantly more likely to indicate a preference for a different facility.

Ten percent of the individuals in this study reported having had at least one overnight stay in a hospital, less than the 12% rate of short-stay yearly admissions to United States hospitals in 1988.[15] As expected, more of the individuals over age 64 had been admitted between June 1988 and January 1990, compared with the under 64 age group (Cramer's $V = 0.19, p < 0.001$). A majority of individuals were admitted to a rural facility. This preference for rural hospitals rather than urban hospitals is consistent with a June 1990 report by the Health Systems Agency of western New York, Inc, and suggests a continued, important role for rural hospitals.

The proportion of visits to hospitals or clinics varied from county to county. The number of physicians practicing in each county did not account for these differences. Of the three counties that had the highest proportion of individuals seeking care at a hospital or clinic, one (Allegany County) had the lowest non-federal physician-to-population ratio, another (Wyoming County) had an average ratio of physicians-to-population, and another (Chautauqua County) had one of highest physician-to-population ratios.[21] Planners have historically focused on the hospital as the unit of measure in health care access. Although important, hospitals provide only a small proportion of routine health care services in rural western New York. An assessment of health care services must reflect the consumer's use of non-hospital-based providers as well as acute inpatient services.

Traveling more than 20 miles to access health care was also not associated with the geographic size of the counties, as two of the largest counties—Chautauqua and Cattaraugus—included a smaller proportion of individuals who traveled 20 miles or more to their visit (Table IV). Distance traveled was also not associated with physician density. Allegany County had the lowest non-federal physician-to-patient ratio, and a high percentage of 20-mile visits, but Wyoming County had an average ratio, and Genesee County had a better than average ratio (Table V).[22]

A significant number (38%) of responders perceived a shortage of health care services in their communities. This is in spite of only 14% traveling over 20 miles and 15% identifying a regular source of health care. Although some barriers were identified, 80% expressed overall satisfaction with their last visit for health care. Four of five persons in the 1982 National Access Survey also were satisfied with their last visit.[5]

The results of our exploration of access indicators concur with those from past access studies in the United State indicating that people with a single doctor as a regular source of care are generally satisfied with all aspects of their care, and that satisfaction with care is high by most standards.[5] However, areas of dissatisfaction and barriers to receiving health care justify continued monitoring to facilitate both the planning and performance evaluation of local and national health care delivery systems.[5]

REFERENCES

1. Penchansky R, Thomas JW: The concept of access. *Med Care* 1981; 19: 127–140.
2. Kitagawa EM, Hauser PM: *Differential mortality in the United States: A study in socioeconomic epidemiology*. Cambridge, Havard University Press, 1973.
3. Marcus AC, Stone JD: Racial/ethnic differences in access to health care: Further comments on the use-disability ratio. *Med Care* 1982; 20:892–900.
4. Aday LA, Anderson R: *Access to Medical Care*. Michigan, Health Administration Press, 1975.
5. Aday LA, Anderson R: *Access to Medical Care in the US: Who has it, Who Doesn't*. Chicago, Pluribus Press, 1984.
6. Mayer J: Patterns of rural hospital closure in the US. *Soc Sci Med*, 1987; 24:327–334.
7. Newhouse JP: Where have all the doctors gone? *JAMA* 1982; 247:2392–2396.
8. Newhouse JP: Does the geographic distribution of physicians reflect market failure? *Bell J Econ* 1982; 13:493–505.
9. Ermann D, Gabel J: The changing face of American health care. *Med Care* 1985; 23:401–420.
10. Clifford WB, Brannon E: Rural-urban differences in mortality. *Rural Sociol 50* (Summer) 1985; 210–224.
11. Cordes SM: Rural health and rural development: Invited testimony presented to the Joint Economic Committee, US, Congress, Washington, DC, May 22, 1985.
12. Kelly K: Characteristics of financially distressed hospitals. National Center for Health Services Research, Pub No 83-3352, 1983.
13. Memories still echo in hospital corridors (Arnold Gregory Memorial Hospital). *Buffalo News* March 15, 1989, p B1.
14. Decrease in rural doctors, patients, causes closing of Salamanca Hospital. *Business First* March 26, 1990, p 11.
15. United States Bureau of the Census: Statistical abstract of the United States, Washington, DC, US Government Printing Office, 1989, 109th ed, p 15–18.
16. Aday, LA: *Designing and Conducting Health Surveys*. San Francisco, Jossey-Bass, Inc, 1989.
17. Statistical package for the social sciences, ed 3, Chicago, SPSS, Inc, 1988, B 242; C 207–209.
18. Corey CR, Freeman HE: Use of telephone interviewing in health care research. *Health Services Research*, 1990; 25 (pt1):129–144.
19. National Center for Health Statistics: United States Health 1989 and

Prevention Profile. Washington, DC, US Department of Health and Human Services. 1989, pp 3–11.

20. Williams AP, Schwartz MD, Newhouse JP, et al: How many miles to the doctor? *N Engl J Med* 1983; 309:958–963.

21. Roback G, Randolph L, Seidman B, et al: *Physician characteristics and distribution in the US*. Chicago, Department of Data Release Services, American Medical Association, 1987.

22. US Bureau of the Census: 1980 Census of the Population (vol 1): Characteristics of the Population, Part 34. Washington, DC, US Government Printing Office, 1982.

Health and economic impact of cigarette smoking in New York State, 1987–1989

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ABSTRACT. This study provides estimates of deaths attributable to smoking, years of potential life lost (YPLL), and economic costs for each of New York's 57 counties and New York City for the period 1987 to 1989. Results show that in New York State, cigarette smoking was responsible for an average of 30,359 deaths, 409,129 YPLL, and nearly $4 billion in economic costs annually. Overall, smoking-attributable deaths comprised 17.7% of all deaths, or more than the combined total from accidents, AIDS, homicide, and suicide. Findings demonstrate the enormous health and economic burden caused by tobacco use and reinforce the need for policies that discourage smoking, such as increased cigarette taxes, limited smoking in public places, enforcement of youth tobacco access laws, and funding for tobacco education programs.

(NY State J Med 1992; 92:469–473)

Cigarette smoking has been identified as the single most important preventable cause of premature mortality in the United States today.[1] Epidemiologic studies show that cigarette smoking is responsible for one third of deaths from cancer, 30% of deaths from coronary heart disease, and 80%–90% of deaths from chronic obstructive lung diseases.[1] A recent report from the Centers for Disease Control estimated that in the United States in 1988, 434,000 deaths and 6,028,000 YPLL were attributable to cigarette smoking.[2]

In spite of the enormity of the smoking problem, public health efforts to prevent tobacco use are disorganized and grossly underfunded. A recent survey of state tobacco prevention and control activities conducted by the Association of State and Territorial Health Officers found that, as of October 1990, only 12 states had specific tobacco prevention plans, and that the average state budget devoted to tobacco was under $75,000 annually.[3] In 1989–1990, the New York State Health Department reported devoting $290,000 of its total $963.2 million budget (0.031%) to tobacco prevention and control activities.[3]

This paper presents estimates of the mortality attributable to smoking, YPLL, and economic costs of smoking in each of New York's 57 counties and New York City. The findings from this analysis underscore the tremendous health and economic burden caused by tobacco use and the need to promote more aggressively tobacco prevention and control activities in every community.

METHODS

Calculations were performed using Smoking-Attributable Mortality, Morbidity, and Economic Cost (SAMMEC II) software, which includes relative risk estimates for 23 adult (ie, >35 years of age) smoking-related diseases as shown in Table I.[4] Disease-specific relative risk estimates are the same as those reported in the 1989 Surgeon General's Report on Smoking and Health.[1] Age and gender-specific mortality data for each of the diseases listed in Table I were obtained for each of New York's 57 counties and New York City for the period 1987–1989. A three-year observation period was selected to allow more stable estimates of disease-specific mortality.

Age- (two age groups: 35–64 years and 65+ years) and gender-specific smoking prevalence rates were estimated from the 1988 Behavioral Risk Factor Surveillance System Survey conducted in New York State.[5] The smoking prevalence figures used in the calculations are shown in Table II.

The number of deaths attributable to smoking in each county was computed by multiplying the number of deaths from each disease (by gender and five-year age groups) times the smoking-attributable fraction for each disease. Smoking-attributable fractions for male and female current and former cigarette smokers were based on data collected in the American Cancer Society's Cancer Prevention Study II.[1,4] Smoking-attributable YPLL were calculated for each smoking-related diagnosis by multiplying the number of deaths by the number of expected years of life remaining for each gender and age group. Values for the years of life remaining were based on United States life expectancies for all races combined.[4]

Smoking-attributable economic costs include three cost categories: direct health care costs, indirect mortality losses, and indirect morbidity losses. Direct health care costs were defined as personal health care expenditures to treat diseases related to cigarette smoking. To estimate these costs, we used New York State per capita health care figures for hospital care, physician services, nursing home expenses, medications, and other professional services. Because health care expenditures are not broken down by disease category, we used national estimates of the proportion of personal health care expenditures attributed to neoplastic, circulatory, and respiratory diseases.[6] Smoking-attributable direct health care costs were computed by multiplying the combined costs for these three disease categories by the smoking-attributable risk fractions for health care utilization. These risk fractions were based on the gender-specific prevalence rates for current smokers and the differential rates of medical care utilization for current smokers relative to never smokers.

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TABLE I. Relative Risk Estimates for Male and Female Current and Former Smokers Relative to Never Smokers for Smoking-Related Diagnoses—United States, 1988*

ICM-9-CM Adult Diseases	Diagnoses	Males Current Smokers	Males Former Smokers	Females Current Smokers	Females Former Smokers
Neoplasms					
140–149	Lip, oral cavity, pharynx	27.48	8.80	5.59	2.88
150	Esophagus	7.60	5.83	10.25	3.16
157	Pancreas	2.14	1.12	2.33	1.78
161	Larynx	10.48	5.24	17.78	11.88
162	Trachea, lung, bronchus	22.36	9.36	11.94	4.69
180	Cervix uteri	—	—	2.14	1.94
188	Urinary bladder	2.86	1.90	2.58	1.85
189	Kidney, other urinary	2.95	1.95	1.41	1.16
Cardiovascular diseases					
390–398	Rheumatic heart disease	1.85	1.32	1.69	1.16
401–404	Hypertension	1.85	1.32	1.69	1.16
410–414	Ischemic heart disease				
	ages 35–64	2.81	1.75	3.00	1.43
	ages 65+	1.62	1.29	1.60	1.29
415–417	Pulmonary heart disease	1.85	1.32	1.69	1.16
420–429	Cardiac arrest/other heart	1.85	1.32	1.69	1.16
430–438	Cerebrovascular disease				
	ages 35–64	3.67	1.38	4.80	1.41
	ages 65+	1.94	1.27	1.47	1.01
440	Atherosclerosis	4.06	2.33	3.00	1.34
441	Aortic aneurysm	4.06	2.33	3.00	1.34
442–448	Other arterial disease	4.06	2.33	3.00	1.34
Respiratory diseases					
010–012	Respiratory tuberculosis	1.99	1.56	2.18	1.38
480–487	Pneumonia, influenza	1.99	1.56	2.18	1.38
490–492	Bronchitis, emphysema	9.65	8.75	10.47	7.04
493	Asthma	1.99	1.56	2.18	1.38
496	Chronic airways obstruction	9.65	8.75	10.47	7.04
Other conditions					
809–899	Burn deaths	†	†	†	†

*Source: see references 1, 2 and 4.
†Smoking-attributable burn deaths determined from injury surveillance data. No relative risk estimate used.

Indirect mortality costs were defined as the cost of lost income and productivity resulting from premature death due to smoking-attributable disease. We estimated these costs by adding the totals of the present value of future earnings in 1988 dollars stratified by five-year age groups and gender, times the number of smoking-attributable deaths for each of the 23 diagnoses by age group and gender. We used 1985 estimates of the present value of future earnings with a 6% discount rate to adjust for future inflation. For our calculations, we inflated these figures to 1988 values.

Indirect morbidity costs were defined as the cost of lost income and productivity for persons disabled by smoking-attributable diseases. These costs were estimated using national data on total smoking-attributable economic costs.[7] Our indirect morbidity cost estimates assume that the ratio of indirect morbidity costs to direct health care costs and indirect mortality costs in each of New York's 57 counties and New York City is the same as the ratio observed in the United States. Using this ratio, and the values for direct health care costs and indirect mortality costs, we estimated the indirect morbidity costs for each county and New York City.

TABLE II. Prevalence Rates for Current and Former Cigarette Smokers by Gender and Age in New York State: 1988*

Age Group (yrs)	Males Current Smokers	Males Former Smokers	Females Current Smokers	Females Former Smokers
35–64	23.1	36.2	26.9	23.4
65 and over	12.9	50.1	7.2	31.3

*Source: Behavioral Risk Factor Surveillance System Survey[5]

RESULTS

Table III provides estimates of the smoking-attributable deaths and economic costs for each of New York's 57 counties and New York City for the three-year period 1987 to 1989. Dividing the number of deaths and economic costs by three gives an estimate of the average annual number of deaths and costs attributable to smoking. Overall, we estimate that between 1987 and 1989, cigarette smoking was responsible for 91,078 deaths, or 30,359 deaths per year. Smoking-attributable deaths comprised 17.7% of all deaths.

Between 1987 and 1989, the total economic costs of smoking in New York State were $11.8 billion, or an average of $3.9 billion per year. Direct health care costs comprised 33% of this total; indirect mortality costs, 57%; and indirect morbidity costs, 10%. Smoking-attributable economic costs varied widely between localities as a function of population size. However, even in the least populated county (Hamilton County), the estimated economic cost of smoking exceeded $1.6 million per year.

Table IV gives the statewide estimates of the number of smoking-attributable deaths due to cancer, cardiovascular diseases, respiratory diseases, and fires for males and females. Cardiovascular diseases accounted for the largest percentage of smoking-caused deaths (47%) followed by deaths from

TABLE III. Smoking Attributable Mortality and Economic Costs for New York's 57 Counties and New York City[*]; 1987–1989

	Smoking Attributable Mortality		Smoking Attributable Costs[†] in Millions	
County	Deaths (no)	Percentage of all Deaths	Direct Health Care Costs	Total Costs
Albany	1,655	18.7	$ 63	$ 190
Allegany	278	19.9	11	32
Broome	1,256	19.9	47	141
Cattaraugus	506	20.1	17	55
Cayuga	463	19.8	18	49
Chautauqua	854	18.6	32	93
Chemung	621	20.9	20	78
Chenango	284	18.8	11	33
Clinton	384	21.7	18	54
Columbia	387	19.2	13	49
Cortland	253	19.1	11	27
Delaware	309	19.5	11	32
Dutchess	1,249	19.5	58	169
Erie	6,067	19.6	214	701
Essex	248	19.9	8	25
Franklin	319	22.4	8	30
Fulton	349	19.8	12	38
Genesee	297	18.4	13	36
Greene	272	18.9	9	30
Hamilton	40	20.3	1	5
Herkimer	398	19.5	15	42
Jefferson	565	20.5	24	69
Lewis	137	19.9	6	16
Livingston	273	18.8	13	35
Madison	322	19.7	15	40
Monroe	3,118	17.7	157	424
Montgomery	369	19.4	12	34
Nassau	6,301	18.1	295	838
Niagara	1,302	19.8	48	154
Oneida	1,495	18.5	55	166
Onondaga	2,299	19.0	104	290
Ontario	464	19.2	21	55
Orange	1,431	19.4	64	187
Orleans	220	19.9	9	25
Oswego	620	20.9	27	80
Otsego	333	18.4	13	35
Putnam	334	20.1	19	51
Rensselaer	868	19.9	34	104
Rockland	1,110	18.4	60	162
St. Lawrence	660	21.1	25	77
Saratoga	735	19.7	38	101
Schenectady	917	19.2	34	98
Schoharie	155	18.0	7	18
Schuyler	101	19.5	4	12
Seneca	179	19.7	7	23
Steuben	631	21.4	22	67
Suffolk	6,147	19.4	295	870
Sullivan	475	19.8	16	53
Tioga	219	19.7	12	30
Tompkins	334	19.3	20	43
Ulster	911	19.4	37	110
Warren	331	19.8	13	38
Washington	337	20.9	13	41
Wayne	421	19.6	20	54
Westchester	4,466	18.3	194	554
Wyoming	215	19.9	9	27
Yates	137	19.9	5	14
New York City	35,657	15.8	1,642	5,071
Total	91,078	17.7	$3,999	$11,804
Average over 3 years:	30,359		$1,333	$ 3,935

[*] New York City includes Bronx, Kings, Manhattan, Queens, and Richmond.

[†] Costs are rounded to the nearest million in 1988 dollars.

TABLE IV. Smoking Attributable Mortality for Males and Females Due to Cancer, Cardiovascular Diseases, Respiratory Diseases, and Fires in New York State, 1987–1989

	Number of Deaths Due to Smoking		
Disease Category	Males	Females	Total
Cancer	20,759	9,945	30,704
Cardiovascular diseases	26,814	16,309	43,123
Respiratory diseases	9,962	6,706	16,668
Burn deaths	320	263	583
Total deaths due to smoking	57,855	33,223	91,078
Average over 3 years:	19,285	11,074	30,359
Percentage of all deaths due to smoking	22.1	13.1	17.7

cancer (34%), deaths from respiratory diseases (18%), and fire deaths (1%). Overall, we estimate that 27% of all cancer deaths and 23% of deaths from cardiovascular disease were attributable to cigarette smoking.

Table V shows the YPLL due to smoking for each of New York's 57 counties and New York City for the period between 1987 and 1989. Smoking-attributable deaths resulted in the loss of approximately 1.2 billion YPLL, or an average of 409,129 potential years of life lost per year. The average YPLL per smoker death was 13.5.

DISCUSSION

The findings from this analysis document the huge health and economic impact of cigarette smoking in each of New York's 57 counties and New York City. Overall, we estimate that cigarette smoking kills more than 30,000 citizens and costs New Yorkers nearly $4 billion annually.

Although our estimates of smoking's adverse impact are based on extensive epidemiologic research, there are a number of limitations to the methodology we have used to estimate smoking-attributable deaths by locality. The smoking-attributable risk fractions used in the computations depend in part on the relative mortality risk for smokers compared with nonsmokers. These risk estimates may be affected by other behaviors that are correlated with smoking, such as alcohol use. Our risk estimates were restricted to persons 35 years of age and older, thus excluding deaths caused by smoking among younger persons (eg, perinatal deaths). Such events, although relatively rare, often carry enormous medical costs and many more YPLL than the average we have calculated. In addition, our calculations did not consider deaths due to environmental tobacco smoke (ETS). In this regard, our smoking-attributable mortality estimates are likely to be conservative. Finally, the smoking prevalence estimates used in these computations were based on statewide estimates, since local prevalence data were not available. However, an examination of smoking prevalence rates by geographic region indicates that prevalence rates are fairly uniform across New York State.

The methodology used to compute the economic costs of cigarette smoking also has limitations. For example, statewide averages were used to estimate personal health care expenditures, medical care utilization for specific illnesses was based on rough approximations, and morbidity costs were based on incomplete data regarding the differential morbidity between smokers and nonsmokers. In spite of these limitations, there is reason to believe that our calculations underestimate the economic costs associated with cigarette smoking. For instance, costs associated with treating smoking-caused diseases among former smokers were not considered. Cost estimates were limited to smoking attributable neoplastic, circulatory,

TABLE V. YPLL Due to Smoking for New York's 57 Counties and New York City*; 1987–1989

	YPLL Before Life Expectancy		
County	Males	Females	Total
Albany	12,770	8,523	21,293
Allegany	2,126	1,403	3,529
Broome	9,876	6,141	16,017
Cattaraugus	4,143	2,402	6,545
Cayuga	3,732	1,892	5,624
Chautauqua	6,519	4,192	10,711
Chenango	2,268	1,409	3,677
Chemung	4,820	2,866	7,686
Clinton	3,489	1,853	5,342
Columbia	3,011	1,731	4,742
Cortland	1,968	1,092	3,060
Delaware	2,435	1,383	3,818
Dutchess	10,630	6,148	16,778
Erie	49,935	30,775	80,710
Essex	2,046	1,035	3,081
Franklin	2,405	1,636	4,041
Fulton	2,803	1,591	4,394
Genesee	2,412	1,393	3,805
Greene	2,218	1,230	3,448
Hamilton	365	171	536
Herkimer	3,045	1,889	4,934
Jefferson	4,467	2,995	7,462
Lewis	1,153	548	1,701
Livingston	2,132	1,436	3,568
Madison	2,687	1,420	4,107
Monroe	24,789	16,505	41,294
Montgomery	2,657	1,721	4,378
Nassau	51,651	32,427	84,078
Niagara	10,562	6,920	17,482
Oneida	11,322	7,770	19,092
Onondaga	18,464	11,640	30,104
Ontario	3,746	2,111	5,857
Orange	11,842	7,295	19,137
Orleans	1,773	1,053	2,826
Oswego	5,478	2,845	8,323
Otsego	2,365	1,638	4,003
Putnam	2,961	1,576	4,537
Rensselaer	7,073	4,365	11,438
Rockland	8,901	5,986	14,887
St. Lawrence	5,290	3,500	8,790
Saratoga	6,284	3,489	9,773
Schenectady	6,880	4,460	11,340
Schoharie	1,143	862	2,005
Schuyler	838	531	1,369
Seneca	1,547	891	2,438
Steuben	5,099	2,916	8,015
Suffolk	51,979	33,656	85,635
Sullivan	4,104	2,143	6,247
Tioga	1,772	1,109	2,881
Tompkins	2,424	1,578	4,002
Ulster	7,379	4,476	11,855
Warren	2,569	1,693	4,262
Washington	2,796	1,706	4,502
Wayne	3,683	1,723	5,406
Westchester	34,214	23,140	57,354
Wyoming	1,806	1,035	2,841
Yates	1,112	598	1,710
New York City	298,082	200,835	498,917
Total	746,040	481,347	1,227,387
Average over 3 years:	248,680	160,449	409,129
Average YPLL per smoker death	12.9	14.5	13.5

*New York City includes Bronx, Kings, Manhattan, Queens, and Richmond.

and respiratory diseases. Therefore, our economic estimates do not include smoking-attributable costs associated with illnesses and deaths from fires, perinatal injury, or ETS.

Comparing cigarette smoking with other health problems provides perspective on the relative importance of tobacco use as a public health problem in New York State. Between 1987 and 1989, cigarette smoking accounted for more deaths in New York State than accidents, AIDS, homicide, and suicide combined.

In view of the magnitude of the tobacco problem in New York State, it is distressing to observe the low level of tobacco control activities carried out by government agencies charged with protecting public health. A 1990 survey of county health departments revealed that those reporting any activity in general restricted their tobacco control activities to the distribution of literature (unpublished data, Bureau of Adult and Gerontological Health, NYS Department of Health, February 1990). At the state health department level, tobacco prevention and control activities account for only a small fraction of the overall budget (0.031%).[3] While many nongovernmental agencies sponsor a wide range of tobacco control activities, in all communities, the resources devoted to combat the tobacco problem are substantially lower than the resources expended by the tobacco industry to encourage smoking.[1]

While the findings from this study support the need for public health agencies to consider allocating a larger share of their resources to tobacco prevention and control activities, the evidence also suggests that tobacco use can be reduced by increasing the price of cigarettes through taxation.[8,11,12] At a time when the state government is operating at a deficit, increasing the cigarette excise tax not only makes fiscal sense but public health sense as well. Increasing the excise tax on cigarettes will produce new revenue and, more importantly, will reduce demand, particularly among potential new nicotine addicts, such as teenagers.[1,9] Policy makers in New York State should consider earmarking a portion of tobacco tax revenues to finance tobacco prevention and control activities, as other states have done.[3,10]

Taxing tobacco products is only one element of an overall comprehensive approach to reducing tobacco use.[8,12] Clearly, there is still a need to continue to educate people about the dangers of tobacco use and for policies that restrict smoking in public places, limit tobacco advertising and promotion, and reduce youth access to tobacco products. The 16 tobacco-related objectives recently outlined in the US Department of Health and Human Services *Healthy People 2000: National Health Promotion and Disease Prevention Objectives*, provide a useful guide for planning and assessing progress in preventing and controlling tobacco use at the local, state, and national level.[13]

REFERENCES

1. Centers for Disease Control: Reducing the health consequences of smoking: 25 years of progress. A report of the surgeon general. Rockville, MD: US Department of Health and Human Services, Public Health Service, 1989, DHHS publication no. (CDC) 89-8411.
2. Shultz JM: Smoking-attributable mortality and years of potential life lost—United States, 1988. *MMWR* 1991; 40:62–63, 69–71.
3. Centers for Disease Control: State tobacco prevention and control activities: Results of the 1989–1990 Association of State and Territorial Health Officials (ASTHO) survey final report. *MMWR* 1991; 40(No. R-11):1–41.
4. Shultz JM, Novotny TE, Rice DP: Quantifying the disease impact of cigarette smoking with SAMMEC II software. *Public Health Rep* 1991; 106:326–333.
5. Centers for Disease Control: Behavioral risk factor surveillance, 1988. *MMWR* 1990; 39:1–21.
6. Levit KR: Personal health care expenditures by state: 1966–1982. *Health Care Financing Rev* 1985; 6:1–49.
7. Rice DP, Hodgson TA, Sinsheimer P, et al: The economic costs of the health effects of smoking, 1984. *Milbank Quart* 1986; 64:489–547.

8. Kaiserman MJ, Rogers B: Tobacco consumption declining faster in Canada than in the US. *Am J Public Health* 1991; 81:902–904.

9. Cummings KM, Sciandra R: The public health benefit of increasing tobacco taxes in New York State. *NY State J Med* 1990; 90:174–175.

10. Bal DG, Kizer KW, Felten PG, et al: Reducing tobacco consumption in California. Development of a statewide anti-tobacco use campaign. *JAMA* 1990; 264:1570–1574.

11. Peterson DE, Zeger SL, Remington PL, Anderson HA: The effect of state cigarette tax increases on cigarette sales, 1955 to 1988. *Am J Public Health* 1992; 82:94–96.

12. Simonich WL: *Government Antismoking Policies.* New York: Peter Lang Publishing Inc, 1991.

13. US Department of Health and Human Services: *Healthy People 2000: National Health Promotion and Disease Prevention Objectives.* Washington, DC, US Department of Health and Human Services, Public Health Service, 1990, DHHS publication No (DHS) 90-50212.

FROM THE LIBRARY

CIGARETTE ADVERTISEMENTS

It seems strange that tobacco, or a device for its use, should find a place in the advertising columns of a medical journal, and yet it is a fact that several medical journals on our exchange list do carry advertisements of cigarettes and cigarette holders. The psychology of cigarette advertisements is the subject of the following editorial taken from the *New York Times* of January twenty-fifth.

"The Federal Trade Commission, striking for honesty in claims, has given national good taste a push forward. The advertising manager who conceived the large idea which the commission has buried began with an emotion. It was to prove that his client's cigarettes could go several millions higher in annual sales. The first objective was 'big names.' Whether they smoked Vocos made no difference; pay them $1,000 and let them imply that whatever quality the world admired them for had something to do with the use of that particular brand. From that beginning it was a short step to the premise that health is the foundation of success; and, on the stage, health joined to beauty. Day after day pictures and endorsements from obviously healthy and beautiful—and successful—folk linked them with 'the respondent's brand.' It was fine until envious competitors, and joshing by friends of non-smokers whose endorsements and photographs had appeared in the advertising columns, interested the Federal Trade Commission. Now the cigarettes have to fall back upon their, doubtless, excellent intrinsic merits as smoking material only."

ADVERTISEMENTS
(*NY State J Med* 1930: 30; 434)

MEDICAL CONFERENCE

Widespread complications of lupus erythematosus

Case Presentation

Vito Mercurio, MD (Resident in Internal Medicine): A 29-year-old Haitian man presented to the North Shore Emergency Department with increasing fatigue, muscle weakness, and abnormally slow speech over the preceding two weeks.

Nine months prior to admission, the patient presented with polyarthritis, discoid lupus, and Raynaud's phenomenon; at the time he was diagnosed as having systemic lupus erythematosus (SLE). Serological studies revealed a speckled anti-nuclear antibody in a serum titer of 1:2560; double-stranded DNA antibodies studies were positive 1:160 (*Crithidia luciliae*), together with positive rheumatoid factor, negative extractable nuclear antigens, negative Scl 70, low third component of complement (C3) at 27 mg/dL, (normal range 80–180 mgL), low fourth component of complement (C4) 13 mg/dL (normal range 20–50 mg/dL), and normal T-cell subsets.

Shortly thereafter, he was admitted to another hospital with complaints of polyarthritis and mental status changes. After admission, he developed loss of sensory and motor function of his lower extremities with microangiopathic hemolytic anemia and thrombocytopenia. He lapsed into coma, with multi-organ failure; was treated with pulse steroids, cyclophosphamide, and plasmapheresis with much improvement in his neurological status over the subsequent six-month course of his hospitalization. He experienced one episode of supraventricular tachycardia with congestive heart failure, thought to be due in part to medication with verapamil.

During the three to four months after discharge, while undergoing rehabilitation, his condition remained unchanged, until his arrival at our Emergency Department. He was readmitted with a history of increasing fatigue, muscle weakness (lower extremity greater than upper), abnormally slow speech, and occasional dyspnea on exertion. He denied allergies, fever, chills, headache, photophobia, abdominal pain, and weight loss.

His past medical history included hypertension; medications included prednisone 15 mg daily, enalapril maleate, metoprolol tartrate-hydrochlorothiazide, and furosemide.

His family history was negative for SLE, connective tissue disorders, and cancer. He worked as a physician's assistant, and denied homosexual contacts, smoking, alcohol abuse, or intravenous drug abuse.

Physical examination disclosed a thin young man in no acute distress. His blood pressure was 100/50 mm Hg; respiratory rate 16 was per minute, pulse rate was 116/min, and temperature was 38° C. Positive physical findings included cotton wool exudates in both ocular fundi; the discs and disc margins were sharp. He showed no cervical venous distention, and no palpable lymphadenopathy. A few scattered crackling rales were present at the lung bases. The cardiac point of maximal impulse was in normal location; the first and second heart sounds (S1, S2) were normal; no gallops, murmurs, or rubs were heard. The abdomen was soft and non-tender; bowel sounds were normal; no organs were palpable. The peripheral pulses were normally palpable throughout; there was full range of motion of all joints. The finger tips were cold bilaterally. There was no cyanosis, clubbing, or edema. Hypopigmentation of the hands and knees were present.

The neurological examination revealed an alert, well-oriented man, with some slowing of speech and slowed mentation. Motor strength was normal and symmetrical in the upper extremities; a mild degree of proximal muscle weakness was present in the lower extremities, but peripheral strength was normal. There was no sensory defects. His gait was wide based; the deep tendon reflexes were considerably diminished in the lower extremities, with absent ankle reflexes and moderately diminished responses in the upper extremities bilaterally. The Babinski sign was positive bilaterally. He also had primitive reflexes, with positive frontal release signs.

The urine was yellow, clear, with a specific gravity of 1.025, and 3 plus proteinuria; microscopic examination revealed 5–9 white blood cells (WBC) and 10–14 red blood cells (RBC) per HPF, with granular and hyaline casts. The hemoglobin was 10.8 gm/dL, the hematocrit was 31.4%, the WBC count was 3,300/μL, and the peripheral blood smear disclosed normochromic normocytic RBC with schistocytes. The platelet count was 278,000/μL, the prothrombin time was 11.4 seconds, and the partial thromboplastin time (PTT) was 34.4 seconds. The erythrocyte sedimentation rate was 60 mm/hour; the serum electrolyte levels were the following: sodium 130 mmol/L, potassium 4.5 mmol/L, chloride 101 mmol/L, and carbon dioxide 24 mmol/L. The blood urea nitrogen was 16 mg/dL, serum creatine was 0.8 mg/dL, phosphate was 4.3 mg/dL, serum albumin was 2.9 gm/dL, cholesterol was 258 mg/dL, total bilirubin was 0.4 mg/dL, indirect bilirubin was 0.3 mg/dL, serum alkaline phosphatase was 143 U/L, lactate dehydrogenase was 199 U/L, serum aspartate aminotransferase was 24 U/L, serum alanine aminotransferase was 33 U/L, and serum creatine phosphokinase was 26 U/L.

The radiograph showed no infiltrates or effusions. The ECG recorded sinus tachycardia with poor R-wave progression in the precordial leads.

After admission, computed tomography (CT) and magnetic resonance imaging (MRI) disclosed diffuse cerebral atrophy and possible right basal ganglia ischemia. A lumbar puncture revealed no positive findings, and treatment with intravenous methylprednisolone was begun. The patient's mental status slowly deteriorated with progressive inability to vocalize, urinary incontinence, bradykinesis, the appear-

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Based on a medical conference held at the North Shore University Hospital, Manhasset, NY on October 11, 1991.

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ance of upper extremity weakness, and ultimately quadriparesis.

Polyarteritis nodose was considered, but the results of electromyography with nerve conduction velocity studies were within normal limits initially. The electroencephalogram was consistent with encephalopathy.

The patient's progressive deterioration was similar to that observed on his earlier admission to the other hospital, except for the absence of microangiopathic hemolytic anemia. Plasmapheresis was performed and continued for several treatments without improvement; cyclophosphamide was given with improvement in his mental status and a lesser degree of improvement in upper extremity strength.

He then developed respiratory distress secondary to congestive heart failure. An echocardiogram disclosed global left ventricular dysfunction.

Over the ensuing months, he experienced multiple episodes of sepsis with various organisms; he was intubated and extubated on multiple occasions and developed Guaiac-positive stools and a decubitus ulcer. His complement levels C3 and C4 normalized with a negative double-stranded DNA test at one point when he seems to be improving neurologically. However, they also remained normal through episodes and times of neurological decompensation. He eventually developed polymicrobial septicemia, suspected left subclavian thrombosis, and multi-organ system failure requiring inotropic blood pressure support. He expired on the 110th hospital day.

Melissa Sapan, MD (Attending Radiologist): A CT scan of the brain shortly after admission was entirely unremarkable except for generalized atrophy. A T_1-weighted MRI of the brain at the level of the basal ganglia showed no abnormal increased or decreased signal. The corresponding cut on a T_1-weighted image with gadolinium also disclosed no enhancement or abnormalities. The T_2-weighted image in the region of the basal ganglia showed a very faintly increased signal, more on the right than the left, and is highly indicative of basal ganglia ischemia.

The chest film two days before the patient's death showed bilateral air space disease with air bronchograms, which could be secondary to pulmonary edema, adult respiratory distress syndrome, or pneumonia. Severe subcutaneous emphysema can be seen along the soft tissues of the thorax and extending into the soft tissues of the neck. At that time the patient was intubated and had a central line and a naso-gastric tube. The cardiac silhouette appears enlarged.

David Cooper, MD (Assistant Professor of Medicine, Cornell University Medical College): Did the patient have a follow-up MRI later in his hospital course?

Dr Sapan: Not to my knowledge.

Dr Cooper: Was it felt that the patient had an encephalitis, or more likely an encephalopathy?

Dr Mercurio: At the time of admission, several possibilities were considered. Progressive multifocal leukoencephalopathy was a good possibility, as well as central nervous system (CNS) vasculitis caused by SLE or possibly caused by PAN, particularly since the peripheral nervous system was also involved. A brain biopsy was contemplated but not performed.

B. Robert Meyer, MD (Associate Professor of Clinical Medicine and Pharmacology, Cornell University Medical College): How did you address the treatment of these various possibilities?

Dr Mercurio: Initially steroids were administered by the pulse method in light of the possibility of SLE vasculitis, but the patient failed to respond. Then, because his present clinical picture resembled that which had developed at the other hospital, that is, findings similar to that of a thrombotic thrombocytopenic purpura (TTP)-like picture, the patient underwent plasmapheresis but without significant effect. Once cyclophosphamide was administered, his mental status appeared to improve.

Steven L. Allen, MD (Associate Professor of Clinical Medicine, Cornell University Medical College): I have two questions: were the retinal cotton-wool exudates believed to represent *Candida* ophthalmitis consequent to the steroid therapy; and secondly, what was the status of the patient's HIV antibody titer?

Dr Mercurio: The fundal changes were thought to be the result of vasculitis and not a fungal infection. An human immunodeficiency viral (HIV) test was discussed with the family, but they would not allow us to perform it. The T-cell subsets initially at the other hospital were normal; but when obtained here were entirely consistent with an HIV infection. However, the patient had been treated for a long time with steroids, which can generate the same changes in T-cell subsets as can HIV infection.

Dr Meyer: I have asked Dr Furie, the patient's attending physician, to discuss the way in which this patient's clinical problem was managed.

Richard Furie, MD (Assistant Professor of Medicine, Cornell University Medical College; Division of Rheumatology, Allergy, and Clinical Immunology, North Shore University Hospital): The best description of this patient's clinical appearance at the time of admission was one of extreme slowness. Bradykinesia was his chief presenting symptom. Although his behavior was entirely appropriate in following commands and speaking, his movements and speech were extremely slow. He required about ten minutes to walk the 15-foot corridor in our office. He had a very broad-based gain. When asked to undress, he required 20 minutes to remove all of his clothes.

Initially, we thought that he might be suffering from an encephalopathy, except that his thought processes were intact. The neurological consultant who saw him on that same day was convinced that the patient was not encephalopathic, but that his motor deficit was caused by a lesion localized in the basal ganglia. Our view here today of the MRI is not the best, but in fact, it suggests abnormalities that were thought to be vascular in etiology in the region of the basal ganglia. In summary, at the time of admission, we thought that the patient presented a focal deficit in the CNS.

The pathogenesis of CNS disease in SLE is a rather confusing subject.[1] It can be classified into different types:

I. Vascular disease
   A. Affecting large blood vessels, producing focal abnormalities, typically strokes
      1. Rarely caused by vasculitis; or
      2. Caused by thrombotic disease with or without vasculitis; or
      3. Caused by embolic disease from carotid atherosclerotic plaques or from the heart.
   B. Affecting small blood vessels
      1. Caused by vasculitis; or
      2. Caused by non-inflammatory microangiopathy: thrombotic, associated with thrombotic microangiopathy, TTP-like, with diffuse CNS

involvement; or occlusive, associated with the antiphospholipid syndrome, also causing diffuse CNS involvement.

II. Parenchymal CNS disease. This is well described in SLE, although the pathogenesis is not well understood. It generally presents as meningitis, seizures, and encephalopathy, or psychiatric disease. At times, infections secondary to treatment regimes are the cause of neurologic dysfunction.

Initially, this patient was believed to have CNS vascular disease secondary to SLE; however, during the week after admission, his clinical condition changed from the appearance of a focal basal ganglia deficit to one of more widespread disease with coma. In addition, within about a week after admission his extremity weakness progressed to a flaccid quadriplegia with absent deep tendon reflexes.

His disease was no longer limited to the CNS but had spread to the peripheral nervous system as well. Involvement of the latter is not unusual in SLE, since vasculitis can lead to peripheral nerve infarction.

The pathogenesis of this widespread disease could be best understood by the presence of vasculitis, even though the cotton-wood exudates in the retinae were the only clinically obvious stigmata of the vasculitis. There were no cutaneous changes that supported a diagnosis of vasculitis.

The possibility of a vasculopathy with multiple widespread vascular occlusion was considered, in the form of either thrombotic microangiopathy or occlusive vasculopathy. With this patient, our experience now includes seven lupus patients who have developed thrombotic microangiopathy, some with CNS manifestations.[2]

I first saw this patient at another hospital one year prior to admission here. At that time, he was comatose and had both CNS and peripheral nervous system disease, thrombocytopenia, and a hemolytic anemia with schistocytes on the blood smear. He did not respond to large doses of steroids but improved within a few days after plasmapheresis. We concluded at that time that he was suffering from thrombotic microangiopathy, that is, an illness resembling TTP, and he responded to the appropriate treatment for that condition. In fact, he recovered almost completely from that episode.

When he appeared at our hospital a year later, we considered the possibility that he had thrombotic disease in blood vessels supplying both the CNS and the peripheral nervous system. However, there was insufficient clinical evidence to support this supposition. On this occasion, he did not repeat his earlier clinical pattern of thrombocytopenia or hemolytic anemia which we had observed a year earlier. Therefore, it was difficult to be sure that he had a thrombotic microangiopathy along the lines of TTP.

The other possibility, although remote, was that of an occlusive vasculopathy which had previously been described in the antiphospholipid syndrome, either in the primary syndrome or as a complication of SLE.[3] Although the pattern of occlusion in the antiphospholipid syndrome is typically one of regional occlusion of small arteries or veins, a few reports of disseminated disease exist. This syndrome may lead to occlusion of blood flow to many organs, but thrombocytopenia and microangiopathic hemolytic anemia are not characteristic of the syndrome.

Although we were seriously considering vasculitis, we were initially disturbed that there was no observable cutaneous evidence of that condition. Therefore, thrombotic or occlusive vasculopathies remained viable alternatives. However, with the passage of time, about six to eight weeks after admission, the cutaneous evidence of vasculitis eventually appeared in that he developed widespread gangrene.

This patient's course passed through three phases:

1. The initial insult to his central and peripheral nervous systems
2. After aggressive therapy, the phase of improvement in mentation and in upper extremity but not lower extremity movement; and finally
3. The relapse of nervous system disease, now with evidence of gangrene of the fingers and toes that indicated the presence of vasculitis. Shortly thereafter, he developed the first of numerous septic complications that eventually led to his demise.

Our failure to understand the pathogenesis of his neurologic disease contributed to difficult decision-making regarding therapy. Because of the possibility of an exacerbation of SLE with vasculitis, he received intravenous "pulse" steroids (methylprednisolone 1 g/day for three days), and later he received intravenous "pulse" cyclophosphamide (one gram).

In spite of the lack of evidence of microangiopathy on his blood smear and in his laboratory findings, the early phase of his illness was so similar to that of his earlier hospitalization elsewhere that we decided to employ plasmapheresis as well. Thereafter, he improved mentally and regained a small measure of strength in his upper extremities, to the degree that he could move his arms against gravity. The insults inflicted by his disease affected his peripheral nervous system more than his CNS, and as a result he was quadriplegic. In addition, he also suffered repeated septic complications.

Our overall impression was that diffuse vascular disease would constitute the best unifying overall explanation of both the CNS and the peripheral nervous system disease. Whether it was true vasculitis or a vasculopathy remains to be seen. My bias is toward an inflammatory process, that is, a vasculitis, even though on his earlier admission to the other hospital the clinical picture suggested more strongly thrombotic microangiopathy.

Dr Allen: Statements in the medical literature urge that plasmapheresis be used aggressively in patients with TTP, over at least a two-week period to assess any evidence of its effectiveness.[4–7] Sometimes staphylococcal protein A columns are useful in increasing adsorption and the efficacy of the plasmapheresis.[8,9] Was that used in this case?

Dr Furie: At the other hospital, he was given the usual plasmapheresis regimen with seven exchange infusions of fresh frozen plasma. In this hospital, I am not sure of the number of exchanges, but we encountered complications which at first delayed the procedure and then forced its discontinuance.

Dr Mercurio: I believe that he received three cycles of plasmapheresis in this hospital.

Dr Furie: There was no truly valid reason for attempting plasmapheresis on this admission. His platelet count was normal, and there was no evidence of hemolytic anemia. However, since he was deteriorating so rapidly and in view of his earlier favorable response to plasmapheresis at the other hospital, we tried it empirically.

Final Clinical Diagnosis

SLE, with previous thrombotic microangiopathy; more recently with widespread central and peripheral nervous system damage secondary to disseminated vascular occlusive disease on the basis of vasculitis or a non-inflammatory occlusive vasculopathy.

PATHOLOGY PRESENTATION

Maryanne Mason, MD (Resident Pathologist): At autopsy, external examination revealed ulcerations at the tip of the nose and of both upper and lower lips; gangrene of all digits on all four extremities; an 8-cm grade IV sacral decubitus ulcer; and 3 plus edema of the scrotum and of the lower extremities.

On opening the thoracic cavity, the mediastinum was emphysematous, and the pleural cavities contained 250 cc of fluid on the right and 200 cc on the left. The abdominal cavity contained 750 cc of ascitic fluid.

Each kidney weighed 200 g; the left kidney revealed a large 8 × 4 × 4 cm infarct and multiple smaller infarcts. The right kidney showed congestion of the cortex with multiple petechiae in the pelvic mucosa. Microscopic sections of the hilum of the left kidney showed old thrombi in the arteries and veins; the elastic stain revealed a normal internal elastic lamina in the renal arteries, indicating no evidence of vasculitis. The glomeruli displayed slight mesangial hypercellularity and focal thickening of the capillary wall with wire-loop formation. Electron microscopy disclosed epimembranous granular deposits with spikes consistent with membranous glomerulonephritis (Fig 1), lupus nephritis class V, according to the classification of the World Health Organization (WHO).

In view of the extensive digital gangrene in all four extremities, tissue samples were obtained from popliteal and axillary regions bilaterally, all of which showed extensive venous thrombosis in varying degrees of organization (Fig 2). The popliteal artery showed a narrowed lumen secondary to medial hypertrophy, and the lumens of smaller arteries were narrowed by dome-shaped, subintimal, fibromuscular proliferation, as well as organizing intraluminal thrombi. The elastin stain revealed subintimal, fibromuscular proliferation in both the large and small arteries with intact internal elastic lamina (Fig 3).

Longitudinal and cross sections of the peripheral nerves from the popliteal and axillary regions disclosed normal nerve tissue with minimal interstitial fibrosis. Sections of voluntary muscle tissue from these areas revealed marked atrophy of fibers with proliferation of sarcolemmal nuclei, focal necrosis, and features of muscle regeneration. The trichrome stain revealed varying degrees of endomysial and perimysial fibrosis and fatty infiltration. These findings are indicative of end-stage muscle disease, the cause of which, whether neuropathic, myogenic, or metabolic, cannot be determined with certainty. Nevertheless, the long-standing and profound ischemia secondary to the extensive arterial and venous thromboses appears to be the most likely cause of the advanced muscle disease.

The heart, weighing 500 g, displayed massive globular dilation of the left ventricle. Mural thrombi were present in the inferior left ventricle associated with a resolving subendocardial infarction.

The right lung weighed 1300 g, and the left 700 g. Sectioned surfaces of the lungs showed multiple emboli in the pulmonary arteries with areas of infarction and marked pneumonic consolidation with microabscesses. Microscopic study revealed a confluent bacterial and fungal pneumonia, and septic infarcts undergoing liquification necrosis with cavitation. Gram stains in these areas revealed gram-positive cocci, probably *Staphylococcus aureus* because the blood cultures were positive for that organism. GMS stains

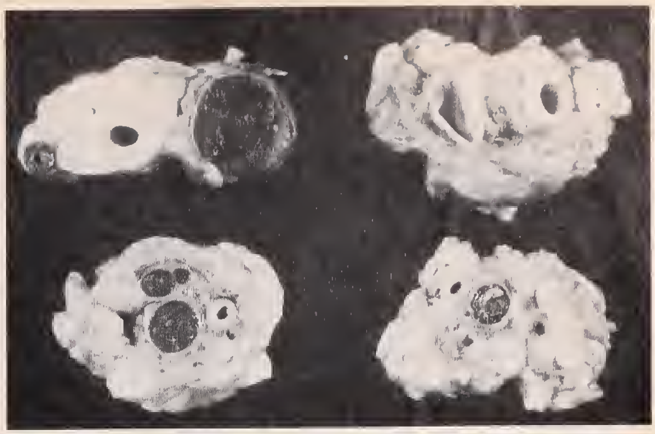
FIGURE 2. Gross specimens of tissues from bilateral popliteal (upper sections) and axillary regions (lower sections) showing venous thrombosis involving large and small veins.

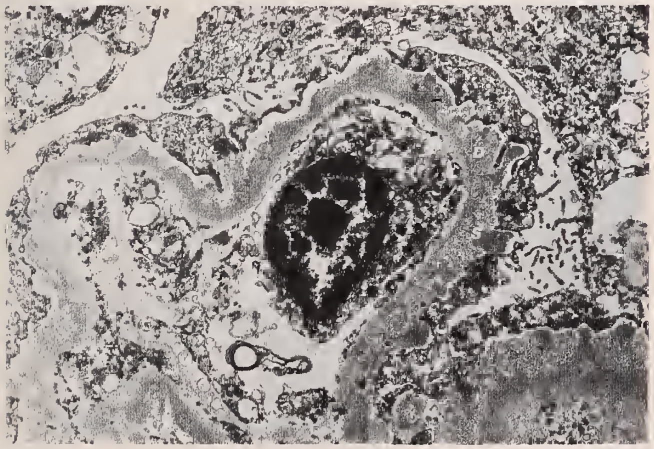
FIGURE 1. Electron micrograph of glomerulus showing epimembranous granular deposits (D) associated with spike-like projections (S). (Original magnification × 11,500.)

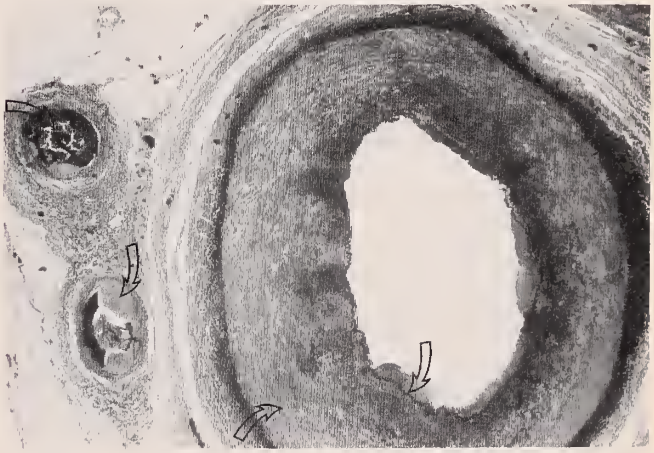
FIGURE 3. Photomicrograph of left popliteal artery showing intact elastic lamina (right arrow) with medial hypertrophy and subintimal fibromuscular proliferation (left arrow). Two small arteries show domed-shaped subintimal fibromuscular proliferation (arrow at lower small artery) and organizing thrombi (arrow at upper small artery) causing marked stenosis of their lumina. (Elastic von Gieson stain; original magnification × 40.)

were positive for hyphae and spores consistent with *Candida* species.

There was disseminated candidiasis involving the trachea, pharynx, digestive tract, urinary bladder, heart, and liver. The brain, weighing 1,340 g, contained areas of subacute encephalomalacia involving the basal ganglia and cerebral cortex. In addition, multiple recent microinfarcts were noted in the cerebrum and brain stem. Sections of the spinal cord showed intact anterior horn cells and normal nerve roots.

In summary, the autopsy revealed lupus glomerulonephritis (WHO class V), a dilated cardiomyopathy with mural thrombi, multiple thromboemboli in both arterial and venous system with pulmonary, renal, and cerebral infarcts; end-stage muscle disease of uncertain etiology; and terminal disseminated candidiasis.

Because of his extensive venous and arterial thromboses, we considered the possibility of the antiphospholipid antibody (anticardiolipin) syndrome. One disturbing fact, however, was that his serum anticardiolipin antibody level was only marginally elevated to immunoglobulin G (IgG) 19 (normal range 0–14) and IgM 5 (normal range 1–5). A review of the medical literature offers two possible explanations for the low anticardiolipin antibody levels:

1. A fall in antiphospholipid antibody level at the time of a thrombotic event is seen, in contrast to the antibody levels before and after the thrombo-occlusive episode; therefore, the result of the antiphospholipid antibody test may be misleadingly negative at the time of thrombosis.[10]
2. The administration of glucocorticoids, Ig, azathioprine, cyclophosphamide, plasmapheresis, or a combination of these therapies has been used to suppress antiphospholipid antibody levels.[11]

Dr Meyer: Did you not find vasculitis in any region of the body? Were the internal elastic laminae intact throughout?

Dr Mason: The elastic laminae were intact; there was no evidence of vasculitis anywhere.

Dr Furie: Is it possible that the vasculitis was healed by the time of the patient's death?

Dr Mason: In the presence of healed vasculitis, the internal elastic lamina should be disrupted and may be replaced by fibrosis. All of the arteries stained for elastic fibers in this patient showed an intact internal elastic lamina. We do not believe that vasculitis was present in this case.

Dr Furie: This patient had a very impressive quadriplegia, and yet the neurological findings at autopsy appear to be minimal.

Dr Meyer: And his electromyogram showed essentially no function at all.

Dr Mason: We could not find evidence of peripheral nerve injury, and the anterior horn cells of the spinal cord and the spinal nerve roots were unremarkable.

Dr Meyer: Could that be the result of a sampling error?

Peter Farmer, MD (Neuropathologist, North Shore University Hospital): Of course, that is always possible. It is possible that there were small nerve branches to the muscles, which were abnormal. However, our study failed to show any abnormalities.

Dr Mercurio: The antiphospholipid antibody test was only minimally positive, there was no abnormality of the PTT time, and the VDRL was negative. So, I am uncomfortable with the diagnosis of antiphospholipid syndrome.

Dr Meyer: What is your opinion in that matter, Dr Furie?

Dr Furie: I'm not comfortable either. I think that the absence of coagulation abnormalities and of the other serological criteria already pointed out makes it difficult to establish the diagnosis of the antiphospholipid antibody syndrome. However, similar histologic changes have been reported in one series consisting of three patients with the primary antiphospholipid syndrome.[3] The vascular changes witnessed in this patient are rare.

We are now recognizing that there is a vast spectrum of vascular complications in SLE that may be better understood through exercises such as this one.

Peter Bluestone, MD (Associate Professor of Clinical Medicine, Cornell University Medical College): This patient had documented membranous nephropathy. Did he have proteinuria?

Dr Meyer: I believe that the evaluation at the other hospital one year earlier revealed a proteinuria of 3 g/24 hours.

Dr Mercurio: This is correct.

Dr Meyer: Certainly there is a tendency toward a hypercoagulable state in association with the nephrotic syndrome, and this may have been a contributing factor in this patient.

Dr Allen: Did we obtain an HIV antibody serological test post-mortem.

Dr Mason: No.

Elliott Chartash, MD (Assistant Professor of Medicine, Cornell University Medical College): The family resisted all our attempts to clarify his HIV status during life.

Mark Kaplan, MD (Chief, Division of Infectious Disease, Department of Medicine, NSUH; Associate Professor of Medicine, Cornell University Medical College): I would like to point out that this patient did not present the clinical picture of HIV infection. Dr Mercurio reported the abnormal T-cell subsets, but the patient had no opportunistic infections as supporting evidence of HIV disease. His terminal *Candida sepsis* is fairly common in a patient who had treatment with broad-spectrum antibiotics together with high dosages of corticosteroids. In these circumstances, fungal infections are not indicative of HIV disease.

The T-cell profile associated with steroid therapy is not exactly like the profile characteristic of HIV infection. I am reasonably certain that this patient did not have HIV infection as an explanation of his disease process. I would also like to point out that the family's permission is not necessary for a post-mortem HIV antibody test which is part of an autopsy.

FINAL ANATOMICAL DIAGNOSES

SLE with lupus nephritis WHO Class V.

Dilated cardiomyopathy with mural thrombi, arterial embolism, and infarction of multiple organs.

Venous thrombosis involving the popliteal, iliac, and axillary veins bilaterally, with pulmonary embolism.

End-stage muscle disease involving upper and lower extremities.

Bacterial pneumonia, polymicrobial sepsis, and disseminated candidiasis.

REFERENCES

1. Feinglass EJ, Arnett FC, Dorsch CA, et al: Neuropsychiatric manifestations of systemic lupus erythematosus: Diagnosis, clinical spectrum, and relationship to other features of the disease. *Medicine* 1976; 55:323–339.

2. Jain R, Furie R, Chartash E: Systemic lupus erythematosus complicated by thrombotic mircroangiopathy. *Arthritis Rheum* 1991; 34:593.

3. Alarcon-Segovia D, Cardiel MH, Reyes E: Antiphospholipid arterial vasculopathy. *J Rheumatol* 1989; 16:762–767.

4. Bukowski RM, King JW, Hewlett JS: Plasmapheresis in the treatment of thrombotic thrombocytopenic purpura. *Blood* 1977; 50:413–417.

5. Bukowski RM, Hewlett JS, Harris et al: Exchange transfusions in the treatment of thrombotic thrombocytopenic purpura. *Semin Hematol* 1976; 13: 219–232.

6. Rock GA, Shumak KH, Buskard NA, et al: Comparison of plasma exchange with plasma infusion in the treatment of thrombotic thrombocytopenic purpura. *N Engl J Med* 1991; 325:393–397.

7. Bell WR, Braine HG, Ness PM, et al: Improved survival in thrombotic thrombocytopenic purpura—hemolytic uremic syndrome: Clinical experience in 108 patients. *N Eng J Med* 1991; 325:398–403.

8. Korec S, Schein PS, Smith FP, et al: Treatment of cancer-associated hemolytic uremic syndrome with staphylococcal protein A immunoperfusion. *J Clin Oncol* 1986; 4:210–215.

9. Mittelman A, Bertram J, Henry DH, et al: Treatment of patients with HIV thrombocytopenic and hemolytic uremic syndrome with protein A (Prosorba Column) immunoadsorption. *Semin Hematol* 1989; 26:15–18.

10. Case Records of the Massachusetts General Hospital, Case 11–1990. *N Eng J Med* 1990; 322:754–769.

11. Case Records of the Massachusetts General Hospital, Case 37–1988. *N Eng J Med* 1988; 319:699–712.

FROM THE LIBRARY

THE EFFECT OF CORTISONE AND ADRENOCORTICOTROPIC HORMONE (ACTH) ON DISSEMINATED LUPUS ERYTHEMATOSUS AND THE MESENCHYMAL TISSUES (Abstract)

The beneficial effects of cortisone and pituitary adrenocorticotropic hormone (ACTH) on patients with rheumatoid arthritis have been amply confirmed and extended to a variety of connective tissue or mesenchymal diseases. In three cases of disseminated lupus erythematosus treated with ACTH such beneficial effects occurred as prompt disappearance of fever and pain, decrease in inflammatory reaction of serous surfaces, general improvement in well-being, and a return to normal of such abnormal laboratory findings as hyperglobulinemia, elevated erythrocyte sedimentation rate, and positive cephalin flocculation. One of the patients developed hypertension, and treatment was discontinued following a subarachnoid hemorrhage. Another patient developed parotitis and parotid abscess requiring incision and drainage; no granulation tissue was noted for three weeks, but the wound healed well when ACTH was discontinued. The third patient suffered a fatal pulmonary infarction while under treatment. In the two patients who survived, symptoms returned promptly when ACTH was withdrawn, and both subsequently died.

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J. WALLACE BLUNT, MD; KARL MEYER, MD; CHARLES RAGAN, MD
(*NY State J Med* 1950;50:2446)

Smallpox in an 18th-century Italian princess

SAUL JARCHO, MD

In the spring of 1991, the author received, through the exemplary courtesy of the Archivio di Stato of Modena, the microfilm of a collection of documents (Estense 1223 = alpha. N.8.II; protocollo 2070 [v 9]) related to the activities of Dr Francesco Torti (1658–1741), professor of medicine at the college of Modena, physician to the ducal family of Este of the Modenese principality, and famous for his successful treatment of pernicious malaria, described in his *Therapeutice Specialis*.[1]

The microfilm, in an enlargement prepared by Mr Luciano Otero of the New York Academy of Medicine, reproduces approximately 71 pages of text. An exact count of the original documents cannot be given, since some pages represent covers which can be assigned with equal probability either to succeeding or preceding items. The documents themselves bear no pagination; page numbers were therefore inserted during the present study and appear in annotations in the single text herein translated. As might be expected, the pages differ in clarity. Many have been blurred, as if they had been used as blotters. Marks of punctuation have not been reproduced faithfully in the microfilms.

All of the texts are in Italian except for the single document herein reproduced, which is in Latin and is not an original but a copy of the official report, made by three consultants, of the case of a princess afflicted with smallpox. It has not been possible to ascertain her identity with certainty.

A provisional copy of the entire collection of manuscripts, transcribed line-for-line, is being deposited at the National Library of Medicine. The microfilm of the collection, the expanded positive prints, and a xerox copy of the entire transcript, are being deposited in the library of the New York Academy of Medicine. Gracious permission to publish the text of the report has been received from the Archivio di Stato of Modena.

In the following translation the complicated sentences, customary in Italian literature of this period, whether written in Latin or in Italian, have been preserved without comminution. An impressive, if not overpowering, example will be found in the translation of pages 66–67.[2] Several hypertrophied paragraphs have been divided.

COPY OF THE MEDICAL REPORT OF TORTI, DAVINI, AND ALBERTINI ON THE ILLNESS OF THE PRINCESS OF MODENA[2]

Merely summarizing the history of the Most Serene Princess of Modena, we go to the sixth day. The beginning of the illness occurred in like manner on the sixth day of the present month of September, with a mild exacerbation of fever, followed a short while afterward by a fluid bowel movement which resembled those that preceded it. At the same time the menses were suppressed; this caused internal heat, heaviness of the head, and difficulty in breathing. For these symptoms alleviation was soon produced by water of artemisia, the cordial water of Saxony, and the hysteric water of Quercetanus, administered in turn. Finally, for the hours of the night a little diascordium was given and the volatile salt of hartshorn was thereupon suspended immediately. The next morning, after the patient had a quiet sleep and a bowel movement, the fever remitted. The menses again flowed moderately and pocks (variolae), quite tiny,[2(p62)] numerous, and elevated, appeared. Very many of them were found to be entirely translucent and filled with fluid.

THE SYMPTOMS INCREASE

At the start of the seventh day, in the afternoon a mild exacerbation of the fever ensued. The Most Serene Princess tolerated this easily; her menstruation progressed as usual and everything else went on quietly enough. Nevertheless, to preserve expulsion, especially through the skin, a little diascordium was given at bedtime, with a few grains of a bezoardic. During the course of the night, blood flowed from her nostrils, approximately in the amount of an ounce and a half. By coincidence this happened when she herself was cleaning her nostrils rather vigorously. She had even attempted this earlier and had used a little force to keep them open. A little while later[2(p63)] she had a quiet sleep. The pulse was high but somewhat frequent, and the menses continued properly. In the morning the pocks began to turn white, with a trace of maturation. The same fever, a fuller pulse, and commendable transpiration, ended the seventh day.

In the evening everything was the same: moderate increase in fever, dwindling menstruation, no additional diarrhea. The night was restless but sleep in the morning compensated for the preceding harmful insomnia. The pocks from now on became whiter and coarser. The urine was of good quality. About midday the pulse was a little slower and transpiration was free. Menstruation and the eighth day ended.

A little after midday, the ninth brought on a little restlessness[2(p64)] because it was painful and difficult for the patient to lie on the pocks. The fever was hardly greater. The urine was more abundant, with somewhat rough coarseness. There was no further bowel movement since the sixth day. Toward nightfall, lest matter in the vessels, having begun to be disturbed and agitated again, should turn away from the skin, the usual offering of a bolus was repeated with diascordium and bezoar stone. This caused not the slightest difficulty, and not long afterward the bowel moved abundantly twice. The remainder of the night brought not sleep but tossing and increased fever. Mean-

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while, the pocks were observed to be subsiding somewhat and to be drying. In the morning profuse diarrhea occurred three times. The new day likewise had come to a disappointing end.

The Fastigium

In a similar manner the tenth began unhappily, the coarse diarrhea having persisted.[2(p65)] It was much more frequent and abundant, and the urine was thin and altogether watery. The pocks, especially those on the face, were satisfactorily dry, and had almost disappeared. The few that remained were less vesicular. To correct the very serious symptom of profuse diarrhea and at the same time to recall matter to the domain of the body, and indeed, to repress by some soothing aid both the restless tossing and a certain inclination, already evident, toward delirium, a bolus was administered. It consisted of extract of tormentilla root, with a few grains of fresh bezoartic powder and half a grain of opiate laudanum. A little later, the subdelirium, which had been indistinct, declared itself clearly. It did not last long but was quieted by the arrival of a short sleep.

Although the fever burned excessively and the other symptoms worsened, her mind[2(p66)] remained so clear and acute that at about the fourth hour of the night the Most Serene Patient was able to receive the sacred rite of the Eucharist with a holy and commendable greatness of spirit such as drew tears from everyone and at the same time could cause no pain but could console sufferers. Since no tendency to sleep supervened but instead the agitation persisted, in order to produce a degree of tranquility an emulsion was prescribed. It was made from seeds of melon, white poppy, and fresh almonds, and was taken at about the eighth hour of the night. Sleep was awaited in vain, and uninterrupted jactitation lasted into the morning. Brief disturbance by phantasms occurred before daybreak.

A Decision

Therefore, when morning had come, since the fever, as was usual at that time, had declined somewhat from its maximum, and the diarrhea had abated from its greatest frequency,[2(p67)] and since in addition a new and perhaps more severe attack of the same symptoms was feared as the eleventh day approached, because the dregs of what was leaving the skin of the face were evidently fluctuating and boiling irregularly in the vessels, for these reasons, in order to prevent some violent calamity and to restrain the febrile effervescence—since the expulsive movement toward the skin was now complete—in preference to the other measures which were reviewed, a venesection in the foot was approved unanimously, for reasons that are valid in circumstances of this kind and are not trivial.

However, because the Most Serene Patient was not assenting with unmistakable heartiness to the aforementioned operation, nor for that reason was it very readily considered easy to omit—at least as a freer will might perhaps permit it during the persistence of indications[2(p68)] for a remedy of this kind—and since in the rest of the body, except the face, the pocks even now appeared to be retaining some moist softness, for this reason and also, in order to maintain such transpiration and to promote it more abundantly in whatever manner was wished, instead of removal of blood its further purification and invigoration were chosen, by substituting, in place of the proposed operation, merely a compound made from a few grains of the volatile salt of vipers and the confection of hyacinth, to be taken at the proper intervals. By its use, begun immediately, the Most Serene Princess became noticeably quieter, and at last the tenth day had its allotted end.

When the evening of the eleventh day arrived, the exacerbation of the fever was somewhat milder. The diarrhea was scantier and its color was less tawny, perhaps because abundant drink[2(p69)] had caused dilution. The urine, in contrast, was more copious but not always constantly thin or crude. Toward nightfall the aforementioned bolus was repeated, with the addition of a paregoric and mildly thickening emulsion, to be taken as needed for the throat if it became dry in the course of the night. The latter was more tranquil, since sleep and rest were often interspersed, although the fever displayed a certain vigor, with a barely favorable pulse accompanied by mental alertness and retention of bodily strength.

Defervescence

At the height of the morning the fever was found to have diminished and all the symptoms were correspondingly milder. Therefore, going to the end of the eleventh day (we found that) there was light moisture over the entire skin and, appropriately, a sticky desiccation of the pustules which besieged the rest of the body [sic]. At the same time, since the urines were favorable and the[2(p70)] diarrhea and thirst had abated, the same eleventh day began to unfold with the happy ending of sorrow and fear.

The twelfth day had the same tenor. Although the evening exacerbation certainly was not absent, it was mild and was followed by a quiet sleep during the night and by an even more tranquil morning. The remission extended to noon.

The course of the thirteenth day was better also; it lacked all exacerbation and to a great extent it shook off the old ferment itself. Sleep in the daytime and at night, and uninterrupted transpiration.

By the grace of God the extremely favorable fourteenth day brought almost complete resolution of the illness. Perchance a happier time had not been seen, up to the present,[2(p71)] by this court, or this city, or the entire realm of the Este dynasty from the Apennines to the Po.

Francis Torti
John Baptist Davini

Comment on the Text

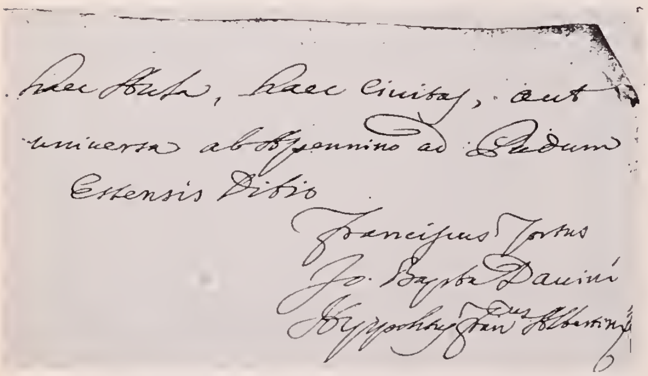
The statement opens with part of an archivist's note (Fig 1). Unfortunately, the final words of his annotation do not appear in the expanded microfilm. Neither the patient's name nor the year is stated, either here or in any other part of the document.

It is possible to guess that if the illness of the princess was part of an epidemic outbreak, it occurred in 1717, since the valuable topographic and chronological index in Corradi's *Annali Delle Epidemie Occorse in Italia*[3] shows that outbreaks of smallpox occurred in Modena in 1692, 1717, and 1737. In 1692, Albertini's doctoral diploma was only three years old. By 1737, Davini had been dead for four years. Hence, the probabilities favor 1717. If this be the case, it is probable that the princess may have been one of the three daughters of the ruling duke, Rinaldo III (1655–1737) and his wife, Charlotte Felicitas of Brunswick-Lüneburg who died in 1710. These daughters included two spinsters, Princess Benedetta Ernestina (1697–1777) and Princess Anna Amalia Giuseppa (1699–1778). A third daughter, Princess Enrichetta Maria (1702–1777), married twice, first in 1728 and again in 1740.[4]

FRANCISCUS TORTI MUTINEN. PHI. & MED. DOCT.

FIGURE 1. Francesco Torti (1658–1741). Professor of Medicine at the College of Modena. (From his *Therapeutice Specialis*, ed 2, Modena, 1730 [slightly retouched]. Engraving by F. M. Francia.)

The names of the three physicians appear at the end of the report. The first, the famous Francesco Torti, has been introduced on the first page of the present analysis. The second, less illustrious, Giovanni Battista Davini, served as archiater to the Duke of Modena, and died in 1733. The third, Ippolito Francesco Albertini (1662–1738), was the nephew of Malpighi, a teacher of Morgagni, and professor at the University of Bologna. A clinician of extraordinary ability, he is famed for his essay *Observations on Certain Diseases that Produce Difficulty in Breathing and Are Caused by Structural Damage of the Heart and Precordia,* presented in 1726 and first published in 1731 (Fig 2).[4,5]



haec Aula, haec Ciuitas, aut
uniuersa ab Appennino ad Padum
Estensis Ditio
Franciscus Tortus
Jo. Bapta Davini
Hyppolitus Fran. Albertini

FIGURE 2. Final page of the report (secretarial copy): *haec Aula, haec ciuitas, aut uniuersa ab Apennino as Padum Estensis Ditio* (this court, this city or the entire realm of the Este dynasty from the Apennines to the Po), followed by copies of the three consultants' signatures (courtesy of the Archivo di Stato Modena, Modena, Italy).

The physicians' report mentions in its second sentence[2(p61)] a fever which can be interpreted as that which commonly precedes the eruption of smallpox. It indicates also that the patient had diarrhea previously but it does not state whether this was chronic. The next sentence mentions "suppression of menses" which was alleged to have caused internal heat, since it was thought that noxious menstrual products were being retained within the body.

For the menstrual suppression three medicines were prescribed. The first, water of artemisia, is listed in the *Antidotarium of the Medical College of Bologna* as an emmenagogue.[6] The second, designated in full as *Aqua cordialis temperata Saxoniae*, is a mixture of 24 ingredients, such as borage, bugloss, rue, and myrrh, and is credited to one Hercules of Saxony. A cordial (ie, a medicine for the heart) it was advocated in the words of the *Antidotarium*, "for faintness of the soul and sudden terrors." The third medicine, the hysteric water of Quercetanus, contains only ten ingredients, such as musk, cinnamon, aloes, and mace. Its name is derived from the Greek *hystera*, the womb, and points to the ancient belief that a host of symptoms, many of which are now regarded as circulatory, spastic, or psychic, emanated from the uterus.

For use at night the patient was given the diascordium (electuary) devised by Girolamo Fracastoro, famed for his writings on syphilis and on infection; it included scordium, styrax, cinnamon, gum arabic, and ten other ingredients. According to the aforementioned *Antidotarium,* this mixture was to be prescribed in malignant, putrid, and pestilential fevers.

An interesting feature of the report is its description of the rash in various stages. When the eruption was first noticed, on the sixth day of September, many of the lesions were already vesicular or papulovesicular. Their distribution is not described. No macular or maculopapular phase is mentioned, nor is anything said about the appearance of the mouth or throat. Subsequent statements report: incipient whitening of the pocks, with traces of maturation[2(p63)] on the seventh day; increased whitening, accompanied by thickening[2(p63)] on the eighth day; incipient subsidence of the pocks, and incipient drying,[2(p64)] on the ninth day; dryness and partial disappearance of pocks on the face, while pocks elsewhere were less vesicular than they had been [2(p68)] on the tenth day; and drying of residual pocks[2(p69)] on the eleventh.

Mention of "the present constitution"[2(p62)] refers to the composition of the atmosphere and climate, here alleged to be responsible for difficulty or delay in resolution of the pocks. This concept of constitution is prominent in the writings of Sydenham and is well known to readers of the corpus Hippocraticum.

On the afternoon of the seventh day,[2(p67)] there was a slight exacerbation of fever. Although instruments had been devised in the 17th century for measurement of body temperature, clinical thermometers such as are now familiar were not often used before the 19th century. At the time of the report that is here under discussion fever was judged by palpation of the skin, by observation of sweating, and especially by palpation of the pulse. It should be noted also that the word *fever* had two meanings, increase in body heat, and a disease in which bodily heat is increased.

For the modern reader and translator the interpretation of the report is made slightly difficult by the chronology. It appears probable, but not absolutely certain, that the scribe and the physicians reckoned in accordance with a tradition described as follows in volume 1, page 521 of the *Cambridge*

Italian Dictionary[7] (under the word ora):

ore italiane, unequal hours on the old 24-hour system ending at sunset instead of midnight.

A more extensive definition appears in the second edition of *Webster's New International Dictionary*,[7,8]

Until the 18th century the hour was commonly reckoned as the twelfth part of the time between sunrise and sunset, or between sunset and sunrise, and hence was of varying duration.

Throughout most of the report it is noticeable that fluctuations in the patient's condition were paralleled by her ability to sleep. On the night of the seventh,[2(p63)] she slept quietly. On the night of the eighth,[2(p63)] her sleep was intranquil, but compensatory repose was obtained in the morning. On the afternoon of the ninth,[2(p63,64)] she had pain, caused by lying on the pocks. During the night, she had no sleep, but she tossed about, and her fever increased.

On the tenth, because of aggravation of her pre-existing diarrhea (which apparently had begun before the period of the present record), and also because of continued jactitation and a previous tendency to delirium, she was given a bolus which included laudanum. The subdelirium did not last much longer, but yielded to a brief sleep.[2(p65)] The fever and other symptoms increased but the patient's mind was clear, and at about the fourth hour of the night the sacraments were administered. The patient now being sleepless and agitated, an emulsion containing white poppy was given. Shortly before daybreak, there was "perturbation from fantasies" (*phantasmatum perturbatione*).

By morning, the fever had declined, as was usual at that time of day, and the diarrhea was somewhat milder,[2(p67)] but a renewed attack of these symptoms was feared, since the residues of the material that was departing from the skin of the face were believed to be still fluctuating and boiling irregularly in the vessels. Therefore, to prevent catastrophe and control the febrile effervescence, an entity prominent in the writings of Thomas Willis, the physicians unanimously decided that the patient should be given a phlebotomy (from the foot!) in preference to other treatments.

This decision was later annulled, in part because over most of the body[2(p68)] the pocks were still somewhat soft and moist. To preserve this condition a little longer and encourage free transpiration, additional purification and strengthening of the blood were deemed desirable. To attain these ends, the volatile salt of viper and the compound confection of hyacinth were administered, apparently in the form of a bolus. According to the Bologna pharmacopoeia of 1800, the first of these drugs, because of its alkalinity, was useful to counteract acid diseases of the humors. The second, an extremely complex product, is listed in the same compilation as a "cardiac confection." As soon as she started to take these medications, the patient became more tranquil, and the tenth day reached its end.

In the subsequent evening, the usual elevation of fever was noted to be smaller and the diarrhea was scantier, but the urine[2(p69)] was more abundant. The bolus was repeated and a paregoric and gently thickening emulsion was added to relieve dryness of the throat. In consequence, the night proved relatively quiet. The princess slept at intervals, but the fever was not lacking in vigor, nor did the patient lack alertness.

By the height of the morning, the fever had declined and all symptoms were reduced proportionately. By the end of the 11th day the skin was slightly moist, the remaining pocks were somewhat dry, the urine was "laudable," and diarrhea and thirst were mild. Sorrow and fear were ending—presumably for the patient, her noble kinsmen, the conscientious physicians, and the attendants.

The 12th day[2(p70)] was favorable likewise. A mild exacerbation in the evening was followed by quiet sleep at night and continued remission in the morning. The 13th day, even better, produced no exacerbation, since the ferment, now old, had been largely dispelled (*ipsum fermentum vetus maxime ex parte discussit*). The patient slept during the day and the night and had continuous sweating. The 14th day was joyous to the court and realm of the Este dynasty.

CONCLUSION

It is appropriate to consider what the reader may gain from the consultants' report. The patient had smallpox. It was uncomplicated except for diarrhea, which seems to have been present previously. The smallpox terminated favorably after a course which was rather brief but did not lack intensity. The period of observation and report lasted only nine days—from the sixth day of the month until the 14th, and the latter brought almost complete resolution (*solutionem denique morbi Deo dante jam ferme integram invexit*). Most physicians in our century would have continued surveillance until desquamation was farther along and would have commented on the extent of the scars. However, by the 14th day of the month, recovery being well advanced, it may have been felt that the presence of eminent consultants was no longer necessary and detailed reporting could be discontinued. Most modern consultants would have mentioned isolation and other sanitary precautions. It is not impossible that the patient's exalted rank exempted her from the restrictions.

The modern reader, especially if a physician, will have noted that the report lacks anything like a complete clinical history and a physical examination. Neither was usual in the 18th century, but in our time a consultant's note not rarely summarizes both of these components. The appearance of Her Highness' mouth and throat is not described, and nothing is reported about her abdomen, although she had diarrhea. In contrast, the urine and stool are described repeatedly.

The positive features of the report are numerous. The changes in the skin are described regularly and minutely. The patient's psychic state is recorded repeatedly, and the intensity of the illness is reported daily, without recourse to clinical thermometers.

Emphasis was placed on the treatment of individual symptoms. In this connection, it is interesting that an attempt was made to keep the skin lesions soft by means of internal remedies;[2(p68)] no mention is made of topical treatment.

One of the most impressive features is the detailed description of the reasoning that governed the treatment, especially the decision to perform phlebotomy,[2(p67)] and the subsequent reversal of that decision.

Also of interest, is the contribution made by different centuries, from the Hippocratic clinical outlook and the ancient notion of menstrual suppression to the 17th-century Willisian fermentationism; the present day social historian would not fail to observe that a princess of the house of Este had at her command three professors of medicine, at least two of whom gained enduring fame and one of whom was brought from Bolongna. Necessarily a detailed report was rendered, and it was kept in the official archives, where Italian courtesy continues to make it accessible.

ACKNOWLEDGMENTS

This paper has been written and contributed as a compliment to the editorial achievements of Dr Pascal James Imperato. The research was assisted by the National Library of Medicine (LM-04505).

REFERENCES

1. Torti F: *Therapeutice Specialis ad Febres quasdam Perniciosas.* Modena, Soliani, 1712.
2. Copy of the medical report of Torti, Davini, and Albertini on the illness of the Princess of Modena, Estense 1223 = alpha. N.*.II; Protocollo 2070/v.9.
3. Corradi A: *Annali delle Epidemie Occorse in Italia.*, Bolongna, Gamberini and Parmeggiani, 1865–1894, 8 vols, Facsimile, Bolonga, Forni, 1972–73, vol 5, p531.
4. Burke: *Royal Families of the World* vol I. Europe and Latin America, London, Burke's Peerage Ltd., 1977 pp334–335.
5. Albertini IF: Animadversiones super quibusdam difficilis respirationis vitiis a laesa cordis et praecordiorum structura pendentibus, *De Bononiensi Scientiarum et Artium Instituto atque Academia Commentarii* 1731; 1:382–404 (reprinted 1748).
6. *Antidotarium Colegii Medicorum Bononiensis,* Venice, Orlandelli, 1800, p57.
7. Reynolds B (ed): *Cambridge Italian Dictionary*, Cambridge University Press, 1962–1981, vol 1, p521.
8. *Webster's New International Dictionary of the English Language,* ed 2, unabridged. Springfield, Merriam, 1946, p1206.

FROM THE LIBRARY

SMALLPOX

Smallpox is one of the oldest diseases known; it existed in China and Hindustan more than 1,000 years before the birth of Christ. Pursuing the track of armies, we find it raging in Egypt in 640, and subsequently following the victories of the Saracens in the eighth century through Italy, France and Spain. By the Saracens it was communicated to the Crusaders, and the latter caused its rapid spread throughout Europe. There was no smallpox in America prior to its discovery by Columbus, 1492. In 1517 the disease was imported into St. Domingo. Three years later, in one of the Spanish expeditions from Cuba to Mexico, a negro covered with the pustules of smallpox was landed on the Mexican coast. From him the disease spread with such desolation that, within a short period, over three million and a half of people were destroyed in that kingdom alone.

It was introduced into Iceland in 1707, when 1,600 persons were carried off by its ravages; or more than one-quarter of the whole population.

It reached Greenland still later, appearing there for the first time in 1733, and spread so fatally as to almost depopulate the country.

The southern countries of Europe furnish the largest annual contingent to the total mortality from smallpox, Rome showing, in 1887, a rate of 35 per 1,000, and Lisbon, 65 per 1,000. Rio Janeiro, for 1887, shows a mortality of 22 per 100 deaths from all causes. Prior to vaccination, it was universally regarded as the greatest scourge to mankind and was estimated as causing one-tenth of all the deaths among the human race.

The cause of smallpox is a specific animal poison. In the winter, '98 and '99, our country was visited in various parts of it, almost simultaneously, by mild epidemics of smallpox; the cause of such invasion was undoubtedly due to the return of our American soldiers from countries infected with that disease.

The disease invading localities in a mild form only, followed its usual course in all the older countries, and in its previous attacks upon this country. The history of these attacks has always been in a mild form, gradually assuming severer forms as it becomes acclimated, or growing in virulence as it passed from one culture media to another from year to year.

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(*NY State J Med* 1903;03:401–402)

Medical futility

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Whether treatment for irreversibly ill or dying patients should be continued or withdrawn is sometimes a matter of disagreement between the patient's physicians and the patient's family. In most instances, the family wants to forego or stop the treatment, and the physicians want to continue it. The recent Wanglie case in Minnesota, illustrates the reverse situation and has catapulted the issue of medical futility to center stage.[1]

Helga Wanglie was an 86-year-old woman who was in a coma and required a respirator to survive. Her physicians and hospital administrators tried to convince Mr Wanglie that his wife's condition was irreversible, that further treatment was futile and that the respirator should be removed. Mr Wanglie insisted that his wife had expressed, on several occasions, her wish that all available treatments be used to sustain her life. To break the deadlock between the physicians and the family, the hospital petitioned the Court to appoint an independent conservator as guardian to make medical decisions for the patient. The petition was denied. Mrs Wanglie died in July 1991 while still attached to the ventilator.

Several months later, a court ordered an Atlanta children's hospital to continue providing life-sustaining treatment for a teen-age girl (identified only as Jane Doe) with an irreversible neurological disorder, even though the physicians considered the care futile and abusive. The girl's father was unalterably opposed, on religious grounds, to the withdrawal of life-sustaining measures, and hoped and prayed for a miracle. The girl died eight days after the court ruling.

More cases of this nature are certain to arise and will require careful and compassionate consideration. When is a treatment medically necessary and when is it medically futile? The distinction cannot always be made with clarity. Although most physicians appreciate the concept of futility, few can provide an operational definition.[2] The various interpretations of futility have recently been summarized.[3] The term futility may express the degree of probability of achieving a specified outcome; but physicians differ widely in their interpretations of terms such as rare, infrequent, and common. The term futility, too, does not express a discrete and identifiable quantity but rather encompasses a range of probabilities and is likely to be interpreted in different ways by different physicians. Determinations of futility may also vary on the basis of the perceived objectives of medical treatment and the criteria used to evaluate outcome. Futility may also be based on criteria that relate to the anticipated length of the patient's survival.

Is it medically futile to implant a second or third heart valve in an intravenous drug addict who continues to abuse drugs? Is it medically futile to hemodialyze a patient in a persistent vegetative state? Is it medically futile to transplant a liver into an alcoholic patient who continues to drink? Is it medically futile to offer mechanical ventilation to a patient with acquired immunodeficiency syndrome (AIDS) with a third episode of *Pneumocystis carinii* pneumonia? Is it medically futile to give a bone marrow transplant to a woman with metastatic advanced breast cancer resistant to chemotherapy? Is it medically futile to provide high technology and/or very expensive care to a terminally ill patient with an irreversible or incurable disease with a life expectancy of only a few weeks?

The question of whether or not a medical intervention is futile can be logically divided into two parts: What is the goal of the physician in performing the intervention and what is the chance of achieving that goal? There are many legitimate goals of medical treatment. Medical treatments are not only directed at curing illness or prolonging life, but may also be directed toward reducing pain or discomfort such as the administration of pain medication to a patient with a terminal illness. Other examples of medical interventions in terminal patients which are not futile but are also not curative include the administration of oxygen, or even the transfusion of blood products. Such interventions are not futile if they aid in reducing the patient's discomfort such as in relieving a patient's weakness or shortness of breath. Other actions may be done with the goal of preserving the patient's dignity. Even permanently comatose patients must be given good nursing care, cleaned, and cared for. Actions intended to give comfort to the patient's family, such as administration of oxygen by nasal canula to a comatose patient, are also legitimate and reasonable, and are not futile. Likewise, the feeding of a terminal patient, in addition to making him feel better, may comfort the family in that they feel that the patient is being cared for.

The second part of the question "Is a medical intervention futile?" that is "Is there a real chance of achieving the desired end?" suggests a more difficult philosophical problem, especially when the medical intervention involved is a

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Prepared as an educational and informational report by the Committee on Bioethical Issues of the Medical Society of the State of New York.

complex one. For example, at what point do we determine when a medical intervention such as surgery or radiation therapy has no real chance of achieving the goal of prolonging a patient's life. When is a therapy futile? Do we define it as when there is little chance of achieving a goal, no chance of achieving that goal, or no statistically significant chance? Physicians are constantly making these subjective judgments of risk and possible benefit and there is no precise formula for answering this question. A national debate is currently underway attempting to define medical futility. Discussions may identify situations where the physician can morally and legally withhold therapy considered to be medically futile even if the patient or family disagree with the medical recommendation. The Committee on Bioethical Issues believes that a therapy is not futile if there is a real chance of achieving some desirable end, whether that end is cure of the patient, patient comfort, patient dignity, or even comfort to the family. Whether or not there is a real chance of achieving any of these goals must be left to the patient's physician to determine in the individual case.

THE NEW YORK STATE RESUSCITATION LAW

On April 1, 1988, New York State enacted a law governing the withholding of cardiopulmonary resuscitation (CPR). Known as New York's DNR (do not resuscitate) law, it can be summarized as follows:[4] Every patient is presumed to consent to CPR unless a DNR order has been issued. If the patient has decision-making capacity, the attending physician must obtain the patient's consent in the presence of two witnesses before writing the DNR order in the patient's chart. If the patient lacks decision-making capacity, two physicians must certify in writing the cause and probable duration of the patient's incapacity and the reason for the DNR order. Notice of this determination must be given to the patient and the surrogate. If the patient understands and objects to the DNR order, it will not be written. The surrogate may consent to the DNR order only after two physicians certify in writing that the patient has a terminal condition or is permanently comatose or that CPR would be medically futile or that it would impose an extraordinary burden on the patient. The decision about CPR or DNR must be based on the patient's wishes, including religious and moral beliefs or, if the patient's wishes are not known, on the patient's best interests. In the absence of a surrogate, a DNR order may be written if two physicians certify in writing that CPR would be futile. For acute care hospitalizations, the DNR order must be reviewed every three days.

The New York State DNR law requires physicians to give futile therapy if the patient or surrogate do not consent to a DNR order. Although the law has not changed the frequency of CPR, it has increased discussion about CPR and DNR between physician, patient, and family.[5]

THE DNR ORDER, CPR, AND MEDICAL FUTILITY

Is the DNR order solely an ethical and legal imperative or should it be a medical decision? Snider posits that although the DNR order has legal and procedural aspects which must be adhered to, this area is primarily the purview of physicians, not lawyers.[6] Physicians should first decide whether it is medically appropriate to use CPR. If it is not appropriate, a DNR order should be written. If CPR is appropriate, continues Snider, its benefits and potential disadvantages should be explained to the surrogate who can then accept or reject the procedure.

The American Medical Association's Council on Ethical and Judicial Affairs recently issued its updated guidelines for the appropriate use of CPR and DNR orders.[3] Consent to administer CPR is always presumed, except in two cases. First, a patient, family, or surrogate may express in advance that CPR be withheld. Second, CPR may be withheld if the physician's judgment is that it would be futile. The AMA guidelines have been criticized because of the following:

> They allow patients with conditions such as widely metastatic cancer or septic shock to insist on CPR, despite the dismal outcomes reported. According to these guidelines, the ethical principle of respecting patient autonomy requires futility to be judged according to the patient's goals. Reasonable physicians might disagree strongly.[7]

Society and the medical profession are not obligated to do everything that a patient requests.[8] Respect for patient autonomy is not an absolute principle because it may conflict with other ethical principles such as nonmaleficence and justice.[9] Certain value judgments may be made unilaterally by physicians as part of reasonable medical practice. According to Tomlinson and Brody, "the mixed messages inherent in requesting patient consent to withhold futile therapy serve to undermine rather than to enhance autonomous choice."[10] These investigators conclude that real patient interests can better be served by a broad public dialogue around judgments of medical reasonableness and medical futility, rather than concern for the form but not the substance of patient autonomy.

Hackler and Hiller decry the hospital policies that typically require CPR unless a DNR order has been written and the family has consented to it.[11] Such policies sometimes compel physicians to perform procedures that are useless or that add to the patient's suffering without corresponding benefit. These authors suggest that when resuscitation would clearly provide no medical benefit to the patient, public and hospital policy should not require that it be discussed with either the patient or the family. Other authors agree that a medical decision to withhold CPR is no different from such a decision about any other futile treatment and need not be discussed with the patient.[12–13] Most investigators, however, take the position that patients or surrogates must be informed of such decisions and kept informed of the entire management plan.[3,6,7,14–20] "The question is no longer whether we should discuss DNR orders with our patients; instead, the issue is how to do so with compassion and caring."[7] Decisions about when to withhold or limit resuscitation must be made "openly, in honest and trusting conversation between doctor and patient."[17]

MODELS FOR APPROPRIATE USE OF CPR AND DNR ORDERS

Murphy and Matchar propose a model for appropriate use of life-sustaining therapy that incorporates effectiveness, utility, and marginal costs:[2]

> If a therapy is rarely effective and rarely desirable, it is considered medically inappropriate. If the marginal cost-effectiveness ratio is inordinately high, it is considered economically inappropriate. If a therapy is either medically or economically inappropriate, it should not be automatically offered.

These authors consider their model superior to advance directives, explicit health care rationing, and futile therapy definitions based on survival predictions, since these alternatives to their model are insufficient strategies to solve the problem of inappropriate life-sustaining care. Hackler and Hiller[11] propose that physicians should be allowed a DNR order over family objections for the following reasons:

1. The patient lacks decision-making capacity;
2. The burdens of treatment clearly outweigh the benefits;
3. The surrogate does not give an appropriate reason in terms of patient values, preferences, or best interests; and
4. The physician has made serious efforts to communicate with the family and to mediate the disagreement.

The AMA guidelines have already been cited earlier in this essay.[3] Essentially, they state that efforts should be made to resuscitate patients who suffer cardiac or respiratory arrest except when circumstances indicate that administration of CPR would be futile or not in accord with the desires or best interests of the patient.

Schneiderman, Jecker, and Jansen offer a model which defines the expected gain from a treatment by the joint product of probability of success and utility of outcome.[13] Unlike decision analysis, their definition of futility treats probability and utility as independent thresholds. These authors propose the following:

> When physicians conclude (either through personal experience, experiences shared with colleagues, or consideration of published empiric data) that in the last 100 cases a medical treatment has been useless, they should regard that treatment as futile. If a treatment merely preserves permanent unconsciousness or cannot end dependence on intensive medical care, the treatment should be considered futile.

AUTHORITY OVER FUTILE THERAPY: PATIENT, PHYSICIAN, OR SOCIETY?

Callahan argues that society as a whole, not just the medical profession, should generate standards regarding medical futility.[21] Once the discussion of futility is resolved through a political process, physicians can make futility judgments on the basis of standards generated by society. This approach, continues Callahan:

> . . . does not bypass democratic participation in standard setting. It does not unilaterally declare physicians unable rightly to make any value judgments (the failure of excessive patient autonomy), nor does it leave too many such judgments in physician's hands (the failure of paternalism). . . . Physicians should, to be sure, talk with their patients, but the question at stake here is whether they need to talk about everything. If the answer is to be no, the reason should be that doctors and patients have together agreed upon the general criteria for not doing so.

Singer and Siegler also believe that futility considerations should be discussed at societal levels such as the institution, state, or country and that physicians must participate in these discussions.[22] These investigators express concern about a patient's care becoming too dependent on which physician happens to be caring for the patient. This violates the principle of justice. For these investigators, the key questions are not whether futility considerations are relevant, but rather at which level of the health care system they should come into play, who should be empowered to declare a situation futile, and what futility criteria the decision-maker should employ. Ackerman argues that judgments about futility should be made by the patient or family since these judgments are questions of values rather than technical medical questions.[1] Brock and Wartman point out that if a patient makes an irrational medical choice, it does not mean that the patient is incompetent to make that choice.[23]

Most investigators, however, do not believe that society or the patient should have authority over futility decisions.[1,21] Most believe that the physician's judgment should be the major if not overriding determinant in such decisions. Rabkin et al argue that physicians should decide about futility but only after consultation with the patient, if competent, and the family.[24] Brett and McCullough assert that for the patient's anticipation of benefit to have decisive weight in the use of a medical procedure, there must be "at least a modicum of potential benefit, as seen from the medical perspective."[8] Searle forcefully argues that "doctors are the responsible moral agents and they have a duty, after proper consultation (with the family and with colleagues) to decide these matters and should be free to do so."[25]

Tomlinson and Brody express confidence that physicians are able to apply their knowledge about the benefits and/or detriments of CPR and the biological and social principles behind that knowledge to each particular case.[10] They add that it is too much to expect patients and families to grasp complicated medical concepts fully and shoulder the burden of being the final arbiters of CPR or DNR decisions. They assert that a procedure or treatment is futile and should not be used if in the physician's judgment it offers no benefit or if it does more harm than good.

Yarborough states that the physician has a duty to explain why futile care is inappropriate and then not to give it or to withdraw it, even if the family wants it continued.[26] The physician also has a duty not to squander the limited resources of society by giving or continuing inappropriate futile therapy. Attempting CPR on patients who are virtually certain to die soon afterwards may deny scarce medical resources to patients more likely to benefit from intensive care.[27]

COMMITTEE ON BIOETHICAL ISSUES' POSITION STATEMENT

The Committee on Bioethical Issues of the Medical Society of the State of New York believes it is part of the professional duties and obligations of a physician to decide whether and when a medical procedure or treatment is futile.

The decision should take into account the likely outcome over weeks and months, not just hours or days. The decision should consider the benefits as well as potential harm of the treatment.

In making the decision, the physician should almost always involve the patient, family, or surrogates in a clear discussion of the issues. In many instances, the physician may need to consult with colleagues and/or senior consultants, or to consider involving the hospital's ethics committee. Occasionally, the physician may have to transfer the patient to the care of another physician. The decision about futile therapy cannot and should not be abdicated by the physician to the patient, family, surrogate, court, or society in general. To abdicate a decision about the futility of a procedure or medical treatment is to abdicate professional responsibility for the patient.

"Symbolic" treatment may be beneficial to the patient's family or to society, but if it is not helpful or is harmful to the patient's best interests, it should not be given. Further research is needed to identify the specific factors which determine successful outcomes or failures of certain procedures and treatments in certain groups of patients.

SUMMARY AND CONCLUSIONS

The term futile is used in many different ways. It is therefore difficult to decide whether a procedure or treatment such as CPR or hemodialysis or blood transfusion would be futile in a given case. The AMA's guidelines on the appropriate use of DNR orders state that DNR decisions should be made openly.[3] Institutions should have policies and physicians should elicit the patient's preferences about CPR. For physicians, the question is no longer whether we should discuss DNR orders with our patients; instead, the issue is how to do so with compassion and caring.[7]

Physicians should share with patients their judgment about what medicine can and cannot do. Then physicians must "make decisions about when to withhold or limit resuscitation openly" in honest and trusting conversation between doctor and patient.[17] Often CPR is an exercise in futility. The medical profession should be vested with the authority to make futility decisions if they are the product of open discussion and shared deliberation between physician and patient, family, or surrogate.

Rationing, triage, and medical futility in relation to AIDS patients require careful deliberation and consideration. What was considered medically futile five years ago for an AIDS patient may be appropriate care nowadays.[28] The need for appropriate use or non-use of life-sustaining therapy for the elderly, the terminally ill, patients with AIDS and other incurable illnesses is evident to patients, health care providers, policy makers, and the public.[2] CPR should only be administered if it is expected to confer lasting benefit to the patient. However, if 10% of elderly patients benefit from CPR in the case of out-of-hospital cardiac arrest,[29] how can one consider this procedure futile?

Although communication between physician and patient about difficult treatment limitation decisions has markedly improved in recent years, it remains a problem, largely because open dialogue with patients and families about futility is a demanding emotional and intellectual task.[16] The medical profession is charged with setting standards for the proper implementation of judgments regarding futility. It cannot and should not abrogate that responsibility to others. Decisions to withhold futile therapy must follow clinical judgments about the chances of success of the therapy, its possible harm, and an explicit consideration of the patient's desires and goals for therapy.[19]

The question is not whether physicians can, should, or must "make caring and conversation" integral to how they decide matters of clinical and social importance, but whether they will. If they do, the hour of death need no longer be anguished, angry, or lonely.[17] New York's late Senator Jacob Javits, dying from amyotrophic lateral sclerosis, said eloquently:[30]

> Birth and death are the most singular events we experience–and the contemplation of death, as of birth, should be a thing of beauty, not ignobility. Everyone must think about dying, young and old alike, although older people are at greater risk. Given the new medical technology that can sustain life even when the brain is gone, we must also think about the right to die and the need for dignity in departing life.

REFERENCES

1. Cranford RE, Rie MA, Ackerman F: Helga Wanglie's ventilator. *Hastings Center Rep* 1991; 21:23–29.
2. Murphy DJ, Matchar DB: Life sustaining therapy: A model for appropriate use. *JAMA* 1990; 264:2103–2108.
3. Council on Ethical and Judicial Affairs, American Medical Association: Guidelines for the appropriate use of do not resuscitate orders. *JAMA* 1991; 265:1868–1871.
4. Swidler RN: The presumption of consent in New York State's do not resuscitate law. *NY State J Med* 1989; 89:69–72.
5. Kramer RS, Dieck EM, McClung JA, et al: Effect of New York State's do not resuscitate legislation on in hospital cardiopulmonary resuscitation practice. *Am J Med* 1990; 88:108–111.
6. Snider GL: The do not resuscitate order: Ethical and legal imperative or medical decision? *Am Rev Respir Dis 1991; 143:665–674.*
7. Lo B: Unanswered questions about DNR orders. *JAMA* 1991; 265:1874–1875.
8. Brett AS, McCullough LB: When patients request specific interventions: Defining the limits of the physician's obligation. *N Engl J Med* 1986; 315:1347–1351.
9. Beauchamp TL, Childress JF: *Principles of Biomedical Ethics*, ed 3, New York, NY; Oxford University Press, 1989, pp 112, 113.
10. Tomlinson T, Brody H: Futility and the ethics of resuscitation. *JAMA* 1990; 264:1276–1280.
11. Hackler JC, Hiller C: Family consent to orders not to resuscitate: Reconsidering hospital policy. *JAMA* 1990; 264:1281–1283
12. Murphy DJ: Do not resuscitate orders: Time for reprieval in long term care institutions. *JAMA* 1988; 260:2098–2101.
13. Schneiderman LJ, Jecker NS, Jansen AR: Medical futility: Its meaning and ethical implications. *Ann Int Med* 1990; 112:949–954.
14. Bartholome WG: "Do not resuscitate" orders: Accepting responsibility. *Arch Int Med* 1988; 148:2345–2346.
15. Hammon J, Ward CG: Decision not to treat: "Do not resuscitate" order for the burn patient in the acute setting. *Crit Care Med* 1989; 17:136–138.
16. Youngner SJ: Futility in context. *JAMA* 1990; 264:1295–1296.
17. Scofield GR: Is consent useful when resuscitation isn't? *Hastings Center Rep* 1991; 91:28–36.
18. Falk RH: The death of death with dignity. *Amer J Med* 1984;77:775–776.
19. Lantos JD, Singer PA, Walker RM, et al: The illusion of futility in clinical practice. *Amer J Med* 1989; 87:81–84.
20. Kjellstrand CM: Who should decide about your death. *JAMA* 1992; 267: 103–104.
21. Callahan D: Medical futility, medical necessity: The problem without a name. *Hastings Center Rep* 1991; 21:30–35.
22. Singer PA, Siegler M: Advancing the cause of advance directives. *Arch Int Med* 1992; 22:22–24.
23. Brock DW, Wartman SA: When competent patients make irrational choices. *N Engl J Med* 1990; 322:1595–1599.
24. Rabkin M, Gillerman G, Ricer NR: Orders not to resuscitate. *N Engl J Med* 1976; 295:364–366.
25. Searle JF: Not to be resuscitated. *Anaesthesia* 1985; 40:617–618.
26. Yarborough M: Continued treatment of the fatally ill for the benefit of others. *J Am Geriatrics Soc* 1988; 36:63–67.
27. Englehardt HT, Rie MA: Intensive care units, scarce resources and conflicting principles of justice. *JAMA* 1986; 255:1159–1164.
28. Zoloth-Dorfman, L. Carney B: The AIDS patient and the last ICU bed: Scarcity, medical futility and ethics. *QRB* 1991; 17:175–181.
29. Longstreth WT, Cobb LA, Fahrenbruch CE, et al: Does age affect outcomes of out of hospital cardiopulmonary resuscitation? *JAMA* 1990; 264: 2109–2110.
30. Javits JK: Life, death and human dignity. *N Y Times* August 18, 1985, pE19.

The Patient Self-Determination Act and patient-physician collaboration in New York State

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The Patient Self-Determination Act (PSDA) which became law on December 1, 1991, requires that hospitals, nursing homes, health maintenance organizations, and home health agencies receiving Medicare or Medicaid funding provide patients with information about advance directives.[1,2] This legislation places the burden of compliance on institutions.[3] Organized medicine argued for this institutional emphasis to spare individual clinicians further regulation.[4] Although the rationale for this approach may be appealing, physician counselling in the primary care setting has recently received wide endorsement.[5]

Patients look to their physicians for guidance in end-of-life decisions.[6] If they discover that physicians have delegated this important task, this effort to protect the clinical encounter against further third-party intrusion could undermine physician authority. It is indeed paradoxical that physicians would knowingly bequeath a responsibility which so informs the basis of their clinical discretion while bemoaning their loss of professional autonomy.[7]

If the PSDA is viewed as yet another bureaucratic hurdle which impedes patient care, we will miss an important opportunity to address our patients' needs and our own professional malaise. As a vehicle to promote meaningful discussion about the goals of medicine and personal choice, the PSDA could ideally prompt physicians to assume the consultative role patients expect and to which we should aspire.

Although physician participation in discussions about advance directives might ultimately serve to reinvigorate the standing of the profession, the goals of the PSDA itself are unlikely to be realized without physicians appreciating why they should value patient self-determination. This is especially important because both New York State and Federal law place minimal obligations on practitioners and because physicians might understandably contend that their time might be better spent meeting other health care needs.

The basis for accepting this added responsibility is our respect for patient autonomy and our acknowledgement of a person's right to determine his/her future. This essential truth is based on the argument that a patient should not be deprived of the right to make important decisions because no one has a greater stake in those decisions than the patients themselves. Beyond this idea of self-interest is our society's basic philosophy that persons should not be deprived of liberty without cause and that being a patient does not constitute sufficient cause. This is a critical warrant for self-determination because it implies that persons should never be forced to submit to decisions or values which are not of their own choosing.

Although this doctrine of self-determination may seem foreign in the guise of an advance directive, this principle is universally accepted as the basis of the doctrine of informed consent. If practitioners appreciate that the loss of capacity does not imply the loss of the right to direct the course of one's treatment, then they are obliged to accept the validity of advance directives. Advance directives make a special claim on us, beyond that made by the informed consent doctrine, because patients who have lost the capacity for informed decisions and autonomous actions are especially vulnerable to the will of others.

Once we acknowledge the importance of ensuring that patient wishes outlined during competency are heeded when preferences can no longer be articulated, we must assume responsibility for having these discussions in our practice.

The PSDA only outlines minimal services which need to be provided to patients, families, and health care agents.[1] Institutions can comply with Federal law by simply providing patients with a brochure detailing institutional policies and their rights to refuse treatment and formulate advance directives based on state laws. Compliance also requires that staff inquire if patients have advance directives and that such documents become part of the medical record. This narrow reading of the Federal mandate, however, misconstrues legislative intent.[3,4] Although an intended by-product of the PSDA is increased use of advance directives, the key end product of this process is a meaningful and sensitive dialogue between clinicians, patients, families, and surrogates about end-of-life decisions.

This paper presents strategies that will enable physicians to introduce discussions about advance directives into their practice and assist patients in clarifying their values and treatment preferences. The special burdens of surrogates and agents empowered by proxy will be considered along with the obligations of the clinician.

INTRODUCING ADVANCE DIRECTIVES INTO THE CLINIC

Conversations about advance directives should be part of ongoing medical care[8] and a way to enhance patient-physician collaboration. Physicians might consider such

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An earlier draft of this work was presented as part of the Hastings Center program "The Patient Self Determination Act: Meeting the Challenge" regionally co-sponsored by the New York Academy of Medicine and the Center for Ethics of the University of Chicago.

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Research for this paper was supported by a grant from the Nathan Cummings Foundation to the Hastings Center.

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dialogues like other preventive services seeking to minimize future burdens through prophylactic interventions. Placing these discussions in this context provides time for thoughtful consideration of these difficult issues and an opportunity for patients to discuss their feelings with family members, friends, and clergy without the undue pressure of an emergent hospital admission.

In addition to providing patients time for thoughtful consideration, such discussions will emerge as timely opportunities to clarify treatment preferences before an impending dementia or unforeseen loss of competence. The importance of outpatient discussions is reinforced by recalling the circumstances of the Cruzan case in which the parents of a young woman left in a persistent vegetative state (PVS) after a car accident sought to discontinue their daughter's artificial nutrition and hydration.[9]

In this landmark case, the Supreme Court recognized the constitutional right of a competent person to refuse life sustaining therapy and a state's prerogative to establish a "clear and convincing" evidentiary standard about decisions concerning life-sustaining artificial nutrition and hydration. These tragic circumstances also prompted Justice O'Connor to suggest an enhanced role for proxy decision makers, chosen by a patient while still competent, who could later direct treatment choices.[10]

If similar unfortunate circumstances were to recur, it would again be impossible to elicit a patient's treatment or proxy preferences along the corridors of an Emergency Room. Cruzan also illustrates that although elderly and infirm patients are more likely to become incapacitated, all adult patients are appropriate candidates for discussions about advance directives.

Although physicians may find it difficult to discuss these issues with patients, a desire to avert another case such as Cruzan should be adequate motivation to initiate the dialogue. Physician initiative is especially important because patients want this discussion to occur and expect their physicians to bring it up.[6] If we fail to address advance directives, patients may wrongly surmise that they are either unimportant or inappropriate. This problem is compounded by our mistaken, and perhaps self-serving belief, that patients would find this topic too difficult to handle.[6] It is important that physicians recognize their own uneasiness about discussing advance directives so that this obstacle to implementation can be overcome.

Advance directives may be introduced as a means to direct medical care during a period of life when patients will no longer be able to speak for themselves. Patients need to be reassured that advance directives only go into effect only when decision-making capacity is lost; they may be amended and revoked at any time during capacity. Clinicians also need to explain that competence is assumed whenever a revocation or emendation is contested. In such cases, whomever questions decision-making capacity is obliged to demonstrate loss of competence in court. The New York State Health Care Proxy Law explicitly states that patient wishes "prevail unless the principal is determined by a court of competent jurisdiction to lack capacity to make health care decisions."[11]

Delegation of decision-making authority requires that patients place their trust in a complex process which will protect their future interests. This trust can first be cultivated through a timely dialogue focusing on patient values as they relate to end-of-life issues. Patients and physicians can mutually benefit from this exploration which provides an opportunity for reciprocal understanding. This process will place patient choice into a rich context that should help physicians appreciate their patients' motivations. As partners in this dialogue physicians identify themselves as caring and concerned individuals in whom patient trust can be placed.

When discussing advance directives physicians must be careful not to be judgmental about decisions patients' reach. The literature suggests that physicians often may not appreciate the preferences of elderly patients with respect to life-sustaining therapies.[12] Physician preferences and valuation of patient quality of life are often at great variance with the perceptions of patients themselves. These valuations can color the clinicians view of appropriate end-of-life decisions even though they may not exert a similar influence among patients.[13] Physicians must recognize these potential biases so as to enable neutral counselling.

Patients who decide not to have an advance directive after physician counselling, must understand that future care is not conditioned on the presence or absence of an advance directive. Federal law explicitly prohibits institutions from such conditioning of care.[1] Physicians should appreciate that patients who decide against an advance directive after time consuming counselling are likely to be uneasy. They may feel that they have disappointed their doctor with their refusal. This guilt should be assuaged quickly because it can disrupt an evolving doctor-patient relationship. By stressing that the goal of advance directive counselling is value clarification and not documentation, physicians can demonstrate to patients that the time spent together was neither wasted nor disruptive. Physician willingness to spend time with patients in an educational venture that does not lead to a demonstrable "end-product" will only serve to bolster patient-physician trust and communication.

Patients and families should appreciate that if an advance directive is not completed, patients and/or families do not lose their right to determine treatment decisions.[14] Expressed preferences outside of formal advance directives continue to have legal effect after the PSDA. Physician notes, for example, can be an especially important source for determining patient preferences in the absence of formal advance directives.[15]

Physicians should stress that a patient does not need a treatment directive, proxy, or living will of any sort to have a Do Not Resuscitate (DNR) order written. If a patient is competent and agrees to a DNR order and then loses capacity, the order stands. If a surrogate or health care agent has been designated, their consent is not needed for a DNR order if the patient had consented to this order before loss of capacity.[16] Family members can still request that patients be made DNR without a written advance directive by invoking earlier wishes, or failing that a best interest standard of decision making.

Recent changes to New York's Public Health Law have established that the Health Care Proxy law supersedes certain provisions of the state's earlier DNR legislation.[17] The law now clearly notes that a health care agent may make decisions about resuscitation for the patient who has lost capacity unless the patient has specifically limited his/her agent's authority. These amendments also specifically assert that a health care agent appointed under the Proxy law has "decision making priority" over other individuals including a surrogate, who might have been appointed under the DNR law. This legislative clarification is important because both a health care agent designated under the Health Care Proxy law and a surrogate appointed under the DNR law could have conflicting authority to make these decisions. Although these provisions clearly delineate the decision-making hierarchy which should guide surrogate decision making in New York State (and rightly recognize that the health car agent's authority is broader than a surrogate's) potential conflicts may still occur if patients are unaware of this aspect of the law.

CHOOSING AN ADVANCE DIRECTIVE

After deciding to complete an advance directive, the patient needs to decide whether he/she would be best served by a treatment directive outlining specific preferences, a health care agent who speaks for the patient, or an agent who is informed by a treatment directive. The advantages and limitations of each type of advance directive require explanation. For example, although the written document may offer specificity for some scenarios, it may not address a patient's specific clinical circumstances. Physicians might explain to patients that a health care proxy provides a procedural mechanism for an agent to relate the patient's previously expressed preferences, whereas a treatment directive directly conveys the patient's specific and substantive written wishes.

Structured treatment directives may help clarify values and provide a framework for decision making. Among such documents are the Values History Form,[18] and New York Living Will,[19] and Medical Directive.[20] Patients may use these for either documentation purposes or as a means to structure their consideration of treatment options or choice of an agent.

Because treatment directives are written documents, they may fail to have the impact of an agent who can serve as a visible advocate for the patient. One group found that preferences corresponded to just 75% of outcome events and suggested that this highlighted the limitations of directives which did not employ a proxy or durable power of attorney component.[21] They suggested that a durable power of attorney might be a more effective means to safeguard patient autonomy. Treatment directives, as such, can also assist agents. Health care agents unguided by a treatment directive may fall prey to their own preferences and philosophies and not accurately predict patient preferences.[12,22,23]

Although I favor a proxy designation over a treatment directive, in patients who have identified an individual who might serve in this capacity, a proxy designation alone does not guarantee that a patient's early wishes will be invoked during incapacity. To avoid future treatment choices by the agent that are inconsistent with earlier wishes, physicians should play an active role in helping patients outline their specific preferences to their chosen agent. Unlike the Emanuels' recent suggestion to augment a proxy designation with a formal instructional directive as one way to qualitatively improve proxy decision making,[24] the parallel substantive process advanced here will not erode the agent's authority. Rather, it will likely bolster the agent's already secure legal standing and credibility as it improves the accuracy of proxy decision making. In this approach, the competent patient designates and then instructs an agent about his/her wishes. This requires that the patient discuss a proxy designation with his or her chosen agent. Although the law does not require that a patient obtain the agent's agreement to serve in this capacity, physicians should encourage their patients to secure their agent's agreement to serve in this capacity.[8]

DECISION-MAKING STANDARDS AND ARTIFICIAL NUTRITION AND HYDRATION IN NEW YORK

When an agent is designated, it is important to inform patients, and ideally the agent, that subsequent decisions must follow the expressed preferences of the patient when they are known. If explicit wishes do not exist, the patient's known beliefs and values should be the guide. In the absence of either of these sources of information, decision making should be guided by the best interest standard. Because the best interest standard is most open to the individual variance of the agent, it is the decision-making route of last resort.[25] This decision-making hierarchy is embodied in the New York State Health Care Proxy Law and pertains to all decisions except ones made by the agent concerning the administration of artificial nutrition and hydration.[26]

The right of a state to set an evidentiary standard for decisions relating to nutrition and hydration established in Cruzan[9] has special importance to patients contemplating a proxy designation in New York State. The New York State Task Force on Life and the Law acknowledged, before Cruzan, that although there are unique concerns about the withholding or withdrawal of artificial nutrition and hydration, these concerns "do not outweigh the right of a competent adult to have his or her wishes honored."[27] Recognizing the difficulty of reaching a consensus on this often divisive issue, and invoking the state's "responsibility to craft policies for critical treatment decisions on behalf of incompetent patients," the Governor in his Approval Message which accompanied the Health Care Proxy legislation wrote of the need for "special safeguards" for decisions made by a health care agent relating to the administration of artificial nutrition and hydrations.[28]

Unlike other treatment decisions made by the agent, nutrition, and hydration decisions cannot lawfully be made on a best interest standard. Although the agent has the authority, subject to any expressed limitations, "to make any and all health care decisions on the principal's behalf that the principal could make,"[29] the agent's authority is limited with decisions pertaining to the administration of nutrition and hydration:

> . . . if the principal's wishes regarding the administration of artificial nutrition and hydration are not reasonably known and cannot with reasonable diligence be ascertained, the agent shall not have the authority to make decisions regarding these measures.[26]

Physicians should inform patients of the limitations placed on the agent's power regarding decisions about artificial nutrition and hydration and urge patients to state explicitly their wishes. Although oral attestations by the principal to an agent about artificial nutrition and hydration can provide an agent with the authority to make decisions in this arena, physicians should counsel their patients to provide written documentation of their future choices to satisfy unequivocally the expectation that the patient's wishes are "reasonably known" by the agent.[26] This intervention may ensure that the advance directive meets the states evidentiary standard for such decisions.

ARTICULATING VALUES

Discussions about difficult issues like nutrition and hydration point to the need for both the patient and the agent to explore values together. Discussions between patient and agent before incapacity may serve to anticipate any irreconcilable differences of philosophy which might prompt the patient to reconsider his/her proxy designation. Physicians might urge patients to think of these discussions as an audition where the patient determines whether his or her choice of an agent is a good one and the agent tries on the role as well. Finally, this dialogue helps to educate the agent about the patients views and preferences. Placing these choices into a richer context of personal experience will give the agent additional guidance.

A structured discussion of possible scenarios may be beneficial. Patients may find it especially helpful and even reassuring to discuss specific disease scenarios which they might encounter because of their personal medical history. It may be helpful to first address the patient's overall value structure about medical treatment. Physicians should encourage patients to consider under what circumstances they would choose to prolong life at all costs, opt for quality of life over prolongation of life, and when they would desire palliation and comfort measures. Clinicians might ask patients to think about what makes life meaningful and enjoyable for them and how this might be tempered by suffering, disability, and infirmity.

Physicians may choose to structure these discussions by employing one of the available directives[18–20] as a document to be completed or as an outline to guide a fruitful dialogue. By discussing several scenarios in which patients are without capacity and treatments they might choose, patient and/or agent will move toward value clarification along with the physician. In guiding these discussions, it is especially important that the patient be able to outline informed choices. This requires that physicians adequately explain the prognoses associated with terms such as PVS, coma, and dementia as well as the pertinent details of life-sustaining therapies such as cardiopulmonary resuscitation, ventilator use, and treatment in intensive care units.

When speaking of these therapeutic interventions it is helpful to couple them with possible outcomes. Vague attestations about the use of a specific intervention may be avoided when a therapy is linked to a likely outcome. Although patients may object to being placed on a ventilator, it is important to explore this preference carefully. For example, a patient may envision inappropriate ventilator use when she is in her ninth decade and unable to either recall her past or anticipate her future. This same patient might agree to a ventilator used for an emergent cholecystectomy 30 years earlier. Thus, when speaking with patients, it is important not to focus only on the therapeutic interventions, but also on the goals they are being used to achieve.

FOLLOW-UP AFTER COMPLETION OF A DIRECTIVE

Once a patient has completed an advance directive, these issues and choices should be revisited to ensure that they continue to reflect the patient's wishes. If there is no major medical or personal event, the annual physical examination might be a good interval to review these issues. Personal events such as bereavement, marriage, divorce, or a new child should prompt a review of these issues because it may affect value choices or the choice of an agent.[30] In some states, such personal developments have legal implications with respect to proxy designation. In New York, for example, a spouse chosen as an agent automatically loses that designation in the event of legal separation or divorce. A former spouse may continue to be an agent, but this needs to be readdressed after a divorce or separation.[31] Changes in medical status are clearly important points for a revisiting of choices. Throughout the ongoing counselling of the competent patient, it is critical to reiterate that an advance directive is neither mandatory nor required and may be revoked at any time. Health care agents also need to be part of these periodic discussions to ensure that their understanding of patient preferences is current and accurate.

ACTIVATING THE ADVANCE DIRECTIVE

A critical juncture for patients, families, surrogate/agents, and health care workers is the determination of incapacity. Physicians must be attentive to the capacity of their patients to ensure the timely authorization of advance directives and the involvement of the agent. A determination of incapacity may be prompted by a request from an agent[32] or during the course of routine medical care surveillance. Physicians should recall that patients may retain decision-making capacity in the setting of cognitive impairment which does not impair their ability to understand treatment choices which may be presented.[25] Clinicians should also endeavor to maximize decision-making ability by correcting reversible causes of incapacity so as to vest authority with the patient whenever this is possible. Discussions about the duration of incapacity should be held with the agent so as to avoid the encroachment of the agent's authority on the patient's right to make decisions.

The integrity of advance directives hinges on the determination of incapacity. A failure to note incapacity would perpetuate a situation in which the patient is unable to speak for him/herself, before the advance directive has become operative. If advance directives are based on the principle of patient autonomy, clinicians are obligated to ensure that either the patient or an advance directive gives voice to treatment preferences. This requires that capacity be periodically reassessed to avoid the error of omission which follows from the failure to not declare incapacity in a timely fashion.

The New York State Health Care Proxy Law specifically requires that the determination of lack of capacity be made "with a reasonable degree of medical certainty" by the attending physician who is primarily responsible for the patient's care.[33] This physician is required to document the likely "cause and nature" of the patient's incapacity and its "probable duration." The responsible physician is obliged to obtain a concurring opinion from a colleague that the patient lacks capacity when a decision to "withdraw or withhold life-sustaining treatment" is under consideration.

COUNSELLING OF THE HEALTH CARE AGENT

One of the most challenging and potentially rewarding aspects of working with advance directives is the role of the clinician in ongoing discussions with the health care agent. Perhaps the best approach is to keep the agent involved from the start. Even if the agent has been involved, the clinician needs to remind him/her of the decision-making standards which must be followed.[15,25] The clinician needs to be sensitive to occasions when the agent may have difficulty pursuing a choice outlined by the patient. Although this may be avoided through the careful choice of an agent, it may be tempered by a clinician who reminds the agent that his/her primary collective obligation is to follow the goals outlined by the patient.

Patient and agent may both ask when an agent may override an advance directive on this basis of a best interest standard of decision-making. Although this may come up as a frequent question, care must be taken to avoid "trumping" advance directives; such behavior undermines both patient choice and the trust placed in advance directives.[8,34] This trust is based on the presumption that what is outlined during competency will be followed thereafter. If advance directives are regularly overridden, future patients will lose faith in this process and be deprived of an important avenue for expressing their autonomy.

Conflicts concerning the demented patient are especially difficult.[8] There may be a hesitancy to accept an advance directive because a patient's current best interest may not seem to coincide with choices outlined in a directive. This can be especially troubling because demented patients may be capable of interaction yet unable to exercise judgment necessary for decision making or to recall an earlier directive. Physicians should specifically discuss decision making in the setting of dementia to anticipate this troubling scenario.[8] Preemptive counselling efforts with patients, which either clarify a treatment directive or which act to inform an agent in the event of such a conflict, will help to reinvest such difficult decisions with the principal's voice and avoid resorting to a best interest standard out of ignorance of the patient's wishes or values.

PROFESSIONAL SELF-REFLECTION

Patient choices outlined in an advance directive may foreshadow untenable situations which conflict with a physician's values. Although practitioners have the right to avoid acts to which they conscientiously object, this prerogative obliges physicians to discuss future conflicts with their patients. Patients are entitled to know about potential sources of conflict when still competent. Such professional frankness will allow patients to make alternative care plans and avoid problems later. Physicians should offer patients the services of an alternative practitioner and help to transfer patient care responsibilities. Alternatively, patient and physician may agree to continuing the therapeutic relationship and discuss a mechanism for transfer of patient care at a later date should this become necessary.

Physicians should explore their own views on such issues as nutrition, hydration, and analgesia to participate in their patient's end-of-life decisions. Such professional self-knowledge might be construed as a prerequisite for such discussions. Although physicians need not disclose their own value system, they must be sufficiently cognizant of their own beliefs to function as counsellors. Such exploration is likely to occur in tandem with patients as they come to understand their own choices.

THE PSDA IN PERSPECTIVE

The PSDA has the potential to strengthen the relationship between patient and physician. This potential can be realized if physicians view enhanced patient participation in decision making as a necessary precondition for a more authentic clinical encounter. By providing an opportunity to explore mutually fundamental values, the PSDA could lead to a richer patient-physician collaboration in end-of-life decisions.

REFERENCES

1. Omnibus Budget Reconciliation Act of 1990. Public law no. 101–508.
2. Greco PJ, Schulman KA, Lavizzo-Mourey R, et al: The Patient Self-Determination Act and the future of advance directives. *Ann Int Med* 1991; 115: 639–643.
3. McCloskey EL: The Patient Self-Determination Act. *Kennedy Inst Eth J* 1991; 1:163–169.
4. McCloskey EL: The spirit of the PSDA. *Hastings Cent Rep* [suppl] 1991; 21:S14–S15.
5. Hayward RSA, Steinberg EP, Ford DE, Roizen MF, et al: Preventive Care Guidelines: 1991. *Ann Intern Med* 1991; 114:758–783.
6. Emanuel LL, Barry MJ, Stoeckle JD, et al: Advance directives for medical care—A case for greater use. *N Eng J Med* 1991; 324:889–895.
7. Doctors in Distress. *N Y Times* Feb 18,19,20, 1990.
8. Wolf SM, Boyle P, Callahan D, et al: Special report: Sources of concern about the Patient Self-Determination Act. *N Eng J Med* 1991; 325:1666–1671.
9. Cruzan v Director, Missouri Department of Health. 497 US, 111L ed 2 224, 110 S. Ct. 2841 (1990)
10. Annas G: Nancy Cruzan in China. *Hastings Cent Report* [suppl] 1990; 20: 539–541.
11. New York Public Health Law Article 29-C. Health Care Proxy Law 2983.5.
12. Uhlmann RF, Pearlman RA, Cain KC: Physicians' and spouses' predictions of elderly patients' resuscitation preferences. *J Geront* 1988; 43:M115–121.
13. Uhlmann RF, Pearlman RA: Perceived quality of life and preferences for life sustaining treatment in older adults. *Arch Intern Med* 1991; 151:495–497.
14. Wolf SM: Honoring broader directives. *Hastings Cent Rep* [suppl] 1991; 21:S8–S9.
15. Sachs GA, Siegler M: Guidelines for decision making when the patient is incompetent. *J Crit Illness* 1991; 6:348–359.
16. New York Public Health Law 2965 (1).
17. New York Public Health Law 2962 (5).
18. Gibson JM: National values history project. *Generations* [suppl] 1990; 14: 51–64.
19. Society for the Right to Die: New York Living Will, New York.
20. Emanuel LL, Emanuel EJ: The medical directive: A new comprehensive advance care document. *JAMA* 1989; 261:3288–3293.
21. Danis M, Southerland LI, Garrett JM, et al: A prospective study of advance directives for life sustaining care. *N Eng J Med* 1991; 324:882–888.
22. Diamond EL, Jernigan JA, Mosely RA, et al: Decision-making ability and advance directive preferences in nursing home patients and proxies. *Gerontologist* 1989; 29:622–626.
23. Zweibel NR, Cassel CK: Treatment choices at the end of life: A comparison of decisions by older patients and their physician selected proxies. *Gerontologist* 1989; 29:615–621.
24. Emanuel EJ and Emanuel LL: Proxy decision making for incompetent patients: An ethical and empirical analysis. *JAMA* 1992; 267:2067–2071.
25. President's commission for the study of ethical problems in medicine and behavioral research. Deciding to forego life sustaining treatment: A report on the ethical, medical, and legal issues in treatment decisions. *US Government Printing Office*, Washington, DC, 1983
26. New York State Public Health Law Article 29-C. Health Care Proxy Law 2982.2
27. The New York State Task Force on Life and the Law: Life sustaining treatment: Making decisions and appointing a health care agent. July, 1987.
28. Cuomo M: Governor's approval message. Memorandum filed with senate bill number 6176-A.
29. New York State Public Health Law Article 29-C. Health Care Proxy Law 2982.1.
30. Emanuel L: PSDA in the clinic. *Hastings Cent Rep* [suppl] 1991; 21:S6–S7.
31. New York State Public Health Law Article 29-C. Health Care Proxy Law 2985.1.
32. New York State Public Health Law Article 29-C. Health Care Proxy Law 2983.2.
33. New York State Public Health Law Article 29-C. Health Care Proxy Law 2983.1.
34. Brock DW: Trumping advance directives. *Hastings Cent Rep* [suppl] 1991; 21:S5–S6.

Total body photography for melanoma surveillance

William E. Slue, Jr

Baseline total body photography of dysplastic nevi patients and family members at risk for malignant melanoma serves as a valuable tool for the physician determining whether and when to perform a biopsy. The photography is a substantial adjunct to written chart documentation relative to the fact that it is cost effective, relatively easy, and usually needs to be done only once, thereby reducing the psychological discomfort that the patient may feel as a result of having the photographs taken.

Set-Up and Equipment

Your usual office will probably yield sufficient room to take full body photographs. There should be a wall space that is about four feet wide, allowing for a seamless backdrop extending from floor to ceiling and enough space to allow you to stand about three feet in front of the patient. I prefer the seamless backdrop to be sky blue, although some will use black as it tends to absorb the shadow that is often cast. A small step stool like the ones used to stand on in surgery should be placed in front of the background. Having the patient stand on this will prevent the floor or baseboard from appearing in the photograph while you are photographing the lower legs.

The equipment for this type of photography is a 35mm camera, a normal micro lens, an electronic flash with fresh batteries and Kodachrome 25 with 36 exposures. Kodachrome is my film of choice, but whichever film is selected, a test roll should be taken to determine proper exposure.

Test Roll

Set the electronic flash on manual exposure and the shutter speed at 1/60 second (or the speed at which your flash syncs with the shutter.) Set the lens at 2.5 feet and take a series of exposure from f/22 to the largest opening of your lens. Make a note on a pad of each exposure in the order they are taken. When the film is processed, the slides are numbered so that you can determine which exposure is best for the given distance required by your needs. I find this method is better than the automatic exposure because automatic exposure is not consistent. In addition, the smaller lens opening used will give you a greater depth of field than the automatic mode.

Once the equipment and space have been obtained, a system of documentation must be created. The system must be consistent, easy, and timely for both the patient and the physician. All of the photographs are to be taken at one focal length. This means that the distance will be preset to 2.5 feet as shown on the barrel of the lens, and then a body focus is performed. Body focus means moving forward and backward until you are in focus sharply. With a stick-on ruler on the back of the patient and all the photos taken in the above manner, it becomes easy to tell the size of all of the nevi. The sun-exposed portions of the body are the areas of emphasis in the photographic protocol. However, if there are lesions noticed in other areas (inner arms and legs), these should also be noted and photographed. Figure 1 shows how the body is sectioned, and examples of the patient's moles are illustrated in Figure 2.

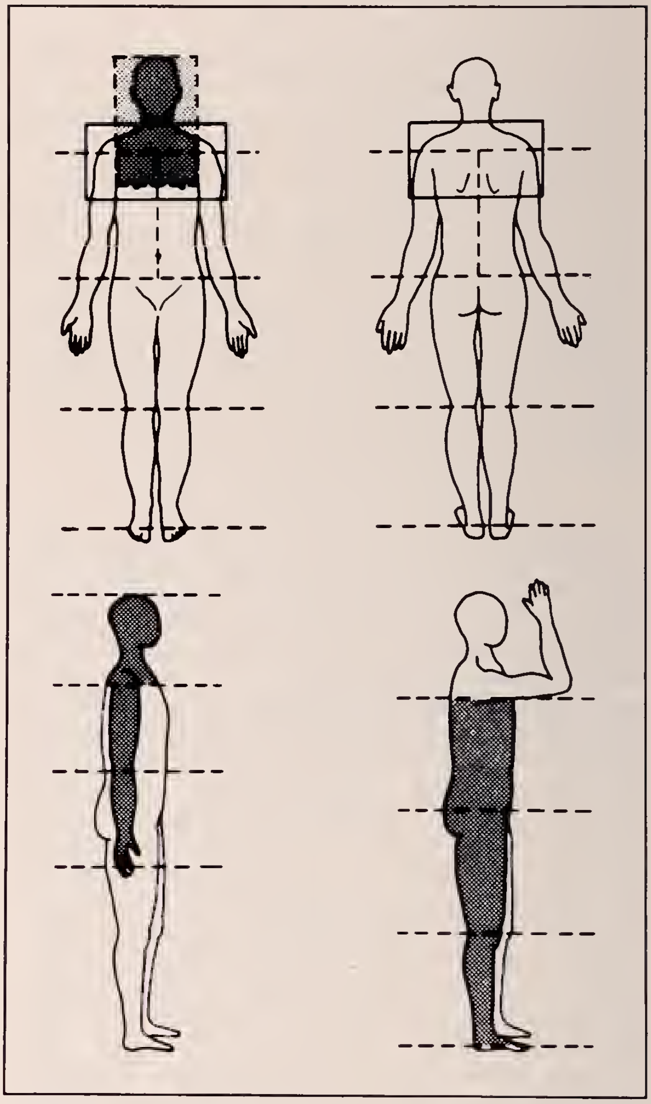

FIGURE 1. This diagram shows how the body is sectioned for the photographs.

Portions of this article previously appeared in an educational series published by Dermik Laboratories for Dermatology residents.

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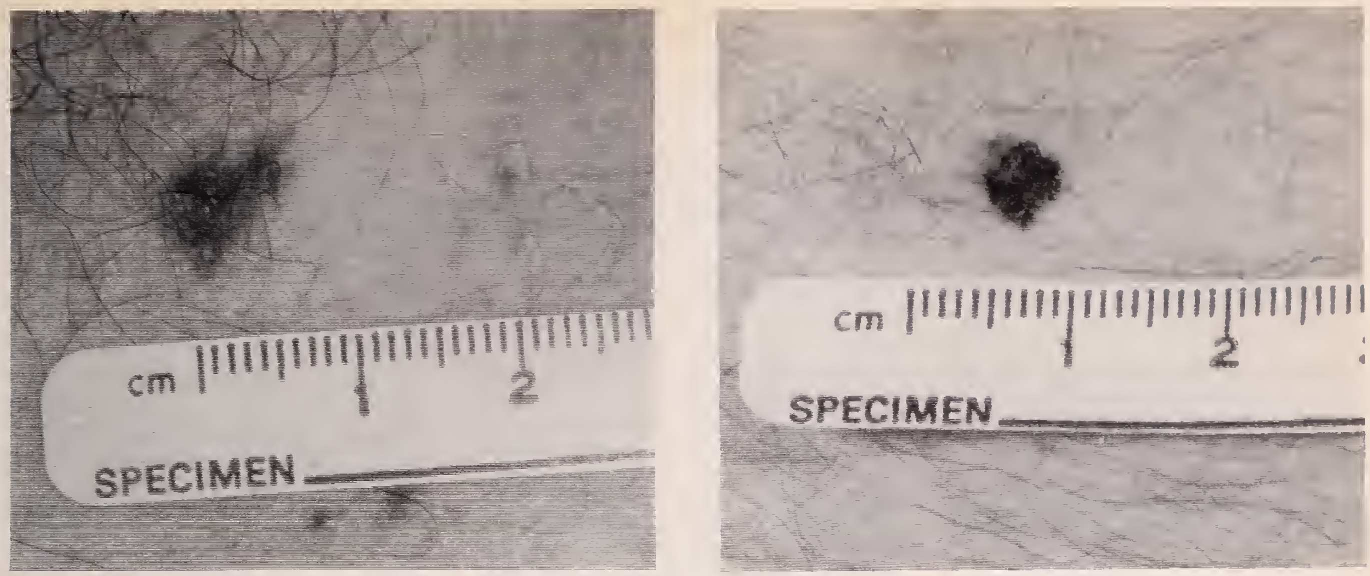

FIGURE 2. Close-up photographs of nevi.

VIEWING

The finished photographs are used in conjunction with a slide viewer such as the Kodak Ektagraphic viewer. The combination of doing the photographs as described and viewing them in the Ektagraphic viewer is very helpful when you realize that the image seen is half the size of the live patient being examined. Using a stack-loader to load the slides, the patient can be viewed with the lights on while comparing their present lesions to those recorded on the slides. Some people prefer to convert their slides to 8″ × 10″ prints. The prints are covered with page protectors, placed in a loose leaf binder, and presented to the patient for them to keep and bring with them during regular visits to their dermatologist.

CONCLUSION

Total body photographic documentation is a useful adjunct to regular cutaneous examinations by the dermatologist and diligent patient self-examination in the detection of malignant melanoma in patients with dysplastic nevi syndrome. Swiftness of procedure, good organization, and sensitivity to the patients dignity are important to the total body photography procedure success.

Hyperlipidemia in monoclonal gammopathy

Zaeem Ansari, MD

Hypocholesterolemia, rather than hyperlipidemia, is the usual lipid abnormality in untreated patients with multiple myeloma, possibly resulting from increased low-density lipoprotein (LDL) receptor activity in malignant cells. Marked hyperlipidemia, with or without cutaneous xanthomatosis, is a less frequent but well-defined accompaniment of myelomatosis. The etiology of immunoglobulin-induced hyperlipidemia is not clear, although autoimmune mechanisms, inhibition of lipoprotein lipase (LPL), and antibodies to lipoprotein have all been considered. This report describes a patient who was initially evaluated for elevated serum cholesterol and triglyceride levels and who was later diagnosed as having IgG-κ monoclonal gammopathy.

Case Report

An 85-year-old man was referred for evaluation and management of hyperlipidemia. He had been under treatment for hypertension and hyperuricemia, but had no other known major medical problems. His history was negative for angina pectoris, intermittent claudication, or symptoms suggesting transient ischemic attacks. He also denied recurrent severe abdominal pains. He was not known to have diabetes mellitus or thyroid hypofunction.

Physical examination revealed an elderly, somewhat thin man in no acute distress. He was 174-cm tall and weighed 75.5 kg. His blood pressure was 100/66 mm Hg, pulse was 80 beats/min and regular, and respiration was 20/min. Prominent arcus senilis was present, and funduscopic examination showed bilateral macular degeneration, but no hemorrhages or exudates. No carotid bruit was audible, and the thyroid was not enlarged. The lungs were clear. The cardiac impulse was not displaced. There were no signs of congestive heart failure, a grade 2–3 systolic ejection apical murmur without radiation was noted. There was no abdominal organomegaly, and the pulses in both legs were palpable.

The total cholesterol level was 779 mg/dL, serum triglycerides were 931 mg/dL, blood urea nitrogen was 44 mg/dL, serum creatinine was 1.4 mg/dL, total bilirubin was 1.1 mg/dL. The transaminase and alkaline phosphatase levels were within normal limits. The thyroid stimulating hormone level was 7.9 mIU/L, T_4 was 3.8 μg/dL, T_3% uptake was 30.2, and free thyroxine index was 1.1. The hemoglobin count was 10.9 g/dL, white blood cell count was 11,100 mm^3, mean corpuscular volume was 109.4 μm^3; mean corpuscular hemoglobin was 40.3 pg, mean corpuscular hemoglobin concentration was 36.9%, and platelet count was 157,000/mm^3. The peripheral blood film showed 32% segmented neutrophils, 12% band forms, 44% lymphocytes, 2% monocytes, 1% eosinophils, five atypical lymphocytes, three metamyelocytes, and one myelocyte. The serum viscosity was 1.6 (normal, 1.4–1.8). The quantitation of serum immunoglobulins showed marked elevation of IgG to 4,994 mg/dL, and decrease of IgA to 30 mg/dL and IgM to 12 mg/dL. The total protein count was 12 g/dL.

Serum protein electrophoresis showed the following levels: albumin, 5.24 g/dL; total globulin, 6.75 g/dL; α_1-globulin, 0.28 g/dL; α_2-globulin, 1.26 g/dL; and β-globulin, 1.29 g/dL. A monoclonal IgG-κ was identified by immunoelectrophoresis.

Except for minimal proteinuria, the urinalysis was unremarkable. The chest radiograph was consistent with chronic obstructive pulmonary disease. A bone scan was normal.

Bone marrow examination revealed hypercellularity with sheets of plasma cells and plasmablasts, and decreased megakaryocytes, consistent with a diagnosis of multiple myeloma.

The patient was asymptomatic during initial evaluation in mid-1989, and it was not until two months later that he experienced bone pain. Because of abnormalities in the serum protein electrophoretic pattern, he was referred to the Hematology Clinic at State University Hospital. The clinical course was complicated by profuse epistaxis. At that time, the white blood cell count was 8,000/mm^3; the hemoglobin level was 9.9 g/dL, and platelet count was 83,000/mm^3. The prothrombin time was 13.0 seconds (control, 11.3 sec); bleeding time was 16 min, fibrinogen level was 254 mg/dL.

In November 1989, the patient was admitted to the hospital because of increasing mental confusion, fever, chills, and lethargy. At the request of the patient's family, treatment with chemotherapeutic agents was not given. He remained septic in spite of treatment with appropriate antibiotics, and he expired while in the hospital.

Discussion

Multiple myeloma (also known as plasma cell myeloma, myelomatosis, Kahler's disease, plasmacytic myeloma) is a malignant proliferation of plasma cells primarily in bone marrow, although other tissues may be involved. A single clone of plasma cells proliferates producing a monoclonal protein composed of one heavy-chain class (γ, IgG; α, IgA; μ, IgM; δ, IgD; ϵ, IgE) and one-light chain class (kappa, κ, or lambda, λ).[1] Multiple myeloma comprises 1% of all malignancies and approximately 10% of hematologic malignancies.[1]

Various clinical manifestions of monoclonal Igs may result from their physicochemical properties (hyperviscosity syndrome, cryoglobulinemia, or coagulation abnormalities), their antibody activity and affinity for certain antigens (red-cell hemolysis, hyperlipidemia, and xanthomatosis), or unknown pathophysiologic factors (pyoderma gangrenosum, systemic capillary leak syndrome).[2]

The patient presented here had monoclonal gammopathy which was identified as IgG-κ, and the bone marrow was infiltrated with immature plasma cells. Although moderate hypercholesterolemia was detected about 18 months before the patient's acute illness, he had not manifested, in spite of his advanced age, any clinical or symptomatic evidence of atherosclerotic involvement of the coronary, peripheral vascular, or cerebrovascular systems.

Although hyperlipidemia has been reported in cases of multiple myeloma,[3–5] hypocholesterolemia occurs more frequently in untreated myelomatosis.[6] Hypocholesterolemia probably results from elevated LDL receptor activity in malignant cells, including the myeloma cells.[7]

The occurrence of hyperlipidemia in the presence of circulating immunoglobulins reactive against plasma lipoproteins has been observed most frequently in multiple myeloma.[8] There are several reported cases of various types of hyperlipidemia associated with[9,10] or without xanthomatosis. Although lipid abnormalities may be discovered incidentally, acute pancreatitis secondary to hypertriglyceridemia[11] or atherosclerosis from hypercholesterolemia[12] may result. Xanthomas, which may develop in myelomatosis in the absence of hyperlipidemia, are either generalized, large, and flat, or tuberous eruptions located on knees, elbows, and buttocks.[10]

The hyperlipidemia in myelomatosis is predominantly of the mixed type. Total cholesterol and triglyceride levels are increased and the serum is lipemic. At times, either hypercholesterolemia or hypertriglyc-

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eridemia may predominate. Beaumont[5] has described three types of myeloma with hyperlipidemia: IgA myeloma with mixed hyperlipidemia and cutaneous, tuberous, and plain palmar/plantar xanthomas; IgG myeloma with predominant hypercholesterolemia and plain cutaneous xanthomas; and the IgG myeloma with predominant hypertriglyceridemia and no xanthomas.

The mechanisms by which monoclonal hypergammaglobulinemia may affect lipoprotein metabolism are not clear, but several theories have been postulated. Studies have shown that impaired lipoprotein catabolism, rather than overproduction, leads to elevated intermediate-density lipoprotein (IDL) in type III hyperlipidemia.[13] In the study by Cortese et al,[13] lipoprotein fractions of very-low-density lipoprotein (VLDL), IDL, and LDL contained IgA, but high-density lipoprotein (HDL) fractions did not; a common feature of VLDL, IDL and LDL is the presence of apoprotein B. It is believed that immunoglobulin-lipoprotein complexes block the sequential catabolism of IDL and LDL.[5,11,14]

Chylomicrons and VLDL are presumed to undergo catabolism by the step-wise action of two enzymes, LPL and lecithin cholesterol acyltransferase (LCAT), resulting in separation of triglycerides from core lipids such as IDL. Myeloma paraproteins may have anti-heparin activity. Heparin or heparin-like substances are required for LPL activation.[10] LCAT deficiency may occur in certain hepatic diseases, and myelomatosis may also result in hepatic dysfunction.

Additional mechanisms of autoimmune hyperlipidemia are a decrease in clearance of lipoprotein-immune complexes by macrophages, diminished excretion in bile, and possibly increased compensatory lipoprotein synthesis.[13,15]

The treatment of autoimmune hyperlipidemia is unsatisfactory, since dietary interventions and hypolipidemic agents from the fibric acid class of drugs, which usually lower cholesterol in type III hyperlipidemia, are ineffective,[13] and experience is limited with the use of inhibitors of 3-hydroxy-3-methylglutaryl-coenzyme A reductase in this form of hyperlipidemia. The management of the underlying disease process may also require plasmapheresis to control hyperviscosity.[2]

Acknowledgments. The author thanks Ms Debra Kronenbitter and Ms Pam Gilbert for their assistance in the preparation of the manuscript.

REFERENCES

1. Kyle RA: Multiple myeloma, review of 869 cases. *Mayo Clin Proc* 1975; 50:29–40.
2. Farhangi M, Merlini G: The clinical implications of monoclonal immunoglobulins. *Semin Oncol* 1986; 13:366–379.
3. Roberts-Thomson PJ, Venables GS, Onitiri AC, et al: Polymeric IgA myeloma, hyperlipidemia and xanthomatosis: A further case and review. *Postgrad Med J* 1975; 51:44–51.
4. Ozer FL, Telatar H, Telatar F, et al: Monoclonal gammopathy with hyperlipidemia. *Am J Med* 1970; 49:841–844.
5. Beaumont JL: Autoimmune hyperlipidemia, in Jones RJ (ed), *Atherosclerosis Proceedings of the Second International Symposium.* New York, Springer-Verlag, 1970, pp 166–179.
6. Van Howe W, Rottiers R, Hamers J: Hypercholesterolemia in multiple myeloma before and after treatment. *Acta Clin Belg* 1973; 28:198–205.
7. Vitols S, Bjorkholm M, Gahrton G, et al: Hypocholesterolemia in malignancy due to elevated low-density lipoprotein receptor activity in tumor cells: Evidence from studies in patients with leukemia. *Lancet* 1985; 2:1150–1153.
8. Kilgore LL, Patterson BW, Parenti DM, et al: Immune complex hyperlipidemia induced by an apolipoprotein-reactive immunoglobulin A paraprotein from a patient with multiple myeloma: Characterization of this immunoglobulin. *J Clin Invest* 1985; 76:225–232.
9. Taylor JS, Lewis LA, Battle JD Jr, et al: Plane xanthoma and multiple myeloma with lipoprotein-paraprotein complexing. *Arch Dermatol* 1978; 114:425–431.
10. Wilson DE, Flowers CM, Hershgold EJ, et al: Multiple myeloma, cryoglobulinemia and xanthomatosis: Distinct clinical and biochemical syndromes in two patients. *Am J Med* 1975; 59:721–729.
11. Glueck CJ, Levy RI, Glueck HI, et al: Acquired type I hyperlipoproteinemia with systemic lupus erythematosus, dysglobulinemia and heparin resistance. *Am J Med* 1969; 47:318–324.
12. Lewis LA, deWolfe VG, Butkus A, et al: Autoimmune hyperlipidemia in a patient: Atherosclerotic course and changing immunoglobulin pattern during 21 years of study. *Am J Med* 1975; 59: 208–218.
13. Cortese C, Lewis B, Miller NE, et al: Myelomatosis with type III hyperlipoproteinemia: Clinical and metabolic studies. *N Engl J Med* 1982; 307: 79–83.
14. Reckless JPD, Stocks J, Holdsworth G, et al: Studies of a variant very low density lipoprotein with an acquired deficiency of apolipoprotein C-II. *Clin Sci* 1982; 62:93–100.
15. Glueck HI, MacKenzie MR, Glueck CJ: Crystalline IgG protein in multiple myeloma: Identification effects on coagulation and on lipoprotein metabolism. *J Lab Clin Med* 1972; 79:731–744.

Primary sarcomas of the breast in women under 20 years of age

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Malignant breast lesions in women under 20 years of age are unusual. Most breast masses in this age group should be managed conservatively. However, the incidence of infiltrating ductal cancer does increase with age. Therefore, as the age of the patient increases, the threshold for excisional biopsy should increase.[1]

Children and adolescents are susceptible to a variety of rare mesenchymal malignant neoplasms, such as lymphomas[2,3] and sarcomas, including malignant cystosarcoma phylloides.[4–6] These rare lesions represent a pitfall for the clinician treating a child or adolescent with a breast mass. We present our experience with primary breast sarcomas in this age group and review the literature with regard to clinical features, diagnosis, treatment options, and outcome.

CASE REPORTS

Since 1951, the Breast Service at the Memorial Sloan-Kettering Cancer Center has treated six patients under the age of 20 for primary malignant diseases of the breast. All six patients were female. Two of these patients had ductal carcinoma and have been previously reported.[7,8] The other four patients had primary breast sarcomas, or malignant cystosarcoma phylloides, and their reports constitute this review. In addition, this institution previously reported seven cases of ductal carcinoma in young patients referred for consultation but not treated at Memorial Hospital,[9] as well as two patients, both aged 14, with embryonal rhabdomyosarcoma metastatic to the breast.[7]

Case 1. A 16-year old girl was referred to Memorial Hospital in mid-1975, with a one-year history of persistent yellow discharge from the right nipple and an enlarging painless mass centrally located in the right breast. The patient's medical history included menarche at age 12, gravida 0 para 0, and an unremarkable family history. Physical examination disclosed a 4 × 4 × 4-cm indurated non-tender mass in the center of the right breast and a 0.5-cm mass in the right axilla. A smaller left breast mass was also noted. A chest radiograph and bone scan were normal. A fine-needle aspirate (FNA) of the right breast mass was non-diagnostic. Biopsy revealed malignant cystosarcoma phylloides with elements resembling fibrosarcoma and rhabdomyosarcoma on the right and a fibroadenoma on the left.

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One month later, the patient underwent a right total mastectomy with removal of Level I lymph nodes. On pathologic examination, all margins were negative for tumor, and there were no lymph node metastases. No adjuvant therapy was given. The patient had been free of disease for eight years when she was lost to follow-up.

Case 2. A 13-year-old girl was referred to Memorial Hospital in early 1983 with a rapidly growing, painless mass in the right breast. A recent incisional biopsy had been read as anaplastic sarcoma. Personal and family history were negative for previous breast problems or hormone use. Medical history included menarche at age 12 and gravida 0 para 0. Physical examination disclosed a 7 × 7-cm non-tender mass centrally located in the right breast with non-palpaple axillary nodes. The chest film was normal. After confirmation of the pathologic reading, the patient underwent a right radical mastectomy.

Review of the pathologic material revealed anaplastic sarcoma, Grade III, with some elements of cystosarcoma. Multicentricity was present. The patient underwent immediate reconstruction and later received adjuvant adriamycin and cisplatin. She has been free of disease since treatment.

Case 3. A 16-year-old girl was noted on a routine examination in the summer of 1987 to have a 4 × 3 × 2-cm firm, non-tender mass in the subareolar region of the right breast. No axillary adenopathy was noted. She was gravida 0 para 0, and there was no family history of breast disorders. The patient stated that the mass had been present without change for years. An incisional biopsy of the mass was read as alveolar soft-part sarcoma. She denied any history of other breast disorders, menstrual irregularities, or hormone use. A chest film, bone scan, and liver function tests were all within normal limits.

After referral to Memorial Hospital, the patient underwent a computer tomographic (CT) scan of the chest which revealed a mass in the right breast abutting the chest wall, with loss of fat between the breast and the pectoralis major muscle. After a review of the pathologic material to confirm the diagnosis, the patient underwent a right total mastectomy with partial resection of the pectoralis major in late 1987. Pathologic examination of the resected specimen revealed infiltration of skeletal muscle with tumor, with clear margins. The patient received no adjuvant therapy and has been free of tumor since.

Case 4. This patient first presented in 1962, at the age of 14, with a mass in the upper portion of the left breast, which proved on excision to be an extra-abdominal desmoid tumor (The patient had a family history of Gardner's syndrome). This lesion recurred later that same year and in 1965 and 1970. Review of these serial recurrences showed increasing atypia, and the latter two lesions were diagnosed as low-grade fibrosarcoma. The lesion did not recur after its third reexcision. However, in spite of subtotal colectomy in 1968, the patient had serious problems from intra-abdominal desmoid lesions. These included uterine bleeding that required a hysterectomy, uremia secondary to ureteral obstruction, and sepsis secondary to biliary obstruction. The patient died of hepatorenal failure and sepsis secondary to biliary obstruction in 1978 at age 30.

Discussion

Although malignant breast lesions in girls aged less than 20 years are rare, the risk increases with age, as does the risk of breast cancer in general.[10] In this young population, breast masses are most often benign; half are fibroadenomas, and most of the remainder comprises mastopathy and virginal hypertrophy. Although rare, the most common malignant lesions in such patients are carcinoma and sarcoma.[10–12]

It is generally recommended that the approach to breast masses be conservative until completion of breast development. Observing the mass through two menses has been reported by some investigators as safe.[1,11] However, because the risk of malignancy is small but real, this practice could result in a delay in the diagnosis of serious pathology in some patients.[1] The availability of FNA has simplified this clinical dilemma. In a study of 28 FNAs in 22 young patients with known cancer, Wakely et al [13] described four girls, aged 8, 14, 17, and 19 years, with suspected metastases to the breast. In all four patients, subsequent open biopsy of the breast lesions proved the findings on FNA to be correct. For all 28 biopsies, accuracy was 96%, sensitivity was 100%, and specificity was 88%. Open biopsy should be performed when FNA of a suspicious lesion is non-diagnostic.[11]

We report four primary breast sarcomas in females younger than age 20, including one patient with a malignant phylloides tumor. Breast sarcomas represent less than 1% of all primary breast malignancies; thus, it is not surprising that there are no literature reviews of this lesion in young patients. All of these cases were soft tissue sarcomas; osteoid-containing sarcomas of the breast are even more rare.[6]

The most recent series of breast sarcomas, reported by Pollard et al,[5] consists of 25 cases with a mean age of 55.5 years and no cases under the age of 20 years. In that series, the frequency of different histologic types paralleled that of soft-tissue sarcomas elsewhere: 44% malignant fibrous histiocytoma, 24% liposarcoma, 16% fibrosarcoma, and 4% (one case) each for clear-cell sarcoma, neurogenic sarcoma, leiomyosarcoma, and alveolar soft-part sarcoma. As is also typical for sarcomas elsewhere, lymph node involvement was rare (one case): radical surgery appeared to prevent local recurrence more than metastatic disease, and the majority of deaths occurred in the first five years. No attempt was made to correlate grade with outcome, although it was noted that these tumors behaved clinically like their histologic counterparts at other sites. Overall, mortality in that series was 64% at five years.

In spite of the presence of a high-grade lesion, the patient in Case 2 enjoyed long-term survival, an outcome that may have been due in part to aggressive therapy with radical surgery and adjuvant chemotherapy. Similarly, although Case 3 had unfavorable histologic findings (alveolar soft-part sarcoma), long-term survival was achieved after radical surgery. The low-grade tumor in Case 4 was controlled by repeated local resections. In no case were lymph node metastases evident. Thus, achievement of local control of the primary tumor may be as important in breast sarcomas as in sarcomas elsewhere.

Cystosarcoma phylloides account for 1%–2% of breast lesions in the adolescent female, similar to its incidence in adults.[4] A fibroepithelial neoplasm with more stromal cellularity than fibroadenoma, the lesion is considered to be malignant if the stromal component demonstrates atypia, anaplasia, and a high degree of mitotic activity. Such stromal features give malignant lesions the characteristics of sarcoma, and they may contain malignant osseous, cartilaginous, muscular, fibrous, fatty, and/or myoid elements.[14] Malignancy in cystosarcoma phylloides is less common in adolescents than in adults (15% vs 20%–50%) and is associated with a higher likelihood of local recurrence than in benign lesions (33% vs 2%–3%). Metastases, most frequently blood-borne to the lungs and bones, are particularly rare in such lesions in adolescents. Complete excision of cystosarcoma phylloides is usually curative, even when it is malignant or recurrent.[4] Because of the large size and central location of the lesion in Case 1, this patient was treated with total mastectomy.

References

1. Ferguson CM, Powell RW: Breast masses in young women. *Arch Surg* 1989; 124:1338–1341.
2. Brustein, S, Filippa DA, Kimmel M, et al: Malignant lymphoma of the breast: A study of 53 patients. *Ann Surg* 1987; 205:144–149.
3. Dixon JM, Lumsden AB, Krajewski A, et al: Primary lymphoma of the breast. *Br J Surg* 1987; 74: 214–217.
4. Molitt DL, Golladay S, Gloster ES, et al: Cystosarcoma phylloides in the adolescent female. *J Ped Surg* 1987; 22:907–910.
5. Pollard SG, Marks PV, Temple LN, et al: Breast sarcoma: A clinicopathologic review of 25 cases. *Cancer* 1990; 66:941–944.
6. Muffarrij AA, Feiner HD: Breast sarcoma with giant cells and osteoid: A case report and review of the literature. *Am J Surg Pathol* 1987; 11: 225–230.
7. Farrow JH, Ashikari H: Breast lesions in young girls. *Surg Clin North Am* 1969; 49:261–269.
8. Ashikari H, Jun MY, Farrow JH, et al: Breast carcinoma in children and adolescents. *Clin Bull* 1977; 7:55–62.
9. McDivitt RW, Steward FW: Breast carcinoma in children. *JAMA* 1966; 195:144–146.
10. Greydanus DE, Parks DS, Farrell EG: Breast disorders in children and adolescents. *Pediatr Clin North Am* 1989; 36:601–639.
11. Bauer BS, Jones KM, Talbot CW: Mammary masses in the adolescent female. *Surg Gynecol Obstet* 1987; 165:63–65.
12. Hickerson R, Golladay ES, Mollitt DL, et al: Breast disorders of childhood and adolescence. *Contemp Surg* 1983; 22:51–57.
13. Wakely PE, Powers CN, Frable WJ: Metachronous soft tissue masses in children and young adults with cancer: Correlation of histology and aspiration cytology. *Hum Pathol* 1990; 21:669–677.
14. McDivitt, RW, Steward FW, Berg JW: Tumors of the breast, in *Atlas of Tumor Pathology* Washington, Armed Forces Institute of Pathology, 1968 pp 117–123.

Morgagni hernia mimicking a lipomatous tumor

Perry S. Gerard, MD; Eric Wilck, MD; Elliot Senderoff, MD; Stuart Golbey, MD

Herniation of omentum into the thoracic cavity may result in an enlarging chest mass. When bowel loops are present within the hernia, the diagnosis can often be made on a plain film. If this is not possible, the ability of computed tomography (CT) scans to differentiate between various densities and to define anatomic structures can help distinguish Morgagni hernias from other causes of intrathoracic masses. We present a case of massive herniation of omentum through the foramen of Morgagni with no evidence of bowel in the hernia on either plain films or CT scan.

Case Report

A 45-year-old, obese woman was referred to this hospital with a diagnosis of an enlarging right pericardial mass. The mass was initially detected on a pre-employment chest radiograph obtained six years before admission. Further evaluation of the mass with repeat chest films concluded that it was a pericardial cyst, and the patient's condition was managed conservatively.

The patient remained asymptomatic until shortly before referral to this hospital, when she developed a persistent dry cough associated with right-sided chest pain. There was also a sense of fullness in her right chest. Follow-up chest radiographs (Figs 1,2) showed enlargement of the mass occupying the anterior mediastinum adjacent to the right heart border. CT scan of the chest demonstrated that the mass was of fat density and contained serpentine linear densities consistent with fibrotic strands or blood vessels (Fig 3). Slices obtained through the region of the liver showed an abnormal fat density situated between the anterior abdominal wall and the margin of the liver. Lower scan images showed colon adjacent to the mass. The colon appeared displaced anteriorly and superiorly. Differential diagnoses of a liposarcoma or a Morgagni hernia were entertained.

The patient's symptoms continued, and she underwent surgery. A right lateral thoracotomy was performed. An encapsulated, fatty mass was identified and subsequently separated from the pericardium; its origin was traced to a defect in the anterior diaphragm. The abdomen was then opened, and a defect in the diaphragm was palpated where peritoneum and omentum had herniated through the foramen of Morgagni. When the intrathoracic specimen that contained omentum was excised, it weighed two pounds. There was no evidence of any visceral organs in the hernia sac. The patient made an uneventful recovery.

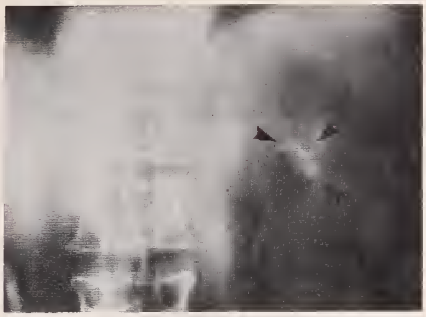
FIGURE 1. Posteroanterior chest radiograph shows a sharply defined mass at the right base obliterating the right heart border (arrowheads).

Discussion

Anterior diaphragmatic hernias have been noted as early as 1761, when Giovani Morgagni described the entity on the basis of a large autopsy study.[1] The defect most commonly occurs slightly lateral to the xiphoid, although it may also occur in a directly posterior position. The etiology of this defect is thought to be failure of fusion of the diaphragm to the costal cartilages and sternum.[2]

Morgagni hernias are associated with obesity, aging, trauma, and other causes of increased intra-abdominal pressure. Morgagni hernias usually occur on the right side, since the heart and pericardium cover left-sided defects. Omentum and transverse colon are the most common structures within the hernia sac, although liver and stomach, or small intestine may also herniate. Patients are usually asymptomatic, although complaints of abdominal and retrosternal pain, nausea, vomiting, or dyspnea may occur.[3,4] Strangulation of bowel within the hernia sac is a rare complication.

The characteristic features of Morgagni hernias have been described with a variety of radiologic imaging modalities, including CT and magnetic resonance imaging (MRI). Several features consistent with the diagnosis include a pericardial fat density, linear densities radiating from the parasternal aspect of the diaphragm consistent with blood vessels, and an abnormally high location of the diaphragm.[3] MRI has the added advantage of depicting the diaphragmatic defect directly in the coronal and sagittal plane.[5]

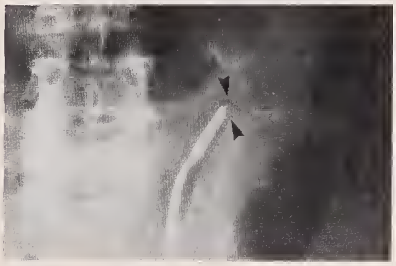
FIGURE 2. Lateral film of the chest shows an anterior mass (arrowheads).

Morgagni hernia is often forgotten in the differential diagnosis of large intrathoracic and mediastinal masses when obvious intra-abdominal organs are not present. The differential diagnosis includes entities such as pericardial cyst, large fat pad, lipoma, liposarcoma, or other neoplasm. Radionuclide liver scans have been reported to be useful in cases in which the herniated sac contains liver.[6] The diagnosis is often made when bowel loops are visualized in the hernia sac on an abdominal radiograph or barium enema examination. Even if no bowel is present within the sac, tenting of the bowel superiorly can suggest the diagnosis.[7]

Surgical repair can be performed via a thoracic or transabdominal approach.[7] The key to all methods of repair is to reduce the content of the hernia, excise the hernial sac completely, and close the defect in the diaphragm to prevent future recurrences.

This case provides an unusual presentation of an uncommon entity. Progressive herniation of omentum through the defect resulted in a large intrathoracic mass. In spite of the massive herniation of omentum, there was no evidence of bowel within the hernia at the time of CT or laparotomy.

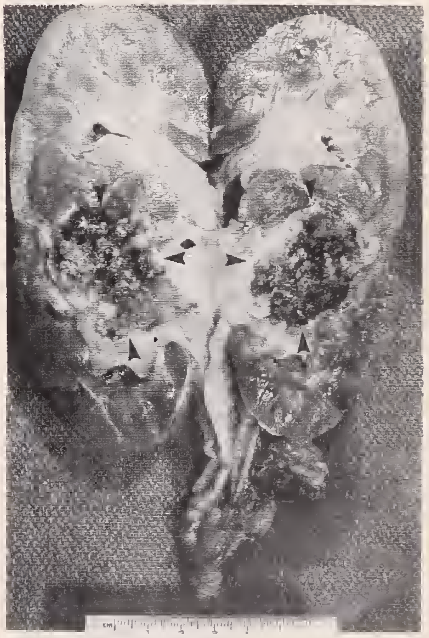
FIGURE 3. CT Scan of the chest demonstrates an anterior mass of fat density (arrowheads) containing fibrotic strands and vessels (arrows).

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REFERENCES

1. Kelly KA, Bassett DL: An anatomic reappraisal of the hernia of Morgagni. *Surgery* 1964; 55: 495–499.
2. Paris F, Tarazona V, Casillas M, et al: Hernia of Morgagni. *Thorax* 1973; 28:631–636.
3. Fagelman, D, Caridi JG: CT diagnosis of hernia of Morgagni. *Gastrointest Radiol* 1984; 9:153–155.
4. Schneidau A, Baron HJ, Rosin RD: Morgagni revisited: A case of intermittent chest pain. *Br J Radiol* 1982; 55:238–240.
5. Yeager BA, Guglielmi GE, Schiebler ML, et al: Magnetic resonance imaging of Morgagni hernia. *Gastrointest Radiol* 1987; 12:296–298.
6. Comer TP, Schmalhorst WR, Arbegast NR: Foramen of Morgagni hernia diagnosed by liver scan. *Chest* 1973; 63:1036.
7. Thomas TV: Subcostosternal diaphragmatic hernia. *J Thorac Cardiovasc Surg* 1951; 63:750.

Transitional cell carcinoma of the renal pelvis in an incompletely duplicated collecting system

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Primary tumors of the renal pelvis and collecting system are relatively uncommon, comprising about 7% of all renal tumors and less than 1% of all genitourinary tumors.[1] Duplication of the renal pelvis and ureters is one of the more common urinary tract anomalies,[2] although neoplasms encountered within this anomaly are rare.[3]

We present, to the best of our knowledge, the first case of a primary transitional cell carcinoma of the renal pelvis in an incompletely duplicated collecting system.

CASE REPORT

A 66-year-old man was admitted with a three-day history of painless gross hematuria associated with progressive weakness and near collapse at home. His urologic history dated back approximately two years, with an isolated episode of painless hematuria which he chose not to have evaluated. He denied easy bruising or prolonged bleeding. There was no history of sickle cell disease. He had smoked one pack of cigarettes a day for 15 years. His medical history was otherwise unremarkable.

On physical examination, the patient's temperature was within normal limits, blood pressure was 110/60 mm Hg; and pulse was 98/min. The abdomen was soft, with no palpaple masses. There was no costovertebral tenderness. The external genitalia were normal, and the prostate was mildly enlarged.

Laboratory results on admission were significant for a hematocrit of 16.7%. The blood urea nitrogen and creatinine levels were within normal limits. The coagulation profile, including the bleeding time, were within the acceptable range. Urinalysis confirmed the presence of blood. A plain film of the abdomen revealed multiple pelvic phleboliths. An intravenous urogram demonstrated non-visualization of the middle and lower pole collecting systems of the left kidney suggestive of a possible duplicated left collecting system with obstruction of the lower pole moiety (Fig 1).

A renal ultrasound revealed an enlarged left kidney with hydronephrosis of the lower pole. A double collecting system was seen with an echogenic irregular mass within the distended lower pole renal pelvis. For further evaluation, cystoscopy was performed. A single right and left ureteral orifice was noted situated in normal position, with blood effluxing from the left orifice. No masses were seen in the bladder. A left retrograde ureteropyelogram outlining the lower pole segment showed an abrupt cut-off with an irregular contour of the proximal left lower pole ureter (Fig 2). Percutaneous transfemoral renal angiography demonstrated a hypovascular lower pole left kidney segment.

The patient underwent a left nephroureterectomy with resection of a cuff of bladder. A polypoid cauliflower-like mass was identified filling the lower pole collecting system (Fig 3). Pathologic examination confirmed duplication of the collecting system with papillary transitional cell carcinoma grade II/IV in the lower limb of the duplex system. Distal ureteral fusion was noted. A follow-up cystoscopic examination at three months failed to reveal recurrence within the bladder.

DISCUSSION

Primary tumors of the renal pelvis and collecting system are relatively uncommon when compared with tumors of the renal parenchyma. They comprise about 7% of all renal neoplasms and less than 1% of all genitourinary tumors. The majority of these tumors are urothelial in origin. The relative incidence of transitional cell carcinoma is 51:3:1 for the bladder, renal pelvis, and ureter, respectively.[1]

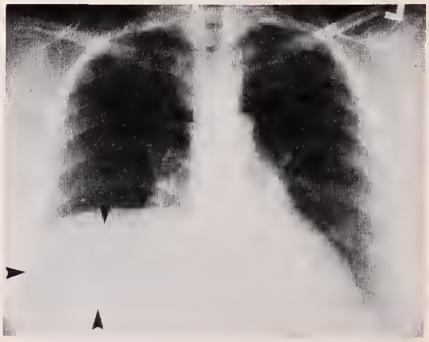

FIGURE 1. Intravenous urogram demonstrating contrast filling the upper pole segment of the left kidney (arrows) with nonvisualization of the lower pole segment.

Complete duplication of the renal pelvis and ureters, although one of the more frequently encountered urinary tract anomalies, is only found in about 1 in 125–150 autopsies.[2] Most individuals with duplicated systems remain completely asymptomatic and the anomaly is never discovered. Incomplete ureteral duplication with a common ureteral stem entering the bladder results from bifurcation of the ureteral bud after it has taken its origin from the mesonephric duct. Except for the so-called "yo-yo reflux," with an increased association with urinary tract infection and pyelonephritis, this anomaly is rarely of clinical significance.

The finding of a primary urothelial carcinoma in one of a pair of ureters in a duplicated collecting system has been previously reported on a number of occasions.[4–6] In contrast, the presence of primary urothelial carcinoma of the renal pelvis in a completely duplicated system has only been reported on a single occasion.[3] In that case, cystoscopy revealed grossly bloody efflux from the lowermost ureteral orifice, and the tumor was localized in the upper pole moiety of a right kidney unit.

The accurate uroradiologic diagnosis of transitional cell carcinoma of the renal pelvis depends on a number of complemen-

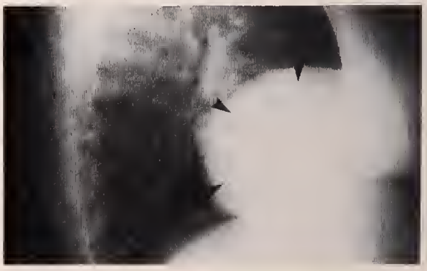

FIGURE 2. Left retrograde ureterogram demonstrating obstruction of the proximal lower pole ureter (arrows).

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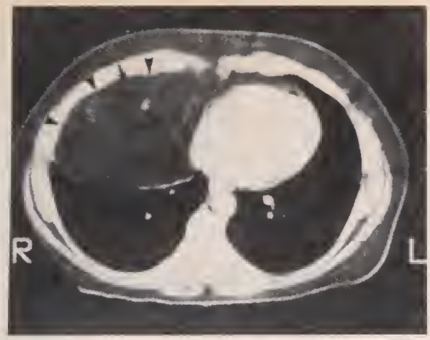

FIGURE 3. Gross bivalved specimen of the left kidney shows a cauliflower-like mass filling the lower pole of the renal pelvis (arrows).

tary imaging modalities. The intravenous urogram may enable the earliest possible identification of a filling defect. Lowe and Roylance[7] have categorized five distinct urographic patterns seen on intravenous urography associated with pelvic-filling defects. Similarly, the presence of the stipple sign represents the appearance of contrast material insinuating within the tumor fronds.[8] Retrograde pyelography may provide detailed mapping of the collecting system; that may not have been apparent on intravenous urography.

Sonography may help differentiate renal pelvic filling defects. Unlike calculi, tumors generally do not produce shadowing. They frequently are less echogenic than fat. At times, they may be confused with blood clots before clot breakdown.

Computed tomography may identify transitional cell tumors of the renal pelvis larger than one centimeter. These tumors are hypovascular and the renal contour is well preserved. Invasion of the renal vein and vena cava have been documented in isolated cases.[9]

Angiography plays a small role in the diagnosis of transitional cell carcinoma of the renal pelvis. These tumors may be hypovascular or occasionally avascular. Similarly, at the present time magnetic resonance imaging does not offer any additional information. Unlike the case of renal pelvic transitional cell carcinoma previously reported, the tumor described in this case report was localized to the lower pole of an incompletely duplicated collecting system.

REFERENCES

1. Richie JP: Carcinoma of the renal pelvis and ureter, in Skinner D, Lieskovsky O (eds), *Diagnosis and Management of Genitourinary Cancer.* WB Saunders Co., 1989, pp 323–336.
2. Perlmutter AD, Retik AB, Bauer SD: Anomalies of the upper urinary tract, in Walsh PE, Gittes RF, Perlmutter AD, and Stanley TA (eds), *Campbell's Urology.* Philadelphia, WB Saunders Co., 1986, pp 1665–1759.
3. Budd JS: Primary transitional cell carcinoma of renal pelvis in a duplicated collecting system. *Br J Clin Pract* 1987; 41:1063–1064.
4. Banya Y, Abe T, Sasaki H, et al: A case of primary ureteral carcinoma in the duplicated renal pelvis and ureter diagnosed by transurethral uretero-renoscopy. *Hynyokika Kiyo* 1986; 32:453–461.
5. Kurnom H, Nanbu K: A case of primary ureteral carcinoma in the duplicated ureter. *Rinsho Hinyokika* 1981; 35:486–471.
6. Tudor RG, Clear JD: Conservative surgery in the management of carcinoma in a duplex ureter. *J R Coll Surg Edin* 1986; 31:323–324.
7. Lowe PP, Roylance J: Transitional cell carcinoma of the kidney. *Clin Radiol* 1976; 27:503–512.
8. Mclean GK, Pollack HM, Banner MP: The "stipple sign" urographic harbinger of transitional cell neoplasms. *Urol Radiol* 1979; 1:177–179.
9. Gatewood OB, Goldman SM, Marshall FF, et al: Computed tomography in the diagnosis of transitional cell carcinoma of the kidney. *J Urol* 1982; 127:876–887.

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Proper obstetric care for women in New York State

TO THE EDITOR: I commend Doctor Richard Schwarz for his comments regarding the issues surrounding the concerns over the delivery of proper obstetric care to the women and families of New York.[1] He was clearly on target in his syllogisms that the only well-established denominator for the lowering of perinatal morbidity and mortality are adequate prenatal care and supervision. In that vein, the United Nations, through UNICEF, has now determined that a measure of the health of any nation is the Under Five Mortality Rate, which is simply the number of living children at age five per 1,000 born in any demographic area. Prenatal care is uniquely reflective of that index as well.

I was disappointed, however, that Dr Schwarz seemed to eschew the real causes behind the shame of not only Syracuse and other urban areas in the state, but also the rest of the country as well. The latest UNICEF report on the state of the world's children singles out the United States because of our continuing rise in child poverty over these past ten years. They point out that contrary to what Dr Schwarz states, the (sic) stock image of the child of color born to an unemployed, unmarried mother on welfare in one of America's big cities is a description that matches only 10% of our nation's so-called poor children. Although inner-city blacks and Hispanics have more than their population share, a clear majority of our many millions of poor children are white, and they live outside of urban centers. Moreover, contrary to stereotypic myths, most live in families with one or two children and in households where at least one of the parents is working.[2] The UNICEF report conclusions are stark. The two reasons given are somewhat embarrassing. First and what Dr Schwarz detailed, there is an erosion of prenatal services to the nations under-privileged; and second, there is the ever steady decrease in the real wages of America's unskilled working force.[2]

When Dr Schwarz called for a change in our policies, I regret that he was not more explicit in his demands. He wrote of the role Governor Mario Cuomo and the New York State Assembly might play, and of the cooperation needed by and with the March of Dimes, the Council of Churches, the Kiwanis, and the League of Women Voters. That all represents mere palaver unless the source of the problem is clearly identified, exposed, and held responsible. Any other suggested solutions become disingenuous. I refer, of course, to the federal government.

Although there is always room for improvement, these past 12 years of Reagan/Bush economics have been devastating to the poor women of America. Just about every social and health program enacted in the country since the New Deal of Franklin Delano Roosevelt has been gutted and underfunded. It was the Women, Infants, and Children (WIC) program of the 1970s that accounted for the drop in perinatal mortality in the early 1980s. That mortality is now on the rise.

As has been stated before, when one asks, "Where will we get the money from?" the answer is easy, "From where it is going." That is precisely what Dr Schwarz avoided stating. The answer does not lie in cooperation with charity groups or in having Albany's legislators adjust one needy group's funds for another. It does not rest with the Governor proposing legislation that replaces the need for infant compensation plans for neurologically impaired infants. It lies squarely with the White House and Congress and in a change in their priorities.

Instead of refurbishing the Emir of Kuwait's palace, we should be spending on America's needs for hospitals, clinics, and schools. Rather than Star Wars, B2 bombers, and Trident submarines, money is needed for America's roads, bridges, libraries, and immunizations for all of our children so that 40 more of them do not die from simple rubella as they did last year. Dr Schwarz must know, as does Governor Cuomo (he stated as much during a recent television interview), that New York City pays more into the state coffers that it receives, and New York State does the same for the federal government. In the union of 50 states as we know it, we must all do our share; it is the union that must contribute to the general welfare of the states.

Dr Schwarz finished with a plea that we all share in the responsibility for maximizing "every child's opportunity for success." I agree. Now let him and all of us point the finger at our egregious national policy. No real change will come about without a change at the top.

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1. Schwarz R: Providing access to obstetric care in New York State. *NY State J Med* 1992; 92:235–236.
2. UNICEF: *The State of the World's Children*, Oxford University Press Inc, 1992.

In reply. Many of Dr Sloan's points are very well taken. It is important to recognize, however, that my editorial commentary was provoked by the article by Korenbrot et al[1] which specifically addressed the issues of professional liability and access to obstetric services in New York State. I fully agree that professional liability is only one part, albeit a significant one, of the problem of access to health care. A solution to the liability problem in New York would surely not resolve all of the state's difficulties in providing maternity services, and it would clearly not, as a stand alone measure, reduce perinatal mortality to desired levels. It would, however, permit more of the state's obstetricians and perhaps family physicians to continue, or resume, the provision of obstetrical care and hopefully, as well, prevent the out flow of newly qualified practitioners to other states.

I agree with Dr Sloan that the ultimate solutions to America's health care crisis cannot be found at the local, or even the state, level. We must have major reform, both medical and social, to accomplish that purpose and it must come nationally. I also agree that we in the health professions must be active in that process and that we must do it with the sense of urgency it merits. Let us not, however, miss an opportunity to press for this piece of reform in professional liability which, although not a panacea, can provide a significant degree of relief.

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1. Korenbrot CC, Phibbs C, Lamb-Abel BJ: Professional liability reform and access to medicaid obstetric care in New York State. *NY State J Med* 1992; 92:237–245.

Cigarette advertising in news magazines

TO THE EDITOR: I recently cancelled my office subscription to *US News and World Report* as it consistently had a full-size cigarette advertisement on the back cover.

I believe that physicians should keep *all* cigarette advertising out of their waiting rooms. Magazines that prominently display such ads have no place in any medical setting. If health professionals refuse to subscribe to publications with cigarette advertising and let the publishers know why, the publishers may begin to get the message.

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Idiopathic segmental infarction of the greater omentum

TO THE EDITOR: Almost a century ago, in 1896, idiopathic segmental infarction (ISI) of the greater omentum was first described by Bush.[1] Since then over 150 cases have been reported with the diagnosis of ISI usually made during laparotomy.[2,3] We report the clinical features of another case.

Case Report. A 53-year-old male with past medical history of left inguinal herniorrhaphy 12 years before admission, presented to the emergency room with a chief complaint of right upper quadrant pain described as pressure-like in nature. The pain began three days before admission after he drank six cans of beer. It was acute in onset, non-radiating, with varying intensity, and not associated with nausea, vomiting, diarrhea, chills, or fever. It was relieved only partially by analgesics. When it became more severe the patient came to the emergency room. The family history was unremarkable.

On admission, the patient was found to have the following: blood pressure, 120/90; pulse, 80 beats/min; respiration rate, 16/min; temperature, 98.6°F. The physical examination was unremarkable, with the exception of the abdomen, which was soft and mildly distended with right upper quadrant tenderness. Murphy's sign was positive with localized rebound. There was no hepatosplenomegaly. The rectal examination was normal, and the stool was negative for occult blood. Laboratory evaluation revealed the following: white blood cell (WBC) count, 10,200/μL; hemoglobin, 15.2 g/dL; hematocrit, 45.4%, platelets, 291×10^9/L; lymphocytes, 29.9%, monocytes, 15.8%, granulocytes, 64.3%; prothrombin/partial thromboblastin time, 12/27; amylase, 34 U/100 mL. The sequential multiple analysis, 18, was normal except for a serum glutamic pyruvic transaminase of 46 U/mL, and a lactic dehydrogenase of 301 U/mL. The urine was negative for red and white blood cells.

The patient was admitted with a clinical diagnosis of acute cholecystitis. A hepatoiminodiacetic acid scan and ultrasound were normal, as was a computed tomography (CT) scan of the abdomen. Over the next 24 hours, the pain became more severe, and the patient was considered to have a surgical abdomen. There was no fever, and the complete blood cell count remained completely normal. The patient was taken to the operating room with a presumed diagnosis of acute appendicitis.

At surgery, a small piece of hemorrhagic omentum adherent to the transverse colon was found twisted around its pedicle. Pathologic examination revealed infarcted adipose tissue. The patient was discharged home on the fifth day post-up.

Discussion. Primary torsion of the greater omentum is a rare cause of abdominal pain.[4] The greater omentum consists of a double sheet of flattened endothelium. Epiploic vessels, lymphatics, and nerves pass in areolar tissue between the sheets and fat is variable. Torsion occurs when the omentum twists on its long axis, causing vascular compromise. Omental torsion has been classified as primary or secondary. Primary torsion of the greater omentum is a rare cause of abdominal pain.

The etiology is not clear, and several theories have been postulated. Because of the high incidence of involvement of the right side of the omentum, as in our patient, developmental abnormalities have been suggested.

Rarity and lack of unique clinical presentation make the diagnosis difficult. The nonspecific presentation can mimic cholecystitis or acute appendicitis. Because there is no diagnostic test available that can rule out acute appendicitis, many of these patients are taken to the operating room with a presumed diagnosis of acute appendicitis. Ceuterick[5] reported a case in which the diagnosis was made by CT scan. In our case, all diagnostic tests were normal, but clinically the patient had a surgical abdomen.

Although the disease may have a benign natural course, there is a possibility of adhesions which might necessitate hemicolectomy. There is also a report by Patchell[6] of a patient who died of sepsis.

Because of the possibility of these complications, resection of the infarcted omentum is the treatment of choice. This condition should be considered in the differential diagnosis of an acute abdomen when no apparent cause is found, after appropriate diagnostic studies, and when there is an absence of fever and a normal complete blood count.

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1. Bush P: A case of hemorrhage into the greater omentum. *Lancet* 1896; 1:286.
2. Tolenaar PL, Bast TJ: Idiopathic segmental infarction of the greater omentum. *Br J Surg* 1987; 74:1182.
3. Quinn AD, Jothi RK: Idiopathic segmental infarction of the greater omentum. *Postgrad Med* 1986; 79:133–134, 139–140.
4. Appleby JP, Simpson WT: Primary omental torsion [letter]. *Can J Surg* 1989; 32:7.
5. Ceuterich L, Baert AL: CT diagnosis of primary torsion of greater omentum. *J Comput Assist Tomogr* 1987; 11:1083–1084.
6. Patchell RD: A fatal case of idiopathic omental infarction. *W V Med* 1977; 73:29–30.

Mucinous cystic neoplasm of the pancreas

TO THE EDITOR: Cystic neoplasms are an uncommon group among pancreatic tumors, accounting for 10% to 15% of pancreatic cysts[1,2] and about 1% of all pancreatic neoplasms.[3] Because of advances in noninvasive diagnostic procedures, these lesions are more frequently detected and surgically treated than they were in the past. Among true cysts, the mucinous neoplasms are unique because of their potential for associated carcinoma. These neoplasms are morphologically heterogenous and show multidirectional differentiation. Early detection of mucinous cystic tumors is important because the prognosis after their complete resection is better than that associated with any other malignancy of the exocrine pancreas.

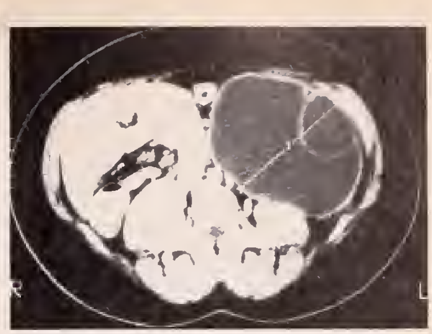

FIGURE 1. CT scan showing a large septated mass in the left upper quadrant.

Case Report. A 20-year-old woman was admitted because of vague pain in the left upper quadrant for the last six months. The pain was colic-like in nature, did not radiate, and was not associated with nausea or vomiting. The patient had no history of alcohol abuse or trauma. Her vital signs were stable. Abdominal palpation revealed a poorly defined mass in the left upper quadrant. This mass was mildly tender, ballottable, and soft. All laboratory values, including the serum amylase, were within normal limits. Radiographs revealed an abnormal soft-tissue density in the left upper quadrant that was displacing small bowel loops. A sonogram showed a large septate cystic mass located predominantly in the left upper abdomen. This mass was displacing the spleen and left kidney.

CT scan of the abdomen showed a large cystic multiseptated mass measuring 13 × 9 cm, in the left upper quadrant adjacent to the body and tail of the pancreas (Fig 1). Endoscopic retrograde cholecysto-pancreatography (ERCP) showed a normal pancreatic duct in the head and body, but a cystic structure in the tail of the pancreas.

Exploratory laparotomy revealed that the large cystic mass arose from and was attached to the distal 5 cm of the pancreas (Fig 2). A distal pancreatectomy was done with the spleen being preserved. The patient had an uneventful recovery. Histologic examination demonstrated that the mass was a mucinous cystadenoma with no malignant change. Follow-up one year after surgery was unremarkable.

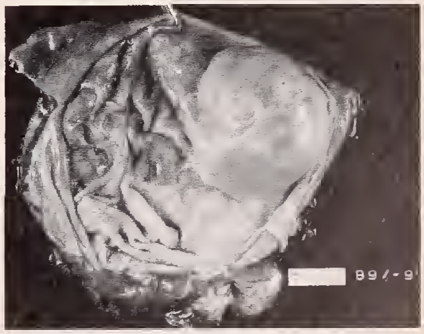

FIGURE 2. Operative specimen showing multiseptated cyst with the portion of the pancreas attached to the inferior portion of the cyst.

Discussion. Pseudocysts constitute approximately 90% of all cystic lesions of the pancreas. True cysts are lined by epithelium whereas pseudocysts are lined by fibrous tissue. The term "cystadenoma of the pancreas" has become entrenched in the literature, although it clearly denotes more than one entity. Although there are probably multiple types of cystadenomas, two of them, serous cystadenoma and mucinous cystadenoma, have been clearly defined.[4,5] Since publication of the landmark paper of Compagno and Oertel[4] in 1978, it has been customary to distinguish between these two varieties of cystadenoma. A crucial point in this distinction is that serous cystadenomas are universally benign, whereas mucinous cystadenomas are distinct tumors, all of which, in time, may develop aggressive characteristics and become mucinous cystadenocarcinomas. Mucinous cystadenomas are notorious for their combination of areas of benign, atypical, and malignant epithelium.[6] Compangno and Oertel[4] recommended that these tumors no longer be referred to a "mucinous cystadenomas" and "mucinous cystadenocarcinomas" but, instead, as "mucinous cystic neoplasms with latent or overt malignancy."

Serous cystadenomas are most common in elderly patients. However, mucinous cystadenomas occur predominantly in younger patients, as in the case we present. In one review, the female-to-male ratio of patients with mucinous cystadenomas was approximately 8:1.[4] The most frequent presenting symptom is vague abdominal pain, as in our case. Occasionally, these tumors cause obstructive jaundice. Fortunately, these tumors apparently occur in the tail of the pancreas more frequently than in the head.

Plain radiographs of the abdomen reveal areas of pancreatic calcification in 10% or more of all patients with cystadenoma. CT scan allows accurate localization of the mass, visualization of septae within it, and demonstration of invasion of the tumor into adjacent structures, all of which can help differentiate a cystadenoma from a pseudocyst. It has been suggested that ERCP could be used to distinguish cystic tumors from pseudocysts, because the pancreatic duct communicates with 70% of all pseudocysts, whereas neoplastic cysts are usually separate from the pancreatic duct. Warshaw et al[7] found no instance of communication with the pancreatic duct in pancreatograms of 37 patients with neoplastic cysts. However, our patient's cyst did communicate with the pancreatic duct, and this communication was shown by ERCP.

Histologically, mucinous cystic tumors consist of tall columnar epithelium with papillary projections. The tumors may be unilocular or multilocular. Mucin stains are highly positive. Thorough examination of multiple sections from these tumors is essential to detect malignant foci.

The surgical procedure depends on the location of the tumor. Distal pancreatectomy is the procedure of choice for lesions in the body or tail of the pancreas. Lesions in the head of the pancreas require pancreaticoduodenectomy. The prognosis after complete resection of the tumor is better than that associated with conventional carcinomas of ductal cell type.

It is generally recommended that cystic tumors of the pancreas be resected to relieve the patient's symptoms and to treat the cancer, if present. Some investigators[8] have suggested that asymptomatic serous cystadenoma, the only cystic tumor that has never been malignant, can be observed safely, especially in poor risk elderly patients. Although this practice is theoretically sound, in practice it is difficult to determine with certainty that a given lesion is a serous cystadenoma and not one of the other, more ominous varieties. We recommend that any true cystic lesion of the pancreas needs resection, which is also recommended by George et al.[9]

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1. Wolfman NT, Ramquist NA, Karstaedt N, et al: Cystic neoplasm of the pancreas: CT and sonography. *AJR* 1982; 138:37–41.
2. Didolakar MS, Malhotra Y, Holyoke ED, et al: Cystadenoma of the pancreas. *Surg Gynecol Obstet* 1974; 140:925–928.
3. Cubilla AL, Fitzgeral PJ: Classification of pancreatic cancer. *Maya Clin Proc* 1979; 54:449–458.
4. Compagno J, Oertel JE: Mucinous cystic neoplasms with overt and latent malignancy (cystadenocarcinoma and cystadenoma). *Am J Clin Patol* 1978; 69:573–580.
5. Campbell JA, Cruickshank AH: Cystadenoma and cystadenocarcinoma of the pancreas. *J Clin Pathol* 1962; 15:432–436.
6. Howard JM: Cystic neoplasms and true cyst of the pancreas. *Surg Clin North Am* 1989; 69:651–665.
7. Warshaw AL, Compton CC, Lewandroski K, et al: Cystic tumors of the pancreas: New clinical, radiologic and pathologic observations in 67 patients. *Ann Surg* 1990; 212:432–446.
8. Compagno J, Oertel JE: Microcystic adenomas of the pancreas (glycogen-rich cystadenomas): A clinicopathologic study of 34 cases. *Am J Clin Pathol* 1978; 69:289–298.
9. George DH, Murphy F, Michalski R, et al: Serous cystadenocarcinoma of the pancreas: A new entity? *Am J Surg Pathol* 1989; 13:61–66.

COPE'S EARLY DIAGNOSIS OF THE ACUTE ABDOMEN

Eighteenth Edition. Revised by William Silen, MD. 301 pp, illustrated. New York, Oxford University Press, 1991. $19.95 (softcover)

The 18th edition of this volume, originally authored by Zachary Cope and revised for the third time by Dr William Silen, is one of the few examples of how a complicated clinical entity can be presented in such a way that the information is useful to medical students and physicians in training while serving as a reference text for any professional whose practice, includes patients with this condition.

At a time when ancillary diagnostic procedures (X-ray, CAT scan, echography) and even a large number of laboratory tests seem to be mandatory in order to arrive at a diagnosis and treatment plan, this book emphasizes the clinical examination of the patient. In all the chapters, the authors follow a logical thinking process based on the history of the patient's illness, the clinical findings, and the occasional ancillary test that all lead to logical diagnostic and therapeutic conclusions, a trend which this reviewer supports and applauds.

This 290 page book is divided in three parts. The first 60 pages describe the general principles involved in the diagnosis of the acute abdomen including some laboratory and radiological considerations. The emphasis of this and all other sections is on early diagnosis followed by surgical consultation. The reliance in laboratory findings for a diagnosis is discouraged. Rather, such observations are to be used to optimize the physiological status of the patient and minimize intraoperative or postoperative morbidity.

The largest section of the book is directed to the clinical description of 16 different conditions ranging from acute appendicitis to the acute abdomen caused by vascular lesions, as well as gynecological and urological diseases presenting with abdominal pain. In all these chapters, which can easily be read in whatever order the reader chooses, the authors integrate the patient's history and a detailed clinical examination to arrive at a possible diagnosis and a therapeutic decision. The pitfalls associated with each of these conditions are carefully described and, when necessary, the value of an ancillary diagnostic test is described.

The final four chapters describe the postoperative course for patients who undergo laparotomy, tropical diseases associated with an acute abdomen, and the increasing and complicated subject of the management of the immune-compromised patient who presents with abdominal pain.

As a surgeon, I enjoyed and value this book. It is a "must" on the desk or in the clinic for any physician who may be asked to care for this type of patient.

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HANDBOOK OF SKIN CLUES OF SYSTEMIC DISEASES

Second edition. By Paul H. Jacobs, MD, and Todd S. Anhalt, MD. 150 pp. Malvern, Pa, Lea & Febiger, 1992. $15.95 (softcover)

There are multiple short, pocket-size books available for the medical student or physician requiring a dermatological text. This particular soft cover's main purpose is based on "clues" of specific cutaneous lesions with their relationships to underlying systemic disease. The authors, both dermatologists at Stanford University Medical Center, bring a great deal of experience to this field and have compiled a detailed list of systemic conditions to be considered when confronted with appropriate skin lesions. As such, this book is an interesting addition to the medical literature.

The book contains 21 separate clues ranging from bizarre skin lesions through pigmentary changes, papules and/or nodules, purpura, and bullous lesions, to include a new chapter on nail changes. A full listing of suspected diseases associated with these lesions is included, with two further listings (ie, "Look For or Perform" and "Order"). This enables the reader to zero in on appropriate clinical areas of concern, as well as possible diagnostic aids, frequently including skin biopsies, to elucidate the systemic cause.

The listing of clues is relatively comprehensive, but in a short text of 150 pages it is, obviously, impossible to consider adequately all the potential systemic diseases associated with cutaneous lesions. As such, the book suffers from an excess of "perform clinical evaluations" or "order skin biopsy." If this book is targeted toward the non-dermatologist, one may question a non-dermatologist blindly doing a skin biopsy, which is then frequently read by a non-dermatopathologist, causing further confusion.

Another caution concerns the lack of organization in the suspect list (ie, under the heading Telangiectases is a list of 17 conditions ranging from infections, hereditary syndromes, collagen vascular disease, and Kaposi's sarcoma). Grouping under subheadings would make for much easier reading.

The new chapter on Nail Diseases is welcomed because this is frequently an area of major difficulty for the non-dermatologist seeking to associate nail changes with systemic disease. This is extremely well done with groupings of conditions, including inter alia color changes, clubbing, pigmented bands, and splinter hemorrhages.

The book is easy to read and, with the exception of a few overly long rambling lists, is well organized. There is a short glossary of standard dermatological terms with an index that includes both the skin lesions and all the associated systemic diseases.

The virtues of the second edition outweigh its defects, and for practitioners requiring a concise, ready reference of skin lesions and systemic diseases, this text meets the authors stated objectives of establishing whether or not systemic disease may be associated with presenting cutaneous lesions.

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THE AMERICAN DENTIST: A PICTORIAL HISTORY WITH A PRESENTATION OF EARLY DENTAL PHOTOGRAPHY IN AMERICA

By Richard A. Glenner, DDS; Audrey B. Davis, PhD; Stanley B. Burns, MD. 194 pp, illustrated. Missoula, Montana. Pictorial Histories Publishing Co, 1990. $19.95 (hardcover)

With the fall of Rome, medicine in the western world went into a severe decline. About the only place where simple medicine and surgery were practiced was the monastery. The monks were assisted in performing their simple surgical procedures by the local barbers. However, in 1163, a council of bishops convened by the Pope decreed that "...shedding of blood was incompatible with the holy office of the clergy." Henceforth, all monks and priests were enjoined from doing any manner of surgery. This left the field to their erstwhile assistants, the barbers. By the 14th century, some barbers took for themselves the title "barber-surgeon" because they were doing more surgery than barbering. By the 1600s, some of these practitioners ceased their barbering and confined themselves to surgery and became known as surgeons. In the early 1700s, a number of these surgeons limited themselves to the practice of dentistry, and in 1728, Pierre Fauchard, the father of modern dentistry, himself a Parisian dentist, coined the term "surgeon-dentist."

At that time, France was the leading center of dental practice. In early Colonial

America, however, artisans of all types undertook to meet the dental needs of the population. Paul Revere, the patriot and silversmith, is a good example; he practiced all branches of dentistry for about seven years.

Many French dentists, however, immigrated to this country, and by the early decades of the 19th century, the center of dental knowledge, innovation, and practice had shifted to the United States. American dentistry has since then never lost its pre-eminent position.

The first dental school in the world was established in Baltimore in 1839, with several more being founded in the years immediately following. However, this could hardly meet the need for dentists in a fast growing nation. Thus, most dentists came from two sources: from those physicians who devoted a part or all of their time to the practice of dentistry, and from those individuals who studied with other established dentists as preceptoral students.

American dentistry continues, to this day, to lead the world, and has set standards followed in every land. The story of the development of American dentistry is told in this fascinating book in a concise, yet lively, fashion, using an abundance of photographs.

Stanley B. Burns, MD, one of the authors, is an opthalmic surgeon, who is a noted historian of photography. His personal archives of over 100,000 vintage photographs contain some 20,000 images devoted to medicine and dentistry. The introductory portion of the book, almost 50 pages long, is written by Dr Burns, and includes hundreds of daguerrotypes, ambrotypes, and other early photographs of dental practitioners and their offices. One surprising bit of information that this reviewer learned is that the first camera to be patented in this country was invented by a dentist. Burns' pictures range from the humorous to the topical, and all of them add immensely to the value of the book. Not to be overlooked, however, is the excellent short history of photography Dr Burns provides.

The remaining 21 chapters trace the development of dentistry in America from the earliest days of the itinerant dentist to the modern office. Almost every facet of dentistry in America is covered. For example, dental education, women in dentistry, dental instruments, the dental office, and military dentistry are some topics. There is even a fascinating chapter on dentistry in the movies with wonderful, charming prints from movies starring such actors as Laurel and Hardy and Mickey Rooney.

The authors of this section of the book bring to their work a lifetime of involvement in the history of the healing professions. Richard A. Glenner, DDS, a practicing dentist in Chicago, possesses the greatest collection in the world of pictures of dental equipment, dental instruments, and other things relating to all aspects of the profession. He is a renowned dental historian and has been a member of the American Academy of the History of Dentistry for many years. He was the 1983 recipient of that Academy's prestigious Hayden-Harris Award. He is also the author of *The Dental Office: A Pictorial History* (1984) which deals with the growth and development of the surroundings of the dentist.

Audrey B. Davis, PHD, is the Curator of the Division of Medical Sciences of the Smithsonian Institution. Recognized as one of the world's authorities on medical and dental history, Dr Davis has published widely and has authored a number of books on medical history and medical instruments.

The collaboration of these two prominent historians has made for a fascinating book. The wealth of photographs, literally hundreds of them, makes for interesting reading. What is even more remarkable is the very low price of $19.95. The format is a large 9 × 12; the paper is of heavy coated stock, allowing for great clarity of the photographs. The work has an extensive list of references, happily gathered at the end of the book where they do not interrupt the flow of the narrative. A comprehensive index adds to the book's value as a reference tool.

Anyone even remotely interested in the history of the healing professions should find this book a gratifying, as well as pleasurable source of information about one of the most important branches of the healing arts.

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STRATEGIC HEALTH PLANNING: METHODS AND TECHNIQUES APPLIED TO MARKETING AND MANAGEMENT

By Allen D. Spiegel, PHD, and Herbert H. Hyman, MD. 440 pp, Norwood, NJ, Ablex Publishing Corp, 1991. $45.00 (softcover)

This unique book introduced administrators, practitioners, and students to the concepts, methods, and current state of the art in strategic health planning. Using a six-step planning process approach (problem identification; resource assessment; alternatives generation and analysis; selection and ranking of priorities; implementation; and evaluation), the authors delineate how the strategic planning process, commonly employed in the for-profit business sector, is rapidly being adopted by health care providers and institutions. The increasing trend toward commodification of health care, characterized by federal health policies since 1981, has compelled health care providers to adopt and implement strategic planning to survive in an increasingly competitive and dynamic marketplace. The authors emphasize that institutions must concentrate on developing and controlling segments of the health care market, while de-emphasizing their traditional roles as producers of comprehensive services. As the authors point out in the first chapter, the orientation of strategic health care planning toward the production of profits and consumers who can pay for services sets it apart from comprehensive health planning, which focuses on meeting the health needs of the population as a whole.

Hyman and Spiegel, together and individually, have created a number of texts for health planning over the past two decades. Their writing emphasizes both the systematic and comprehensive in reviewing grounded theory and techniques. In this book, the authors make extensive use of related health planning literature in developing their approach to the strategic planning process, and illustrate techniques discussed in the book with exhibits, tables, and graphics. They discuss the latest health policy issues, including the acquired immunodeficiency syndrome (AIDS), home health services, and cost containment, relating each to the strategic planning process. Although the framework of the book is methodological, the authors review a wide variety of planning techniques, both classical and novel, repeatedly reminding the user that he or she must choose the technique that best fits the organization's needs and goal. The book would best be used as a technical reference by executives, administrators, and planners. As with any reference handbook, the reader should keep in mind that planning products, particularly computer software packages, are constantly changing. The authors did not attempt to evaluate the relative merits or general practitioner acceptance of particular planning tools, so the reader who wishes to know which product has been tested and widely adopted in the field will have to do additional investigation beyond this volume.

The organization of the book is appropriate as a strategic health planning handbook. It could also be used as a secondary technical text in health planning and management courses at both the graduate and undergraduate levels. In terms of content, the book is uneven. The book would have benefited from more careful editing. Parts of the book, including the preface, are poorly written. In addition, the authors make use of non-words, terms and jargon (eg, et al's [sic]; 2,000 [in reference to the year]; repeated use of *re*), which detract from the usability of the book as an academic text.

The authors commitment to systematic exposition makes this more than a static tool kit. Dr Hyman and Dr Spiegal imply that if all health actors made systematic use of population-based information, overall organizational productivity might increase, although the organization might become more segmented and differentiated as a result. There is an underlying interest expressed in a recommitment to policies for system-wide comprehensiveness, including total geographic population coverage and access. Thus, the "seen hand" of organizational sensitivity to all facets of the organization's capacities relative to aggregate area-wide effective demand, could ironically restore a more manageable societal commitment to comprehensiveness. This planning process might blend consensual problem identification, resource assessment, and evaluation, while signaling a more decentralized operational decision-making approach. A clever exploration of strategic management techniques therefore can also be read as a call for macro-system reconstruction, as an imperative for increasing the social productivity of health care organizations.

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COCAINE, AIDS, AND INTRAVENOUS DRUG USE

Edited by Barry Stimmel, MD. 124 pp, Binghamton, NY, Haworth Press, 1991. $8.95 (paperback)

This slender paperback is a collection of seven articles addressing various aspects of cocaine, AIDS, and intravenous drug use. These issues are of great medical and social concern. The articles are prefaced by an excellent discussion/critique by the editors. They are followed by a useful guide to current references. In addition, each article has an extensive reference list.

The editors acknowledge that while many important and urgent topics are discussed, other equally important and urgent topics are not addressed. They are to be congratulated for this compilation of articles for they are especially well-written and make some difficult concepts and ideas understandable to the intelligent lay reader. This is especially true for the first article, "Pharmacotherapeutic Interventions for Cocaine Abuse: Present Practices and Future Directions." In this article, the mechanisms of dopamine depletion, receptor sensitivity, cocaine-induced kindling, and serotonin depletion are discussed in relation to different treatment strategies.

The relationship of cocaine use and methadone treatment is considered from various perspectives, as are connections of cocaine addiction, intravenous drug use, and the acquired immunodeficiency syndrome (AIDS). The last article, "Why Women Partners of Drug Users Will Continue to be at Risk for HIV Infection," is extremely informative and well-done. The author, Dr Judith Cohen, discusses some of the strategies used to reach this group. It is in this context and others that "what works and what doesn't" makes this collection so valuable. It is often especially useful to know "what doesn't work." I was not convinced that the Health Belief Model proposed by Sorenson really "works." However, it is surely an important modality. This essentially cognitive approach involves explication of perceived threat (of AIDS), perceived benefit, self-efficacy (locus of control), and social support.

There is also an excellent article entitled "The Tacoma Syringe Exchange." These programs are always controversial. My understanding of the first New York City program is that the results were disappointing. In conclusion, this is a useful book, and I highly recommend it.

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FIBRINOLYSIS, THROMBOSIS, AND HEMOSTASIS: CONCEPTS, PERSPECTIVES, AND CLINICAL APPLICATIONS

By Sol Sherry, MD. 318 pp, illustrated. Philadelphia, Lea & Febiger, 1992. $59.50 (hardcover)

Dr Sol Sherry has elegantly combined the historical development of various hemostasis mechanisms with the modern day use of various coagulation modulators, especially in relationship to thrombosis and fibrinolysis. This book is easy to read and full of helpful summary tables. Emphasis is placed on mechanisms and pathways. The book is ideal for medical students, house staff, fellows, and attendings, and provides a good background for investigators in hemostasis. Throughout the book, interesting historical facts with appropriate photographs make for pleasant reading. The book reminds us of how quickly new findings get incorporated into patient care. The author was personally involved in the development of clinical applications of thrombolytic and fibrinolytic agents. He provides authoritative descriptions of protocol designs, results analysis, and comparisons of various methods available. The reader will find the book very useful for teaching purposes.

The chapters on thrombolytic and fibrinolytic therapy present great detail, especially regarding the management of acute myocardial infarction and pulmonary embolism. Thrombotic thrombocytopenic purpura (TTP) is not discussed in this book. Disseminated intravascular coagulation is only briefly described. The author's discussion of it is primarily limited to issues concerned with pathophysiology.

The author has appropriately stressed the fact that this is not a procedure manual and that it should be read in conjunction with a textbook for the clinical management of the patients. Several new findings are changing the management of patients with hemostatic defects. The availability of cloned products and better defined treatment strategies are useful for planning future trails, and these issues are nicely addressed in the last part of the book. Overall, this is a well written, concise book that will help to gain a better understanding of hemostasis.

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RENAL AND ELECTROLYTE DISORDERS

Fourth Edition. Edited by Robert W. Schrier, MD. 846 pp, illustrated. Boston, MA, Little, Brown & Co, 1992. $45.00 (hardcover)

The fourth and latest edition of this textbook, first published in 1976, aims to teach the reader through a pathophysiologic understanding of nephrological diseases. The book successfully provides the intended audience of medical trainees, faculty, and practitioners with a solid understanding of normal physiology, as well as pathophysiology and shows how the management of these disorders is derived from this understanding. Its format is traditional with an emphasis on text, supported by occasional figures, diagrams, and tables. Nevertheless, its emphasis on pathophysiology distinguishes this book from most standard texts intended for general rather than research-oriented audiences. Clinicians will appreciate its focus on diseases and their management rather than pure academic concepts. This book has become a standard among medium-sized textbooks of nephrology and the current edition continues in that tradition.

Dr Schrier is an internationally recognized authority in many areas of nephrology including water and sodium metabolism and acute renal failure. In addition, he has authored a number of nephrology texts and contributed chapters to many others. Dr Schrier has again assembled a strong group of contributors, most of whom are also widely recognized in the areas for which they have written chapters. Although there is some variability from chapter to chapter, most of the book is clearly presented, very readable, and flows easily from one section to the next.

The wide variety of topics in general nephrology, covered by this edition, are the same as in the third edition. All 15 chapters have been improved with updated information and the latest analysis of the areas covered as well as ongoing controversies. The following five chapters were completely rewritten: "Disorders of the Renin-

Angiotensin-Aldosterone System," "The Kidney in Hypertension," "Chronic Renal Failure: Manifestations and Pathogenesis," "Obstructive Uropathy: Pathophysiology and Management," and "Proteinuria and the Nephrotic Syndrome." All rewritten chapters are improvements over the strong versions of the third edition. The remaining chapters are also strong and cover water and electrolyte metabolism, acid-based disorders, acute and chronic renal failure, glomerulonephritis, and renal function in pregnancy. An added strength of the book is the attempt made by many contributors to put the nephrological diseases discussed into the context of common problems of medicine such as heart and liver failure, hypertension, and surgical problems. This contextual view of nephrology makes the book attractive to all individuals involved in the care of patients.

In summary, this fourth edition continues and even improves on the high standards set by the earlier editions. It should be a welcome addition to the libraries of all clinicians and those in training.

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HIPPOCRATES' HANDMAIDENS: WOMEN MARRIED TO PHYSICIANS

By Esther M. Nitzberg. 401 pp, Binghamton, NY, Haworth Press, Inc, 1991. $24.95 (softcover)

In this book, the author addresses the frustrations of doctors' wives. However, she offers little constructive help in stating that these wives are "...privileged and powerless" (pg 7). The author's personal belief that "An imperfect male doctor will always have greater value than his imperfect female spouse" (p 89) is a theme that runs through this autobiographical work. "I am only entitled as it relates to my husband the doctor" (p 215), she states, while using anecdotes from other women to validate her own weak self-esteem.

The author's mixed messages confuse her portrayal of victimized women as she continually repeats that self-esteem is low, while, on page 40, contends that the fringe benefits of being physicians' wives "can help self-esteem in a materialistic society."

As I read about her childhood, I felt that she had wandered into Philip Roth territory. She claims that she wed a doctor, in part "...to provide my mother with some personal success in this world" (p 210). Thus, she makes her mother rather one-dimensional. It is difficult for the author to be objective with regard to women's views and needs when she perceives her own role model this way.

I believe that the author could have better assisted Hippocrates handmaidens by acknowledging that the proverbial glass can be viewed as half-full instead of only half-empty. She could have suggested that a sense of humor is necessary when the telephone rings in the bedroom or during dinner. She could have brought out the similarities with the corporate wife, for example, or with any wife who was conditioned by the values of the 1950s or before, rather than allowing personal bitterness to become an invalid syllogism. By page 362, still expounding "insecurity, anger, poor self-esteem, and rage..." rather than offering, as a counselor, some constructive advice to the embittered, this book seems to be primarily a catharsis for the author. This may be of value to some readers but will have little appeal for others.

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BOOKS RECEIVED

BIOETHICS

African-American Perspectives on Biomedical Ethics. Edited by Harley E. Flack and Edmund D. Pellegrino. 223 pp. Binghamton, NY, The Haworth Press, 1992. $29.95 (hardcover)

Euthanasia Is Not The Answer: A Hospice Physician's View. By David Cundiff, MD. 200 pp. Totowa, NJ, The Humana Press, 1992. $17.95 (hardcover)

When Medicine Went Mad: Bioethics and the Holocaust. Edited by Arthur L. Caplan, PHD. 372 pp. Totowa, NJ, The Humana Press, 1992. $22.50 (hardcover)

When Others Must Choose: Deciding for Patients Without Capacity. By The New York State Task Force on Life and the Law. 288 pp. New York, The New York State Task Force on Life and the Law, 1992. $9.00 (softcover)

CARDIOLOGY

Rapid Analysis of Arrythmias: A Self-Study Program. By Emanuel Stein. 229 pp, illustrated. Philadelphia, PA, Lea & Febiger, 1992. $24.95 (softcover)

The Puzzle People: Memoirs of a Transplant Surgeon. By Thomas E. Starzl, MD. 363 pp. Pittsburgh, PA, University of Pittsburgh Press, Inc., 1992. $24.95 (cloth)

GASTROENTEROLOGY

The Office Management of Digestive Diseases. By Joseph T. Danzi and Joseph A. Scopelliti. 233 pp. Philadelphia, PA, Lea & Febiger, 1992. $39.50 (softcover)

GERIATRICS

Congregate Housing for the Elderly: Theoretical, Policy, and Programmatic Perspectives. Edited by Lenard W. Kaye, DSW, and Abraham Monk, PHD. 190 pp. Binghamton, NY, Haworth Press, 1991. $14.95 (softcover)

HEALTH CARE

Marketing Management Issues in Ambulatory Health Care. Edited by Charles D. Schewe, PHD, MBA. 198 pp. Binghamton, NY, The Haworth Press, 1992. $29.95 (hardcover)

Alternative Medicine in Britain. By Mike Saks. 271 pp. New York, Oxford University Press, Inc, 1992. $65.00 (hardcover)

Increasing Physician Involvement In Quality Improvement Programs. Edited by Edward C. Geehr, MD. 66 pp. Tampa, FL, The American College of Physician Executives, 1992. (softcover)

HEMATOLOGY

Fibrinolysis, Thrombosis and Hemostasis: Concepts, Perspectives, and Clinical Applications. By Sol Sherry, MD. 318 pp, illustrated. Philadelphia, Lea & Febiger, 1992. $59.50 (hardcover)

MEDICAL HISTORY

Preserve Your Love for Science: Life of William A. Hammond, American Neurologist. By Bonnie Ellen Blustein. 289 pp, illustrated. New York, Cambridge University Press, Inc, 1991. $54.95 (hardcover)

Curing Their Ills: Colonial Power and African Illness. By Megan Vaughan. Stanford, CA, Stanford University Press, 1991. $37.50 (softcover)

MISCELLANEOUS

Shared Legacy By Alfred P. Ingegno, MD. 291 pp, illustrated. New York, BPI Publishers, 1992. $14.00 (softcover)

Physicians Guide to Rare Diseases. Edited by Jess G. Thoene, MD. 1,224 pp, illustrated. Montvale, NJ, Dowden Publishing Company, 1992. $69.50 (hardcover)

Induced Effects of Genotoxic Agents in Eukaryotic Cells. Edited by Toby Rossman. 167 pp. illustrated. Washington, DC, Hemisphere Publishing Co., 1992. $59.50 (hardcover)

The Prostate Book: Sound Advice on Symptoms and Treatment. By Stephen Rous, MD. 256 pages, illustrated. New York, Norton & Co., 1992. $22.95 (hardcover)

A Handbook For The Traveling Free-Lance Physician. By Gary L. Monroe, MD. 153 pp. Houston, TX, Magellan Publishing Co., 1992. $75.00

A Renaissance Man At Large: Hearts Around The World. By Nathaniel E. Reich, MD. 235 pp, illustrated. New York, Rivercross Publishing, Inc., 1991. $18.95 (hardcover)

Diabetes Type II: Living A Long Healthy Life Through Blood Sugar Naturalization. By Richard K. Bernstein, MD. 359 pp, illustrated. New York, Prentice Hall Press, 1990. $21.95 (hardcover)

Homelessness: A Prevention-Oriented Approach. Edited by Rene I Jahiel, MD, PHD. 409 pp. Baltimore, MD, The Johns Hopkins University Press, 1992. $60.00 (hardcover)

International Pharmaceutical Services: The Drug Industry and Pharmacy Practice in Twenty-Three Major Countries of the World. Edited by Richard N. Spivey, PHD, Albert I. Wertheimer, PHD, T. Donald Rucker, PHD. 629 pp. Binghamton, NY, Pharmaceutical Products Press, 1992. $89.95 (hardcover)

Parents Whose Parents Were Divorced. By Thomas Berner, MA, 148 pp. Binghamton, NY. The Haworth Press, 1992. $14.95 (softcover)

NEUROLOGY

The Dementias: Crossroads Between Neurology And Psychiatry. By Makram Girgis, MD, PHD. 187 pp, illustrated. St. Louis, MO, Warren H. Green, Inc, 1992. $27.50 (softcover)

Neurology For The Psychiatry Specialty Board Review. By Leon A. Weisberg, MD. 144 pp. New York, Brunner/Mazel Publishers, 1992. $19.95 (softcover)

ONCOLOGY

Clinical Oncology For Students Of Radiation Therapy. By John A. Stryker, MD. 307 pp. St. Louis, MO, Warren H. Green, Inc, 1992. $42.50 (softcover)

PEDIATRICS

Disorders of Learning In Childhood. By Archie A. Silver, MD, and Rosa A. Hagin, PHD. New York, John Wiley & Sons, Inc., 1990. $65.00 (hardcover)

Hearing Loss in Childhood. Report of the 102nd Ross Conference on Pediatric Research. 180 pp. Columbus, OH, Ross Laboratories, Inc., 1992. $5.00 (softcover)

PSYCHIATRY

Anxiety: Recent Developments in Cognitive, Psychophysiological, and Health Research. Edited by Donald G. Forgays, Tytus Sosnowski, and Kazimierz. 282 pp. Philadelphia, PA, Hemisphere Publishing Co., 1992. $59.50 (hardcover)

Family Health Psychology Edited T. John Akamatsu, Mary Ann Harris Stephens, Steven Hobfoll, and Janis H. Crowther. 254 pp. Washington, DC, Hemisphere Publishing Co., 1992. $59.50 (hardcover)

Integrating Individual and Family Therapy By Larry Feldman, MD. 224 pp. New York, Brunner/Mazel Publishers, 1992. $25.95 (hardcover)

New Biological Vistas on Schizophrenia, Edited by Jean-Pierre Lindenmayer, MD, and Stanley R. Kay, PHD. 314 pp. New York, Brunner/Mazel Publishers, 1992. $41.95 (hardcover)

Personality, Elevated Blood Pressure, and Essential Hypertension. Edited by Ernest H. Johnson, W. Doyle Gentry, and Stevo Julius. 344 pp. Philadelphia, PA, Hemisphere Publishing Co., 1992. $59.00 (hardcover)

Psychiatric Rehabilitation: A Handbook for Practitioners. By Lynda J. Katz, PHD. 400 pp. St. Louis, MO, Warren H. Green Publishing, 1992. $55.00 (softcover)

Psychotherapy in Independent Practice. Edited by Robert D. Weitz, PHD. 205 pp, illustrated. Binghamton, NY, The Haworth Press, 1992. $24.95 (hardcover)

Self Efficacy: Thought Control of Action. Edited by Ralf Schwarzer. 410 pp. New York, Hemisphere Publishing Corp., 1992. $39.50 (hardcover)

Stress, Distress, and Illness. By Ian Hislop. 198 pp. Sydney, McGraw-Hill Publishing Company, 1991. $30.00 (softcover)

The Chemically Dependent: Phases of Treatment and Recovery. Edited by Barbara C. Wallace, PHD. 364 pp. New York, Brunner/Mazel Publishing, 1992. $45.00 (hardcover)

Women's Madness: Misogyny or Mental Illness? By Jane M. Ussher. 352 pp. Amherst, MA, The University of Massachusetts Press, 1992. $47.50 (hardcover), $16.95 (softcover)

PUBLIC HEALTH AND PREVENTIVE MEDICINE

Violence in America: A Public Health Approach. Edited by Mark L. Rosenberg and Mary Ann Fenley. 199 pp. New York, Oxford University Press, Inc, 1991. $32.95 (hardcover)

MEDICAL EDUCATION

Medical Education in the Age of Improvement. By Lisa Rosner. 273 pp. Edinburgh, England, Edinburgh University Press, 1992. $50.00 (hardcover)

Erwin Alan Appel, MD, Poughkeepsie. Died July 17, 1992; age 62. Dr Appel was a 1956 graduate of Yale University School of Medicine. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Physicians. His memberships included the Dutchess County Medical Society and the Medical Society of the State of New York.

John Heckman Burke, MD, Elmira. Died July 11, 1992; age 82. Dr Burke was a 1935 graduate of the Cornell University Medical College. His memberships included the Chemling County Medical Society and the Medical Society of the State of New York.

Samuel Joseph Castilone, MD, Olean. Died June 30, 1992; age 89. Dr Castilone was a 1927 graduate of the University of Buffalo School of Medicine. His memberships included the Cattaraugus County Medical Society and the Medical Society of the State of New York.

Robert O. Christman, MD, St Augustine, FL. Died December 16, 1991; age 72. Dr Christman was a 1947 graduate of the Chicago Medical School. He was a Diplomate of the American Board of Nuclear Medicine and the American Board of Radiology. His memberships included the Academy of Medicine, the New York Roentgen Society, the Pan American Medical Association, the Westchester County Medical Society, and the Medical Society of the State of New York.

Paul Vincent Downey, MD, Kenmore. Died July 20, 1992; age 80. Dr Downey was a 1936 graduate of the University of Buffalo School of Medicine. He was a Diplomate of the American Board of Radiology. His memberships included the Radiological Society of New York State, the Radiological Society of North America, the Erie County Medical Society, and the Medical Society of the State of New York.

Robert James Keenan, MD, Bronxville. Died June 17, 1992; age 87. Dr Keenan was a 1929 graduate of the Creighton University School of Medicine, Omaha. His memberships included the Bronx County Medical Society and the Medical Society of the State of New York.

Frederick Kleinberg, MD, Fort Lauderdale, FL. Died July 30, 1992; age 84. Dr Kleinberg was a 1933 graduate of the Medizinische Fakultat Der Universitat, Wien. His memberships included the Kings County Medical Society and the Medical Society of the State of New York.

Leo C. Maitland, MD, New York, NY. Died January 31, 1992; age 64. Dr Maitland was a 1953 graduate of the Meharry Medical College, Nashville. He was a Diplomate of the American Board of Surgery. His memberships included the New York County Medical Society and the Medical Society of the State of New York.

Jack Sarnoff, MD, Sarasota, FL. Died July 25, 1992; age 89. Dr Sarnoff was a 1925 graduate of the University of Maryland School of Medicine, Baltimore. He was a Diplomate of the American Board of Otolaryngology. He was a Fellow of the International College of Surgeons. His memberships included the American Academy of Otolaryngology, the Queens County Medical Society, and the Medical Society of the State of New York.

Oscar Schlesigner, MD, New Port Richey, FL. Died May 16, 1992; age 86. Dr Schlesigner was a 1929 graduate of the Universita Komenskeho Fakulta Lekarska Bratislave. His memberships included the American Academy of Family Practice, the Queens County Medical Society, and the Medical Society of the State of New York.

Morris Sonberg, MD, New York, NY. Died January 9, 1992; age 90. Dr Sonberg was a 1928 graduate of the George Washington School of Medicine. His memberships included the Bronx County Medical Society and the Medical Society of the State of New York.

Paul Tartell, MD, Elmhurst. Died July 14, 1992; age 65. Dr Tartell was a 1952 graduate of New York Medical College. He was a Diplomate of the American Board of Radiology. He was a Fellow of the American College of Radiology. His memberships included the Society of Nuclear Medicine, the Radiological Society of North America, the Queens County Medical Society, and the Medical Society of the State of New York.

Milton B. Handelsman, MD
1906–1992

Milton B. Handelsman, MD, former Chief of the Division of Endocrinology and Metabolic Diseases and former Chief of the Diabetes Clinic at the Long Island College Hospital, passed away on September 9, 1992. Born in Bonfeld, Germany, Dr Handelsman received his AB degree from Amherst College in 1926 and his MD degree from Johns Hopkins University Medical School in 1930. He did two years of internship at the Vanderbilt University Hospital in Nashville, Tennessee, and at the Boston Dispensary (Tufts University). In 1932–1933, he spent a fellowship year at Wurzberg University in Germany.

Dr Handelsman entered private practice in Brooklyn, NY, in 1934 when he became a clinical assistant in the Department of Medicine at the Long Island College Hospital. His practice and teaching activities were interrupted by World War II. Between 1943 and 1946, he served as a Lieutenant Colonel with the 37th General Hospital. In 1947, he was promoted to the rank of Associate Attending Physician and in 1954 to that of Attending Physician. He became Chief of the hospital's Diabetes Clinic in 1936 and Chief of the Division of Endocrinology and Metabolic Diseases in 1960, positions he held until his retirement in 1982. In 1979, the Diabetes Clinic was renamed the Milton B. Handelsman Diabetes Clinic, in his honor. He introduced many innovative features to the clinic. Among them was the assignment of two pre-medical students each year from St. Francis College, all of whom eventually went on to medical school. He was also highly successful in securing voluntary contributions to the clinic. Under his direction, the Division of Endocrinology and Metabolic Diseases established a fellowship program which over the years has trained numerous endocrinologists.

In addition to his hospital appointments, Dr Handelsman served on the faculty of the Department of Medicine of the State University of New York Health Science Center at Brooklyn, College of Medicine, rising to the rank of Clinical Professor in 1967. In this capacity, he taught both students and house officers not only at the Long Island College Hospital, but also at Kings County Hospital Center and at the Brooklyn Veterans Administration Hospital. On his retirement in 1982, Dr Handelsman became Clinical Professor Emeritus.

Dr Handelsman was not only widely respected as an outstanding teacher and compassionate clinician, but also as a researcher who made important contributions toward a better understanding of diabetes mellitus. His published papers appeared in numerous peer-reviewed medical journals including the *American Journal of Surgery*, the *American Journal of Digestive Diseases*, the *American Journal of Medical Sciences*, the *Annals of Internal Medicine*, the *Archives of Internal Medicine*, the *New York State Journal of Medicine*, and many others. He had a special interest in small vessel disease in diabetes mellitus and published the results of his research on this topic in several well-known papers.

In 1970, Dr Handelsman headed a research team in Mali, West Africa. There he and his colleagues surveyed three different population groups for the prevalence of diabetes mellitus. On arrival in Mali, he and his wife, Sophie, discovered that most of their baggage had through error been sent to Kenya in East Africa. However, he had hand carried the Accustat apparatus, so vital to his survey. Wearing borrowed clothes, he and his wife set off into the bush where they spent several weeks. He solved the problem of having to use chilled water with the Accustat apparatus when he discovered that water drawn up from wells 200-feet and more deep provided the required cold temperature. The results of this field work were later published in the *Transactions of the Royal Society of Tropical Medicine and Hygiene* (1976).

Dr Handelsman was a member of many scientific and professional organizations including the American Diabetes Association, the New York Diabetes Association, the Federation for Clinical Research, the American Medical Association, the Medical Society of the State of New York, and the Medical Society of the County of Kings. He became a diplomate of the American Board of Internal Medicine in 1941 and a Fellow of the American College of Physicians in 1966. He served as the President of the New York Diabetes Association in 1957–1958.

Dr Handelsman was a great help to the editors of the *Journal* over the years as a peer reviewer and in providing valuable counsel and advice. He brought to this effort the same scholarly and intellectual talents that characterized him as a teacher. In 1979, John N. Edson, MD, then the Director of the Department of Internal Medicine at the Long Island College Hospital made the following comment about Dr Handelsman as a teacher: "You have inspired many young doctors in their quest for medical knowledge with your enthusiastic and erudite discourses." Dr Handelsman brought the same enthusiasm and erudition to the peer review process. In addition, he was a gentle, kind, and thoughtful physician and teacher who encouraged and helped young physician authors.

Dr Handelsman was married in 1928 to Lola Kohn. She passed away in 1967. He is survived by his wife Sophie, his daughter, Marilyn Saypol of Teaneck, NJ, his son, Harold Handelsman of Chicago, IL, his stepson Mark Lubroth of Huntington, NY, and and by several grandchildren and great-grandchildren. A memorial service was held for him at noon on September 11 at The Riverside Chapel in Manhattan.

Pascal James Imperato, MD
Editor

Guidelines for authors

ORIGINALITY

The *New York State Journal of Medicine* welcomes research papers and original essays on the practice of medicine, medical education, public health, the history of medicine, medicolegal matters, legislation, ethics, the mass media, and socioeconomic issues in health care.

Manuscripts should be prepared according to the "Uniform requirements for manuscripts submitted to biomedical journals" (*NY State J Med* 1992; 92:122–125). The requirements were established by the International Committee of Medical Journal Editors, of which the *Journal* is a participating member. These Guidelines are intended to highlight aspects of the *Journal*'s particular style of publication.

A manuscript will be considered for publication if it is original, has not been published previously in whole or in part in either a medical journal or a lay publication, and is not simultaneously under consideration elsewhere. A copy of any possibly duplicative material, such as a reference "in press," should be submitted along with the manuscript.

PREPARATION

An original typewritten or "word-processed" manuscript and two photocopies (to facilitate outside review) are required. If the manuscript is not accepted for publication, the original and one copy will be returned. All figures must be submitted in triplicate. The manuscript should be double-spaced throughout, including references, tables, legends, quotations, and acknowledgments. A separate title page should include the full title of the paper in upper and lower case type, the names of the authors exactly as they should appear in print (including their highest academic degree), and the names of all providers of funding for research on which the paper is based. Information on the amount and allocation of funding is optional.

A corresponding author should be designated in the covering letter. Authors should list their title and affiliation at the time they did the work, and, if different, their present affiliation. The addresses and telephone numbers of all authors should be supplied for editorial purposes. All authors of a manuscript are responsible for having read and approved it for submission.

CATEGORIES

Research Papers should be limited to 3,000 words and should include the following sections: Introduction, Methods, Results, Discussion, and References. Multiple subheadings and numbered lists are discouraged. An abstract limited to 150 words should state the reasons for the study, the main findings, and their implications. Statistical evaluations should be described in the Methods section, and the name and affiliation of the statistician should be included in the acknowledgments if this individual is not listed as a coauthor. Reports of experiments involving human subjects must include a description in the Methods section of the informed consent obtained and a statement that the procedures followed were approved by an institutional research review committee. Anonymity of patients must be preserved. Reports of experiments on animals must note which guidelines were followed for the care and use of laboratory animals.

Case Reports should be limited to 1,250 words. Review Articles should not exceed 3,000 words. Commentaries should be between 1,000 and 1,500 words. Other kinds of original essays should not exceed 2,500 words. A letter of inquiry should be sent to the editor prior to submitting a Review Article or Commentary.

For Research Papers only, scientific measurements should be given in conventional units, with Système Internationale (SI) units in parentheses. Abbreviations and acronyms should be kept to a minimum, and jargon should be avoided. Generic names of drugs should be used instead of brand names.

FIGURES

The submission of color illustrations or slides is discouraged. Only black and white glossy photographic prints or "camera-ready" artwork will be accepted. The *Journal* is unable to provide art services such as the addition of arrows to photographs. A signed consent for publication must accompany photographs in which the patient is identifiable. Illustrations from other publications generally will not be considered. Legends for illustrations should be typed on a separate sheet, and for photomicrographs should include magnification and stain. Each illustration should be lightly marked on the back in pencil with the name of the first author, the number as cited in the text, and an arrow indicating the top. Each set of illustrations should be submitted unmounted in a separate envelope.

Tables should be simple, self-explanatory, and few in number. Each table should be typed double-spaced on a separate sheet.

REFERENCES

References should be limited to the most pertinent. The recommended maximum number of references is 25 for Research Papers and most other original contributions (except lengthier Review Articles), 12 for Commentaries and Case Reports, and six for letters to the editor.

Authors are responsible for the accurate citation of references. Citation of secondary sources is discouraged except where the original reference is unobtainable. Authors may not cite references they have not read, and the use of abstracts as references should be avoided.

References should be indicated in the text by superscript numbers following the name of the author (eg, Smith[2] reported two cases).

The list of references at the end of the article should be typed double-spaced and references should be numbered in the order in which they appear in the text. When there are three or fewer authors, all should be listed; where there are four or more, the first three should be listed, followed by *et al.* Names of journals should be abbreviated according to *Index Medicus* and underlined.

Samples references:

1. Bowen J: Autoimmune paraneoplastic diseases of the nervous system. *NY State J Med* 1991; 91:2–3.
2. Cotran RS, Kumar V, Robbins SL: *Robbins Pathologic Basis of Disease*, ed 4. Philadelphia, WB Saunders Co, 1989, pp 842–845.

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All manuscripts are reviewed by the editors, and most manuscripts are sent to outside referees. Decisions concerning acceptance, revision, or rejection of a manuscript are usually made within three to six weeks. Every effort will be made to assure prompt publication of an accepted manuscript. A galley proof will be sent to the author for approval prior to publication.

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(*continued from p 9A*)

ology, 9111 Old Georgetown Rd, Bethesda, MD 20814-1699. Tel: (800) 257-4739.

Dec 14–17. **Methods of Investigation on the Dead Sea Scrolls and the Khirbet Qumran Site: Present Realities and Future Prospects.** The New York Blood Center. Contact: Conference Department, New York Academy of Sciences, 2 E 63rd St, New York, NY 10021. Tel: (212) 838-0230.

NEW HYDE PARK

Dec 9. **Multidisciplinary Approach to the Fetus and Neonate with Congenital Anomalies.** 6.5 Cat I Credits. Long Island Campus for the Albert Einstein College of Medicine. Contact: June Lewis, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

SYRACUSE

Dec 4–5. **43rd Annual Post-Graduate Review in Ophthalmology.** Hotels at Syracuse Square. Contact: John A. Hoepner, MD, Chairman, Department of Ophthalmology, SUNY Health Science Center at Syracuse, Syracuse, NY 13210.

AROUND THE NATION

CALIFORNIA

Dec 9–13. **American Back Society 10th Anniversary Meeting: The Industrial Back.** Hyatt Regency, San Francisco. Contact: American Back Society, 2647 E 14th St, Suite 401, Oakland, CA 94601. Tel: (510) 536-9929.

Dec 10–12. **Ophthalmic Surgical Complications: Oops, What Do I Do Now?** 12 Cat I Credits. *Dec 10–13.* **Ophthalmic Surgical Complications.** Ritz Carlton Hotel, San Francisco. Contact: Extended Programs in Medical Education, Room LS-105, University of California, San Francisco, CA 94143. Tel: (415) 476-4251.

COLORADO

Dec 28–Jan 1. **Holiday Imaging Update.** 23.75 Cat I Credits. The Ritz-Carlton, Aspen. Contact: Dawne Ryals, Ryals and Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

HAWAII

Dec 28–Jan 2. **Year End Review of Imaging and Intervention.** 25 Cat I

(*continued on p 13A*)

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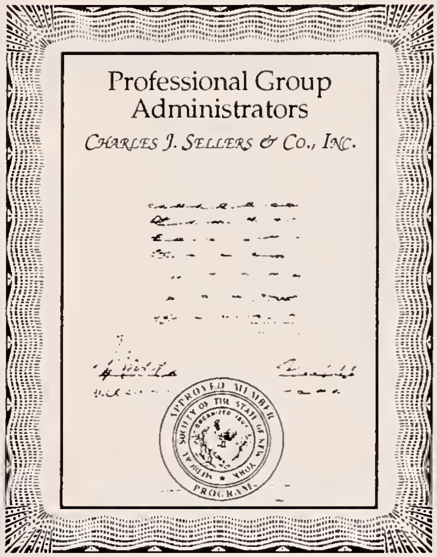

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MARYLAND

Dec 13–15. **Open Conference on Unit-of-Use Packaging: Contemporary Issues.** Hyatt Regency, Baltimore. Contact: Joseph G. Valentino, JD, Associate Director, US Pharmacopeia, 12601 Twinbrook Parkway, Rockville, MD 20852. Tel: (301) 816-8256.

MISSOURI

Dec 5. **Hypertension.** The Ritz-Carlton, St Louis. *Dec 12.* **Management of Hypercholesterolemia: Goals and Strategies.** 4.5 Cat I Credits. Adam's Mark Hotel, St Louis. Contact: Washington University School of Medicine, CME, Campus Box 8063, 660 South Euclid Ave, St Louis, MO 63110-1093. Tel: (800) 325-9862.

PENNSYLVANIA

Dec 2. **Osteoarthritis.** *Dec 3.* **Rheumatoid Arthritis.** *Dec 7.* **Overview of Infections and Anti-infective Therapy.** *Dec 8.* **Major Bacterial Infections.** *Dec 9.* **Antibiotic Therapy I.** *Dec 10.* **Antibiotic Therapy II.** *Dec 11.* **Antibiotic Therapy III.** College of Physicians, Philadelphia. Contact: Laura Anderson, Pharma Design Inc, 30 Technology Drive, Warren, NJ 07059. Tel: (908) 561-4804.

TENNESSEE

Dec 3–7. **Twentieth Assembly Meeting: Hospital Medical Staff Section, AMA.** Opryland Hotel, Nashville. Contact: AMA-HMSS. Tel: (312) 464-4754.

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Dec 2. **The OSHA Bloodborne Pathogen Standard: How to Comply.** 5 Cat I Credits. Loews Anatole, Dallas. Contact: Rod Betts, American Association for Clinical Chemistry, 2029 K St, NW, Washington, DC 20006.

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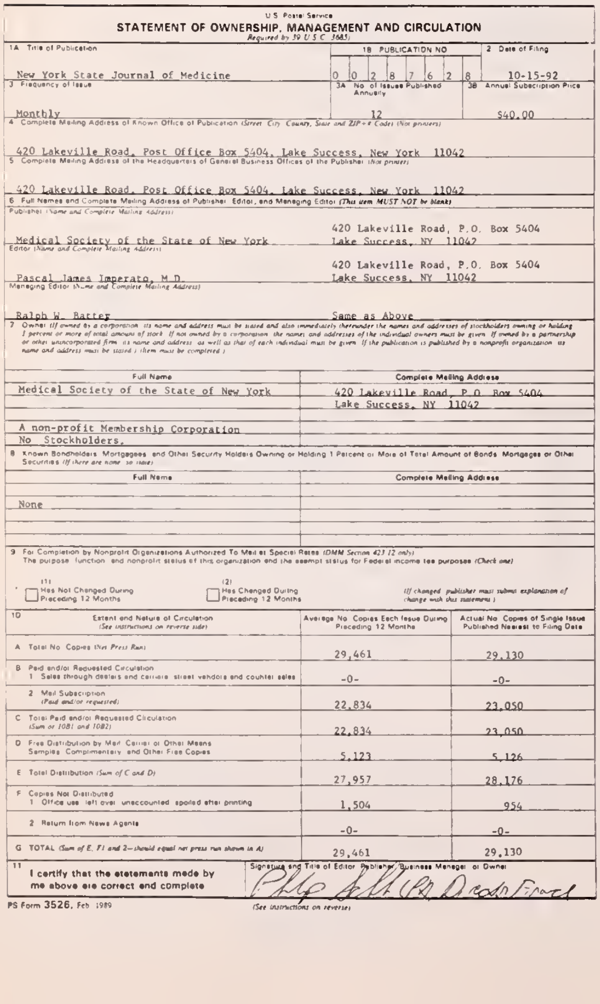

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3 Frequency of Issue: Monthly
3A No of Issues Published Annually: 12
3B Annual Subscription Price: $40.00
4 Complete Mailing Address of Known Office of Publication *(Street City County, State and ZIP+4 Code) (Not printers)*: 420 Lakeville Road, Post Office Box 5404, Lake Success, New York 11042
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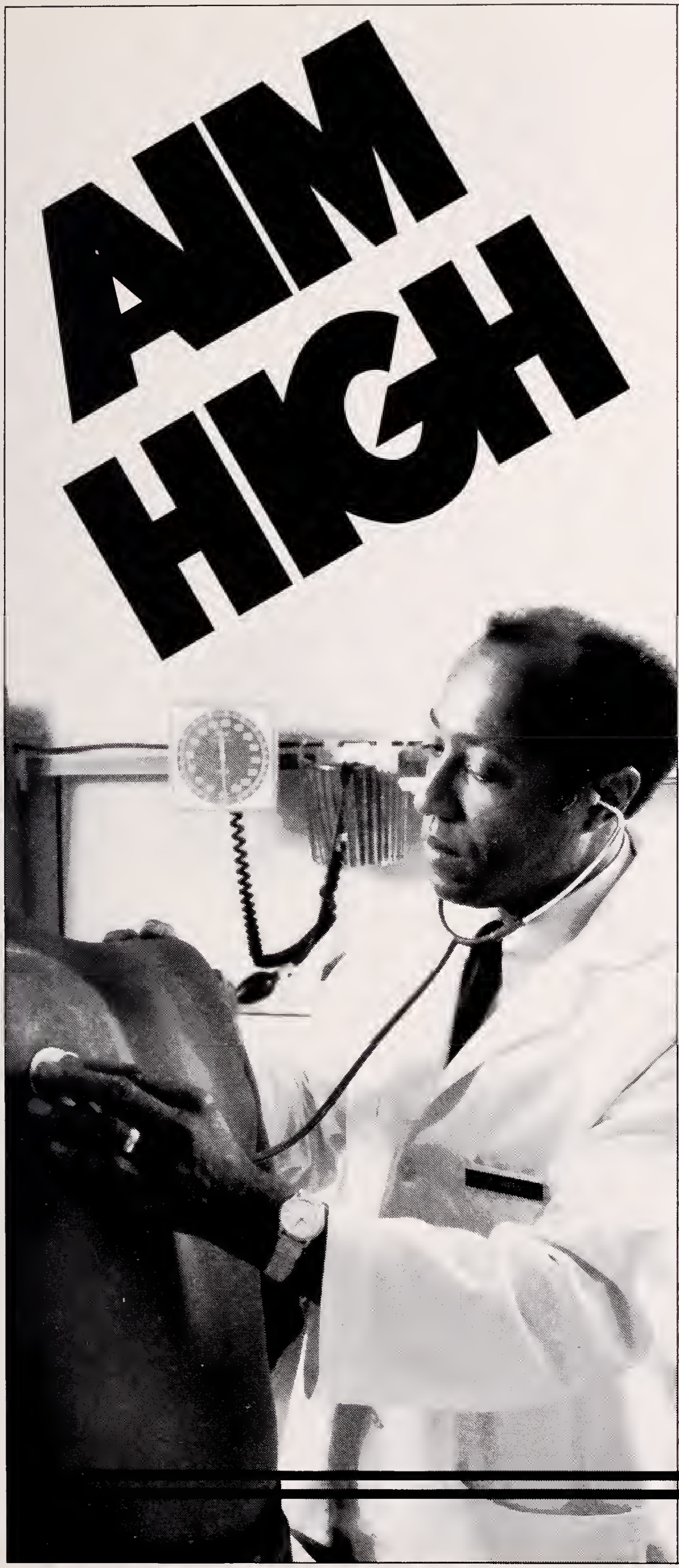

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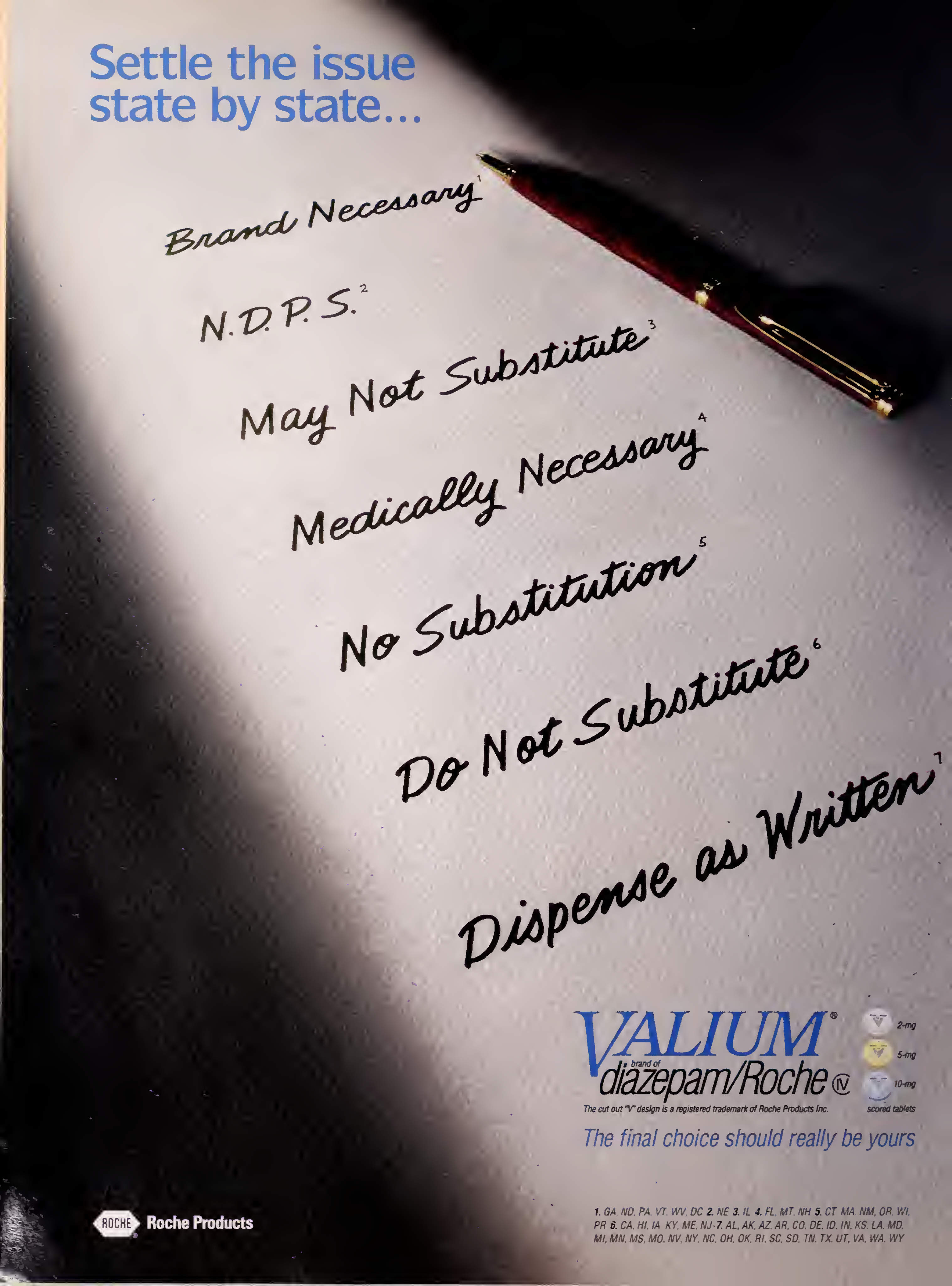

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New York State Journal of Medicine

MEDICAL SOCIETY OF THE STATE OF NEW YORK ORGANIZED 1807

VOL 92
NO 12
DECEMBER
1992

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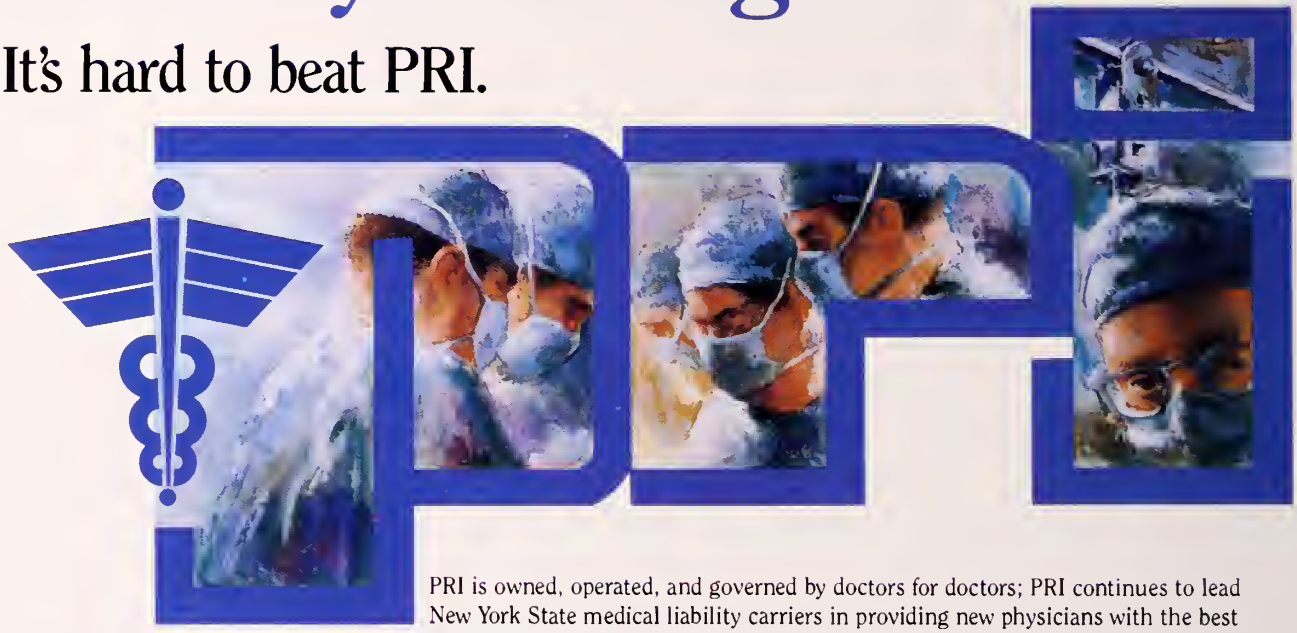

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The address for EDITORIAL, CIRCULATION, and ADVERTISING OFFICES is PO Box 5404, Lake Success, New York 11042. Telephone: (516) 488-6100.

POSTAGE. Second class postage is paid at New Hyde Park, New York, and additional mailing office. POSTMASTER: Send POD Form 3579 to New York State Journal of Medicine, PO Box 5404, Lake Success, New York 11042.

New York State Journal of Medicine

DECEMBER 1992 / Volume 92, Number 12

Contents

COMMENTARIES

513 **Triple jeopardy: The health hazards of unraveling insurance, uncontrolled illness, and economic adversity**
MELVIN I. KRASNER, PHD

515 **Pregnancy and pulmonary arterio-venous fistula**
ANDREW J. SWINBURNE, MD

516 **Mastectomy: Is it necessary for occult breast cancer?**
M. MARGARET KEMENY, MD

RESEARCH PAPERS

518 **Complications of laparoscopic cholecystectomy in a geriatric population group**
RAPHAEL P. NENNER, MD; PASCAL JAMES IMPERATO, MD; CHARLES M. ALCORN, SCD

521 **Economic interventions to discourage the illegal sale of cigarettes to minors in New York State**
K. MICHAEL CUMMINGS, PHD; TERRY PECHACEK, PHD; EVA SCIANDRA

525 **Low level and bystander exposure to lead among factory workers**
ALF FISCHBEIN, MD; CARLA CAMPBELL, MD; JIIN-CHYUAN J. LUO, MD

HISTORY

529 **A history of Jewish attitudes toward nursing**
LEON SOKOLOFF, MD

537 **Bernard John Dowling Irwin, MD—Physician, naturalist, and military hero**
ALLEN D. SPIEGEL, PHD, MPH

SPECIAL ARTICLES

545 **Judicial ruling on a physician violation of New York State law protecting the confidentiality of HIV-related information**
DONALD R. MOY, JD, GENERAL COUNSEL

547 **W. Somerset Maugham, Capgras syndrome, and aging**
JEROME M. SCHNECK, MD

(continued on p 4A)

MEETINGS AND LECTURES

The New York State Journal of Medicine *cannot guarantee publication of meeting and lecture notices. Information must be submitted at least three months prior to the event.*

JANUARY 1993

AROUND THE STATE

EAST ELMHURST

Jan 20/June 9. **14th Annual Internal Medicine Board Review Course.** Wednesdays. LaGuardia Marriott, East Elmhurst. Contact: June Lewis, Office of Continuing Medical Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042. Tel: (718) 470-8650.

HUNTINGTON

Jan 5. **Abdominal Masses and Mild Gut Malformation.** *Jan 12.* **Respiratory Infections—Bronchiolitis.** *Jan 19.* **Croup and Pneumonia.** *Jan 26.* **Evaluation of Metabolic Disorders.** Cat I Credits. Schneider Children's Hospital Grand Rounds at Huntington Hospital. Tuesdays, 8:30 AM to 9:30 AM. Contact: Arthur J. Schwager, MD, 164 E Main St, Huntington, NY 11743. Tel: (516) 427-0530.

MANHATTAN

Jan 20. **Advances in Management of Burns.** Cornell University Medical College, Uris Auditorium. Contact: Association of Practicing Physicians, Update Your Medicine, 445 E 69th Street, Olin 328, New York, NY 10021. Tel: (212) 746-4752.

AROUND THE NATION

ARIZONA

Jan 29–30. **Objective Methods for Assessment of Upper Airway Function.** Tucson. Contact: Office of Continuing Medical Education, The University of Arizona Health Sciences Center, Tucson, AZ 85724. Tel: (602) 626-7832.

CALIFORNIA

Jan 10–17. **Caribbean Cruise Seminars: Neuroimaging/MRI at Sea.** *Jan 17–24.* **Breast Imaging at Sea.** *Jan 24–31.* **Ultrasound at Sea.** Contact: Medical Seminars International, 18981 Ventura Blvd, Suite 303, Tarzana, CA 91356. Tel: (818) 774-9077.

(continued on p5A)

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References:

1. A. Morales et al., New England Journal of Medicine: 1221. November 12, 1981.
2. Goodman, Gilman — The Pharmacological basis of Therapeutics 6th ed., p. 176-188. McMillan December Rev. 1/85.
3. Weekly Urological Clinical letter, 27:2, July 4, 1983.
4. A. Morales et al., The Journal of Urology 128: 45-47, 1982.

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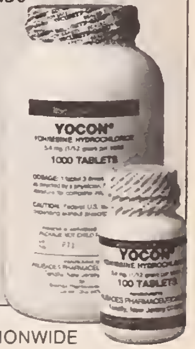

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New York State
Journal of Medicine

CONTENTS, CONT'D

CASE REPORTS

551 **Osler-Weber-Rendu disease—Diagnosis and management of spontaneous hemothorax during pregnancy**
FREDERICK A. BEVELAQUA, MD; STEVEN A. ORDORICA, MD; RICHARD LEFLEUR, MD; BRUCE YOUNG, MD

552 **Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids**
PERRY C. GOULD, MD; HIMANSHU P. DESAI, MD; ROBERT S. BARTOLOMEO, MD; GARY J. SCHWARTZ, MD; LOUIS SWARTZ, MD

553 **Myxedema coma**
JOHN M. DUNN, MD, JOSEPH HUGHES, MD

555 **Occult breast cancer—A case report and review of the literature**
LUCIO FORTUNATO, MD; JOSEPH J. SORRENTO, MD; ROBERT A. GOLUB, MD; ROBERTO CANTU, MD

LETTERS TO THE EDITOR

558 **In support of the *New York State Journal of Medicine***
JOHN S. DAVIS, MD

558 **A plea for keeping the *New York State Journal of Medicine* alive**
CHARLES C. CANVER, MD

558 **Memories of an internship**
DORIS H. MILMAN, MD, LAWRENCE SPIELBERGER, MD

559 **Waiting for the doctor**
ARMANDO E. GIULIANO, MD

559 **History: The great reminder**
KENNETH W. WRIGHT, MD, JOHN N. EDSON, MD

ESSAY REVIEW

561 **William Alexander Hammond, MD—A 19th-century physician-scientist and entrepreneur**
PASCAL JAMES IMPERATO, MD

564 BOOK REVIEWS

568 OBITUARY LISTINGS

570 MEMORIAL—In Memoriam

571 INDEX

3A MEDICAL MEETINGS AND LECTURES

(continued from p3A)

Jan 16–18. **Tenth Annual Mohs Micrographic Surgery Conference of the Skin Care Foundation.** Marriott Rancho Las Palmas Resort, Rancho Mirage, CA. Contact: Mitzi Moulds, The Skin Care Foundation, 245 Fifth Ave, Suite 2402, New York, NY 10016. Tel: (212) 725-5176.

Jan 16–17. **Interventional Radiology Update.** 7.5 Cat I Credits. *Jan 18–20.* **Neuroradiology Update.** 17.5 Cat I Credits. *Jan 21–24.* **Breast Imaging Update.** 17.25 Cat I Credits. Ritz-Carlton Hotel, Laguna Nigeul. Contact: Dawne Ryals, Ryals and Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

Jan 16–23. **38th Tutorial on Clinical Cytology: A Review Course in Cytopathology.** 80 Cat I Credits. Sheraton Universal Hotel, Universal City. Contact: Tutorials of Cytology, 1640 E 50th St, Suite 20B, Chicago, IL 60615-3161. Tel: (312) 947-0098.

FLORIDA

Jan 4–8. **Muscoleskeletal MR Course.** 25 Cat I Credits. *Jan 9–15.* **Annual Meeting and Course: Imaging and Intervention of the Genitourinary Tract: The Cutting Edge.** 29 Cat I Credits. *Jan 25–28.* **The Third Annual Breast Imaging Course.** 18 Cat I Credits. The Ritz-Carlton, Naples. Contact: Dawne Ryals, Ryals and Associates, PO Box 1925, Roswell, GA 30077-1925. Tel: (404) 641-9773.

Jan 14–15. **Quality Management for the Medial and Dental Professional.** Winter Park. Contact: Philip Crosby Associates. Tel: (800) 722-1464.

Jan 25–28. **Fifth Annual International Symposium on Vascular Diagnosis and Intervention.** Fontainebleau Hilton, Miami Beach. Contact: Education Services for the Miami Vascular Institute. Tel: (305) 598-5992.

Jan 25–27. **Pioneering Pediatrics.** 11 Cat I Credits. West Palm Beach. Contact: Laura J. Keech, CME Coordinator, Good Samaritan Medical Center, PO Box 3166, West Palm Beach, FL 33402-3166. Tel: (407) 650-6236.

NEVADA

Jan 17–19. **Ambulatory Obstetrics and Gynecology.** 14 Cat I Credits. Hyatt Regency, Lake Tahoe. Contact: UCD-MC, Office of Continuing Medical Education, 2701 Stockton Blvd, Sacramento, CA 95817. Tel: (716) 734-5390. ☐

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New York State
Journal of Medicine

COMMENTARIES

Triple jeopardy: The health hazards of unraveling insurance, uncontrolled illness, and economic adversity

Although New Yorkers have been struggling with a myriad of health problems for many decades, it is difficult to recall a recent year when the health of our community and the viability of its health services system seemed so seriously threatened on so many different fronts. Not only are we overwhelmed with the health consequences of widespread social morbidity and the devastating impact of the acquired immunodeficiency syndrome (AIDS), but we are now facing an outbreak of tuberculosis, including the emergence of drug-resistant strains, which places many segments of our community at risk because of its relatively simple modes of transmission and the lack of effective infection control measures. Similarly, in spite of continued growth in governmental allocations for health care over the past several years, the resources available to health care providers have barely kept up with the increasing costs of labor, capital, and technology, let alone with the increasing community demands fueled by the aging of the population, rising expectations, and expanding biomedical virtuosity. On top of this worrisome trend, the last round of budget cuts on both the state and the city levels leaves virtually no facility unaffected and no potential user unconcerned. In addition, although we have unfortunately become all too familiar with the gaps and deficiencies in our health insurance mechanisms, only recently have these deficiencies engulfed even the relatively affluent, creating the perception of a health insurance system that is rapidly unraveling.

The last development is especially disturbing; unlike the sudden emergence of a deadly epidemic or the overwhelming consequences of social disintegration and economic recession, the mechanism we use to finance health care and provide coverage is something over which health policymakers presumably have some degree of control. For many decades, our system of employment-related medical insurance—supplemented for the past 25 years by Medicaid for the poor and Medicare for the elderly and disabled—provided a reasonable level of protection in spite of its many flaws. However, the past decade has witnessed a steady and relatively rapid and widespread erosion in the confidence we placed in health insurance.

The most obvious and earliest recognized failing of the system is the lack of coverage for a substantial segment of our population. Although initially directed at limitations of publicly funded programs, particularly Medicaid, dissatisfaction with the situation quickly broadened, as it became apparent that a substantial proportion of the uninsured were employed and as the size of this disenfranchised group grew.

What had been perceived as largely a problem of the poor and low-income workers was soon found to be affecting all segments of society. Increasingly, it seems as if health insurance is available only to that segment of the population that is healthy, affluent, and organized into large groups.

State officials in New York and New Jersey have recently criticized the apparent propensity of insurance companies to avoid risk by seeking to enroll only groups with unblemished health experience instead of assuming and pooling health risks among all members of society. Whereas we can sympathize with the plight of a small insurer suffering from the financial consequences of a disproportionate share of extremely expensive cases, as a society we cannot tolerate a system that denies coverage to those in greatest need.

Even more troubling, however, are the problems and uncertainty faced by those already enrolled in insurance plans and who are presumably covered. Examples of these problems include terminating policy holders when they have the misfortune to develop the illnesses for which insurance protection was sought in the first place and unfairly denying payment for necessary health expenditures.

Similarly unacceptable are the hardships imposed on continuously covered individuals forced to switch insurers because of job changes. They are often compelled to choose between having an existing medical condition excluded from coverage or paying for largely duplicative insurance policies. Perhaps the most extreme example of the disintegration of insurance protection is a recent court battle in which the insurance carriers of two employed parents with family coverage denied responsibility for their dependent, each insurer claiming that the other was responsible. Clearly, we have ample reason to be dissatisfied with our huge and very expensive insurance apparatus that leaves too many people unprotected for too many different reasons.

The fact that these problems may be the result of our own policy action, or more accurately absence of such action, makes them especially annoying, but it also makes the possibility for solving them that much greater. Indeed, since virtually all of the industrialized world has managed to provide universal and comprehensive health care coverage, while spending far less on health services than we do, we can feel quite confident that the solution to this set of problems is related principally to volition rather than to ability.

Unfortunately, we cannot say the same thing about other serious health care problems we face. In the battle against AIDS, in spite of the allocation of a vast amount of resources and scientific energy, the illness remains as tenacious and mysterious as ever. It seems that the more we study HIV, the more we learn of its complexity, elusiveness, and deadly endurance. Although we seem to have made significant progress in some areas, slowing the spread of the disease in some communities and developing new drugs, no one can reliably project what this progress implies in the long term. One thing, however, is certain: For the foreseeable future, we will be facing the monumental challenge of providing care to those already infected with HIV and of preventing the further spread of this devastating illness.

One unmistakable lesson learned from our painful experience with AIDS is that infection control applied swiftly and conscientiously can be remarkably effective in preventing the spread of deadly disease. Regarding illness for which we have yet to find a cure or for which therapy is particularly difficult, arresting the spread of infection is the most promising option for conquering the disease.

The resurgence of tuberculosis after many years of decline has taken New York's, as well as the nation's, health care system by surprise, and although it is considered a curable disease, it presents several formidable public health challenges. It can be difficult to diagnose, it can be spread through the air, and it may require a complex and extended course of treatment. An increasing proportion of cases involving drug-resistant strains, the potential deadliness of disease, and its link to HIV infection and social morbidity further complicate an already difficult problem.

The need for swift and aggressive action to minimize the spread of this disease cannot be overstated. Years of neglect of biomedical research in this area and a dismantling of the public health infrastructure for controlling tuberculosis have left us with a dearth of scientific knowledge and few effective tools to control this illness. Therefore, we must simultaneously mobilize all existing resources to fight the infection and prevent its spread, and undertake intensive efforts to develop new diagnostic tests, more potent therapeutic agents, and more effective public health techniques.

Fortunately, we are beginning to see significant responses at all three levels of government. The city is forging ahead, with the state's financial backing through the Medicaid program, with the implementation of directly observed treatment for tuberculosis patients suffering from poverty, substance abuse, homelessness, or similar problems. With a course of treatment that extends over at least several months and ordinarily involves a variety of medications, tuberculosis patients have a particular need for conscientious outreach, continuous monitoring, and careful follow-up. The city is also taking steps to centralize clinical information on tuberculosis patients, to begin addressing their extensive needs for a variety of supportive services, and to introduce incentives to encourage compliance with and completion of the treatment regimen.

In addition to supporting local efforts for controlling tuberculosis through directly observed therapy, state government is pursuing efforts to identify and treat tuberculosis patients in high-risk populations, particularly in correctional facilities. The federal government has also significantly increased its allocation for tuberculosis control, but the urgency, magnitude, and complexity of the problem dwarf the amount of resources made available.

The potential consequences of inadequate or delayed response to the tuberculosis threat can be overwhelming in both human and financial terms, underscoring the need to control and contain this illness as quickly as possible. The task is daunting not only because we are still searching for effective tools for diagnosis, therapy, and infection control, but also because we are groping in a scientific area where basic knowledge is woefully incomplete and we are facing an environment where medical issues are enormously complicated by a myriad of social, economic, and legal considerations. Nevertheless, we cannot afford to be paralyzed by these obstacles, for only swift, sustained, and forceful action will be able to prevent untold human suffering and avoid immense social costs.

It is unfortunate that this pressing need for additional resources comes at a time when city, state, and federal governments are all facing serious budgetary difficulties. New York City in particular, in spite of an 11th-hour revelation of some unexpected budgetary bounty amidst an otherwise painfully austere fiscal climate, has been adversely affected by the economic recession, suffering significantly deeper and longer-lasting declines than the rest of the nation.

Although we might be inclined to regard the recession and its immediate consequences as being fundamentally transitory phenomena, it is all too easy to overlook the cumulative impact of repeated fiscal crises on the city's health and the viability of its health facilities and programs. On top of the obvious consequences of periodic service cutbacks and unstable funding, the emergence and recognition of the tuberculosis threat at a particularly inopportune time underscore the dangerous results of a roller-coaster economy and volatile revenue sources. Of particular significance is the impact of this confluence of adverse events on hospitals, which provide care to tuberculosis patients when they are most infectious and also serve a very susceptible, immunocompromised population. The financial implications of this role are genuinely frightening, coming at the same time as drastic payment cuts emanating from the state's budget crisis, even if recent adjustments in payment rates appear to have alleviated hospitals' financial stress. Whether or not current payment rates can be characterized as stringent, they certainly could not have anticipated the additional burden associated with tuberculosis control and management.

These are unquestionably formidable challenges, but they are not beyond our ability to meet them. New York City unfortunately does suffer disproportionately from poverty, social morbidity, and related health problems. It has the dubious distinction of having more cases of AIDS and tuberculosis than any other American city and is burdened by the current economic situation more severely than the rest of the country. However, New York City also possesses unparalleled wealth, creativity, talent, and commitment, in quantities more than adequate to tackle even the most difficult problems. To invert a Big Apple slogan, if these problems cannot be solved here, they cannot be solved anywhere.

Having said all that, however, it is important to point out that local government possesses neither the legal authority, the fiscal power mechanisms, nor the social responsibility to address these problems. Regardless of where one sits in the political spectrum, it is obvious that the fiscal and statutory devices to smooth out the adverse consequences of economic cycles are beyond the local level. Likewise, although one may argue whether Albany or Washington is the appropriate locus of efforts to reform our health insurance apparatus, it is clearly not within the city's purview. It will take bold and forceful action in Albany and Washington to allow the city to demonstrate its mettle in battling its problems.

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Pregnancy and pulmonary arterio-venous fistula

Hereditary epistaxis and telangiectasia were first reported by Babington in 1865 and Legg in 1876.[1,2] The pseudonym, Osler Weber-Rendu disease, evolved after more complete descriptions of this disorder were reported between 1896 and 1907.[3–5] Osler-Weber-Rendu disease is transmitted as an autosomal dominant with an estimated frequency in the population of 1:100,000. Both sexes are affected equally, and there is a wide variability in the type, location, and number of randomly distributed malformations. Approximately 15% of patients with Osler-Weber-Rendu disease have pulmonary arterio-venous (AV) fistula, whereas 60% of patients with pulmonary AV fistula have evidence of Osler-Weber-Rendu disease.[6,7]

Bevelaqua in this issue reports a case of a potentially life-threatening hemothorax, occurring during the sixth month of pregnancy in a patient with Osler-Weber-Rendu disease.[8] This is the sixth reported case of progression of pulmonary AV fistula during pregnancy. Four of the five other reports describe life-threatening events, three hemothoraces, and one case of severe hypoxemia necessitating emergency lung resection.[9,10] Hemothorax complicating pulmonary AV fistula is particularly serious, as five of 16 reported hemothoraces caused by pulmonary AV fistula have led to death. The other case involved dramatic enlargement of multiple pulmonary AV fistula on chest x-ray over the course of the pregnancy.[12] Why pregnancy should cause pulmonary AV fistula to enlarge or rupture is not clear. However, as noted by Bevelaqua, the increased blood volume of pregnancy and the effect of changing estrogen and progesterone concentrations on the aneurysmal wall may explain these changes.[8]

It is interesting to observe that in several series describing pulmonary AV fistula, there is a female preponderance supporting the notion that pregnancy may somehow lead to enlargement of AV malformations located in the lung.[7,13,14] There is a clear lesson to be learned from these case reports. Pregnancy in women with known pulmonary AV fistula must be viewed with caution. The patient should understand that there is a risk of rupture of the fistula or increase in the intrapulmonary shunt as a result of the pregnancy.

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1. Babington BC: Heredity epistaxis. *Lancet* 1865; 2:362.
2. Legg JW: Medical Societies: Royal Medicine and Chirurgical Society: case of haemophilia complicated with multiple naevi. *Lancet* 1876; 2:856.
3. Rendu: Epistaxis repetees: chez un sujet porteur de petits angiomes cutanes et muqueux. *Bull et Mem Soc Med d Hop de Paris* 1896; 13:731–733.
4. Osler W: On family form of recurring epistaxis, associated with multiple telangiectases of skin and mucous membranes. *Bull Johns Hopkins Hosp* 1901; 12:333–337.
5. Weber FP: Multiple hereditary developmental angiomata (telangiectases) of skin and mucous membranes associated with recurring hemorrhages. *Lancet* 1907; 2:160–162.
6. Hodson CH, Burchell HB, Good CA, et al: Hereditary hemorrhagic telangiectasia and pulmonary arteriovenous fistula: survey of a large family. *N Eng J Med* 1959; 261:625.
7. Dines DE, Arms RA, Bernatz PE, et al: Pulmonary arteriovenous fistulas. *Mayo Clin Proc* 1974: 49:460–465.
8. Bevelaqua FA, Ordorica SA, Lefleur R, et al: Osler-Weber-Rendu disease: diagnosis and management of spontaneous hemothorax during pregnancy. *NY State J Med* 1992; 92:551–552.
9. Swinburne AJ, Fedullo AJ, Gangemi R, et al: Hereditary telangiectasia and multiple pulmonary arteriovenous fistulas: clinical deterioration during pregnancy. *Chest* 1986; 89:459–460.

10. Gammon AM: Osler-Weber-Rendu disease and pulmonary arteriovenous fistulas: deterioration and embolotherapy during pregnancy. *Chest* 1990; 98:1522–1524.

11. Moore BP: Pulmonary arteriovenous fistula. *Thorax* 1969; 24:381.

12. Hoffman R, Rabens R: Evolving pulmonary nodules: multiple pulmonary arteriovenous fistulas. *AJR* 1974; 120:861–864.

13. Hodgson CH, Kaye RL: Pulmonary arteriovenous fistula and hereditary hemorrhagic telangiectasia: a review and report of 35 cases of fistula. *Dis Chest* 1963; 43:449–455.

14. Steinberg I, McClenahan J: Pulmonary arteriovenous fistula: angiocardiographic observations in nine cases. *Am J Med* 1955; 19:549.

Mastectomy: Is it necessary for occult breast cancer?

The article on occult breast cancer by Fortunato et al in this issue of the *Journal* starts with a quote from 1909, advocating radical mastectomy as a treatment for occult breast cancer. The authors go on to say that many still consider mastectomy the "standard treatment for this disease."[1,2] In 1992, such a statement is an indication of the reactivity of many of today's surgeons who are still doing mastectomies for almost all forms of breast cancer. Much has changed in the treatment of breast cancer since 1909. We now can classify tumors according to hormonal receptors, DNA content, growth rates, oncogenes, and nuclear grade. All of this helps to guide the use of chemotherapy and hormonal therapy after surgery. We have specialized mammography units which are more accurate and use less radiation exposure. Fine-needle aspiration cytology can diagnose cancer in a higher percentage of cases, even with nonpalpable lesions by using stereotactic techniques. Finally and most importantly, through a series of large randomized studies we now know that breast-conserving surgery with the addition of radiotherapy is as effective against invasive breast cancer as mastectomy.[3,4] In spite of all this progress, reports have shown that mastectomy is still the most common operation for primary breast cancer.[5]

Treatment for occult breast cancer is even more controversial. "Occult" in 1992 not only means not palpable, as it did in 1909, but also not visible by mammography. If a lesion is truly occult, what argument can there be for mastectomy when a lumpectomy is sufficient for a palpable lesion?

The patient presented in this issue of the *Journal* is typical of these patients with occult breast cancer in that she had a mastectomy and no tumor was found in the specimen.[1] The authors have done a review of the literature on occult breast cancer and have listed in a table the percentage of cancer found in mastectomy specimens of patients with occult lesions in numerous American studies. The incidence ranges from 8% to 85%. If these studies are looked at more closely, however, there are a number of reasons why these data may be misleading. The first three studies, which have the highest percentage of cancer in the specimens, are from before 1965 when mammography was not being used.[6,7,8] These studies have the highest percentage of positive specimens, but many of these tumors might have been detected on mammography and thus are not what we now consider truly occult lesions. Other studies illustrate this point by citing mammographic results, many of which are positive for disease, such as the study by Westbrook where 17 of the 18 patients had mammograms with ten showing pathology.[9] We do not know if these are the same ten patients as those with positive specimens, but one would assume this is so.

In a recent study done by Ellerbrock in 1990 from the MD Anderson Cancer Center, mammograms were performed on 40 patients. No tumor was seen in the breast on 37 studies.[10] Only one patient had a tumor found on pathology, and this was a lesion that was less than 1 cm. I think this recent report is much more characteristic of present day practice. If the mammograms are negative, the probability of finding a significant tumor on mastectomy is small.

The other factor that has to be taken into account is that even in those studies that report cancer in the mastectomy specimen, often it is an in-situ cancer or a microscopic invasive lesion. In a study this writer performed at the City of Hope, there were only five cancers found in 11 mastectomy specimens. Two of these were in situ, one was microscopic, one was 1 cm, and the last was a large lesion. These microscopic and in-situ lesions were probably incidental findings and not really the source of the axillary metastases.[11] Furthermore, all of the studies comparing outcome in women with occult breast cancer who have had either a mastectomy or only an axillary dissection show no difference in survival between those two modes of treatment. There is no evidence at this time suggesting improved survival with a mastectomy.

There is, however, evidence from studies on the psychosexual adaptation of women who have been treated by mastectomies compared to women who have received a lumpectomy.[12,13] These studies consistently show that women with mastectomies have a poorer body image and more anxiety about their appearance while not suffering any increased anxiety about cancer recurrence.

Taking all this information into consideration I do not feel a mastectomy is justified in a woman with "occult breast" cancer. The idea of doing a radical procedure and not finding tumor in the specimen in over half the cases seems unreasonable at this time. My plea would be that if a woman has a negative mammogram for occult breast cancer, she does not need a mastectomy. She does, however, need chemotherapy. Radiation therapy to the breast would be an additional option although it is not clear it is necessary. It seems to me that mastectomy should be reserved for the large tumors that cannot be treated with conservative surgery. It should not be used when there is no proof it is beneficial. A great deal of evidence has been accumulated to show that it is psychosexually detrimental.

Of course, the final decision as to choice of therapy is up to the patient. If a woman desires a mastectomy because she feels she would be too anxious with anything less, she should have the right to receive this form of treatment.

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1. Fortunato L, Sorrento JJ, Golub RA: Occult breast cancer: a case report and a review of the literature. *NY State J Med* 1992; 92:555–557.
2. Cameron HC: Some central facts regarding mammary cancer. *Br Med J* 1909; 1:577–582.
3. Fisher B, Redmond C, Poisson R, et al: Eight year results of a randomized clinical trial comparing total mastectomy and lumpectomy with or without radiation in the treatment of breast cancer. *New Engl J Med* 1989; 320: 822.
4. Lichter A, Lippman M, Danforth D, et al: Mastectomy versus breast-conserving therapy in the treatment of stage I and II carcinoma of the breast: a randomized trial at the National Cancer Institute. *J Clin Onc* 1990; 10:976–983.
5. Steele DG, Winchester DP, Mench HR, et al: National cancer data base: annual review of patient care, 1992.
6. Owen HW, Dockerty MB, Gray HK: Occult carcinoma of breast. *Surg Gynecol Obstet* 1954; 98:302–309.
7. Fitts WT, Steiner GC, Enterline HT: Prognosis of occult carcinoma of the breast. *Am J Surg* 1963; 106:460–463.
8. Feurman L, Attie JN, Rosenberg B: Carcinoma in axillary lymph nodes as an indicator of breast cancer. *Surg Gynecol Obstet* 1962; 114:5–8.
9. Westbrook KC, Gallager HS: Breast carcinoma presenting as an axillary mass. *Am J Surg* 1971; 122:607–611.
10. Ellerbroek N, Holmes F, Singletary E, et al: Treatment of patients with isolated axillary nodal metastases from an occult primary carcinoma consistent with breast origin. *Cancer* 1990; 125:43–47.
11. Kemeny MM, Rivera DE, Terz JJ, et al: Occult primary adenocarcinoma with axillary metastases. *Am J Surg* 1986; 125:43–47.
12. Kemeny MM, Wellisch DK, Schain WS: Psychosocial outcome in a randomized surgical trial for treatment of primary breast cancer. *Cancer* 1988; 62:1231–1237.
13. Steinberg MD, Juliano MA, Wise L: Psychological outcome of lumpectomy versus mastectomy in the treatment of breast cancer. *Am J Psychiatry* 1985; 142:32–39.

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TECHNIC OF RADICAL MASTECTOMY

An adequate, efficient radical mastectomy according to the fundamental principles of Halsted and Willy Meyer is still the best method of treatment for cancer of the breast.

A general form of therapy should not be chosen on the basis of statistics alone. This dictum applies to the conservative method of treatment by simple mastectomy and irradiation (McWhirter-Bruce), the super-radical mastectomy of Wangensteen with dissection of the lower cervical nodes and superficial and deep mediastinal nodes, and to the resection of the chest wall with the internal mammary nodes as developed by Urban.

These conservative and more radical authors are to be encouraged to continue their splendid work and report five and more years hence on their survival rates. Only in this manner can the final worth of these procedures be established.

At the moment their worth has not been sufficiently established and their indications sufficiently clarified to warrant the advice that the rank and file of general surgeons should try and adopt these procedures. This fact is especially true as far as the extended radical procedures are concerned in the hands of the casual surgeon, who only occasionally performs this operation, or the hinterland surgeon who does not have the equipment or adequate facilities to perform the operation.

Inadequate simple mastectomy followed by inadequate irradiation will sacrifice many lives which might otherwise be saved. As Stuart W. Harrington so wisely states, "We assume a great responsibility when we advise a radical mastectomy, but we assume a still greater responsibility when we refuse it." Every active general surgeon is well equipped and able to give his patient the great benefit of an adequate, efficient radical mastectomy.

HERBERT WILLY MEYER, MD
(*NY State J Med* 1953; 53:2977–2985)

Complications of laparoscopic cholecystectomy in a geriatric population group

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ABSTRACT. The purpose of this study was to assess the complications of laparoscopic cholecystectomy among geriatric patients in New York State who receive their medical care under the Federal Medicare Program.

A 20% random sample survey (304 patients) of the 1,520 patients who underwent laparoscopic cholecystectomy between January 1, 1990, and June 30, 1991, was selected. Coded ICD-9 diagnoses and procedures were carefully reviewed for these patients. In some patients with complications, the medical charts were reviewed by a medical epidemiologist and a surgeon to more fully elucidate the reported complications. The patients in this study were randomly selected from public, proprietary, voluntary, and teaching hospitals.

Seventy-two complications (23.68% of the 304 patients) were identified in 48 patients (15.78% of the 304 patients). Of these complications, 27 (8.88%) were surgical, and 43 (14.14%) were medical in character. There were two deaths (0.66% of the 304 patients). Individual chart review of 32 of the 48 cases with complications revealed physician-related surgical quality-of-care problems which directly contributed to complications in 10 patients (31.25%). The overall complication rate of 23.68% found in this study represents one of the highest so far reported among published studies of laparoscopic cholecystectomy.

The results of this study indicate a significant level of morbidity when laparoscopic cholecystectomy is performed on a geriatric population group. Although age-associated risk factors contributed to this high complication rate, so too did errors of surgical judgment and technique. We conclude that the high morbidity rate observed in this study can be improved by standardized training programs for surgeons that include contemporaneous supervision, appropriate credentialing and privileging standards, and careful patient selection.

(NY State J Med 1992; 92:518–520)

Laparoscopic cholecystectomy is a relatively new procedure that was first introduced in France and the United States. It rapidly gained in popularity because of economic forces and patient preference.[1–3] Patients prefer it over open cholecystectomy because of a shorter hospital stay, less pain, rapid recovery, a better post-operative cosmetic result, and the perception that it is less invasive and carries a lower risk than the traditional procedure. Widespread positive publicity in the press and media has generated significant patient demand, which in turn has forced many surgeons to learn the procedure and hospitals to acquire the necessary instruments to remain competitive. Laparoscopic equipment manufacturers, aware of the large potential market, have actively promoted the procedure and helped surgeons adopt it by offering easily accessible training courses.[4] The end result has been the widespread adoption of this new medical technology before its careful evaluation in controlled studies.[5]

A number of retrospective studies and one major prospective study have shown complication rates ranging from 0% to 9%.[6–8] The complication rates in most studies published so far compare favorably with the 6% rate and less currently found with open cholecystectomy.[5,9] However, such excellent results must be viewed in light of the fact that they are often obtained by experienced laparoscopists working in large research medical centers where careful patient selection also plays a role in reducing the frequency and severity of complications. The purpose of this study was to assess the complications of laparoscopic cholecystectomy among geriatric patients who receive medical care in New York State under the Federal Medicare Program.

MATERIALS AND METHODS

Island Peer Review Organization (IPRO) is a peer review organization that performs Medicare reviews under a contract with the Health Care Financing Administration of the US Department of Health and Human Services. Currently, IPRO conducts annually, approximately 150,000 Medicare retrospective chart reviews and over 100,000 Medicaid acute care, ambulatory, and alternate level-of-care reviews. IPRO's Medicare data base is made up of institutional claims for inpatient and ambulatory services in New York State. These claims originate with the provider and are sent to the fiscal intermediaries and carriers where they are edited, paid, and forwarded to IPRO's data system. The claims data received by IPRO include patient demographic (eg, age, sex, zip code), clinical (recorded diagnoses and procedures performed), and utilization (diagnosis-related group and length of stay) information.

During the 18-month period, January 1, 1990, through June 30, 1991, there were a total of 9,585 admissions with a principal procedure coded as cholecystectomy among the two million Medicare beneficiaries residing in New York State. Of these, 1,520 (15.86%) were identified as laparoscopic cholecystectomies on the basis of ICD-9 procedure codes. A 20% sample ($n = 304$) of the 1,520 cases identified as laparoscopic cholecystectomies were chosen randomly for the study sample. On the basis of the use of sample size and power calculations described by Rothman and Boice[10] and a review of complication rates reported in the literature, it was determined that a 20% sample would be sufficiently large to provide valid incidence rates for various complications.

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For the random 20% sample of laparoscopic cholecystectomies, the coded ICD-9 diagnoses and procedures were carefully reviewed and complications identified in 48 admissions. In 32 of the 48 admissions with complications, the medical chart was reviewed by a medical epidemiologist and a surgeon to elucidate completely the nature of the reported complications. Among the remaining 16 admissions, the nature of the reported complications was apparent from the claims data (eg, urinary tract infection or retention).

RESULTS

The age range of the 304 patients was 65 to 93 years, with a mean of 72.4 years. The mean length of stay in the hospital was 5.9 days (range = 1–82 days; SD ± 8.54 days). Cases with complications had a mean length of stay of 11.1 days compared with a mean length of stay of 4 days for cases with no reported complications. Forty-three percent of the cases with complications had a length of stay that was equal to or greater than 10 days compared to 12.21% for cases without complications. There was a significant relationship ($p < 0.01$) between extended length of stay and the occurrence of complications. During the 18 months of the study period, the number of cholecystectomies among Medicare patients in New York State did not increase significantly.

A total of 72 complications (23.68% of the 304 patients) occurred in 48 patients (Table I). Of these complications, 27 (8.88% of the 304 patients) were surgical in character, and 43 (14.14% of the 304 patients) were medical. There were two deaths (0.66% among the 304 patients). The most common surgical complication, which occurred in six patients (1.97%), was operative bleeding. Three patients (0.99%) sustained lacerations, burns, or contusions of the abdominal wall, and three (0.99%) had retained common duct stones. One patient had a superficial wound infection, and another a sub-hepatic abscess that required percutaneous drainage. Two patients (0.66%) had biliary tract injuries. The procedure was converted to a conventional cholecystectomy in three (6.25%) of the 48 patients with complications because of the following reasons: injury to a branch of the hepatic vein, severe inflammation of the gall bladder, and excessive bleeding from the gall bladder bed.

TABLE I. Surgical and Medical Complications of Laparoscopic Cholecystectomy in a Sample of 304 Medicare Patients, New York State, January 1, 1990–June 30, 1991

Complications*	Number	Incidence (%)
Surgical		
Operative Bleeding	6	1.97
Post-operative transfusion	4	1.32
Converted to an open procedure	3	0.99
Laceration, burn, or contusion of the abdominal wall	3	0.99
Retained common bile duct stones	3	0.99
Biliary tract injury	2	0.66
Pancreatitis	2	0.66
Wound infection	2	0.66
Post-operative cholangitis	1	0.33
Subcutaneous emphysema	1	0.33
Sub-total	27	8.88
Medical		
Pulmonary complications	14	4.61
Atelectasis (4)		(1.32)
Pleural effusion (3)		(0.99)
Pneumonia (5)		(1.64)
Pneumothorax (1)		(0.33)
Pulmonary embolus (1)		(0.33)
Urinary retention	10	3.29
Urinary tract infection	7	2.30
Fever of unknown origin	5	1.64
Post-operative or intra-operative electrocardiographic changes	3	0.99
Prolonged ileus	2	0.66
Gastritis	1	0.33
Post-operative congestive failure	1	0.33
Sub-Total	43	14.14
Death	2	0.66
Total	72	23.68

*Complications occurred in 48 patients.

There were 43 medical complications, of which 14 (4.61%) were pulmonary. Ten patients (3.29%) had urinary retention, seven (2.30%) had post-operative urinary tract infections, and five (1.64%) had a fever of unknown origin (Table I). Five (1.64%) patients were readmitted, two for retained common duct stones, one for a retained common duct stone with jaundice, one for pneumonia, and one for bile reflux gastroenteritis.

The charts of 32 of the 48 cases with complications were reviewed by a surgeon experienced in peer review. Physician-related surgical quality-of-care problems were identified in 10 (31.25%) of the 32 cases reviewed. It was concluded from this review that four patients should have had open procedures; two should have been converted to open procedures; three had inadequate pre-operative work-ups; and one should have had an intra-operative cholangiogram. It was also concluded from this surgical review that these quality-of-care problems directly contributed to the complications in these patients.

DISCUSSION

Since 1990, laparoscopic cholecystectomy has been rapidly and widely adopted by surgeons in the United States. The adoption of this new technology occurred before its careful evaluation in controlled studies in which morbidity and morality data could be compared with those from conventional cholecystectomy.[5] Several published retrospective studies have reported excellent results and morbidity and mortality rates which favorably compare with those associated with the open procedure. The reported complication rates in these retrospective studies have ranged from 0% to 9%, and no mortalities have occurred.[1–2,6–7,11–13] A large, prospective study conducted by the Southern Surgeons Club reported a complication rate of 5.1% and a mortality rate of 0.07%.[8] All these results favorably compare with the 6% and less complication rate, and the 1.7% and less mortality rate currently observed with open cholecystectomy.[9,14] One must view these excellent results with some degree of caution because they reflect the outcomes of experienced and skilled laparoscopists who are often enthusiasts of minimal access surgery.[15]

Compared with most other published studies, the study reported on here represents a random epidemiologic sample of patients drawn from a large Medicare population. In contrast, other published studies to date describe either retrospective or prospective analyses of carefully selected patients operated on by experienced laparoscopists. These studies also reflect careful patient selection which greatly reduces the risk and severity of complications. As a consequence, they do not provide a solid basis for assessing the frequency and character of complications when the procedure is first undertaken by the general population of surgeons.[15]

The overall complication rate of 23.68% found in this study represents one of the highest thus far recorded among published studies of laparoscopic cholecystectomy. This high rate may, in part, be due to the geriatric character of the population group analyzed. The mean age of the population was 72.4 years in contrast to mean ages of from 40 to 51.1 years in other published studies. Co-morbidities and other factors related to age may predispose this geriatric population to higher complication risks. An indicator of the prevalence of illness for this geriatric population is the fact that there were 592 hospital admissions among the 304 patients during the 18-month study period.

It would be misleading, however, to conclude that the higher complication rate observed was only due to patient risk factors. Most of the surgical complications (8.88%) were iatrogenic in character (Table I). This rate alone is higher than most of the combined surgical and medical complication rates published from other studies and in excess of the 6% and less combined surgical and medical complications recorded for open cholecystectomy.[9,14] The fact that physician quality-of-care problems, which directly contributed to complications, were found in 10 (31.25%) of 32 charts reviewed speaks to iatrogenic factors for some of the complications.

The types of complications identified do not differ from those previously reported in other studies. Their frequency, however, is significantly higher. For example, surgical complications were 2.70 times higher, and medical complications were 7.94 times higher in our study group than in the population described by the Southern Surgeons Club.[8] These higher complication rates resulted in greater lengths of stay. Forty-three percent of patients with complications had a length of stay that was equal to or greater than 10 days. On the other hand, only 12.21% of cases without complications had similar lengths of stay. The mean length of stay of 5.9 days (range = 1–82 days) was significantly longer than that of 1.2 days (range = 6 hours to 30 days) reported by the Southern Surgeons Club.[8] There was a significant relationship ($p < 0.01$ level) between an extended length of stay and the occurrence of complications in our study group. The readmission rate of 1.6% found in our study contrasts with that of 0.9% reported by the Southern Surgeons Clubs.[8]

Since the initial enthusiastic adoption of laparoscopic cholecystectomy by surgeons, and early reports of low morbidity and mortality rates associated with the procedure, some have expressed concerns and reservations about the procedure in response to anecdotal reports of a steep learning curve, serious complications, and brief training courses of unequal quality.[5] The high complication rate observed in the present randomly selected population group indicates that these concerns are well founded.

From the experience gained so far, it is clear that laparoscopic cholecystectomy is successful and carries low morbidity and mortality risks when performed on appropriately selected patients by well-trained and experienced laparoscopists. However, when the procedure is performed on geriatric patients, morbidity and mortality rates are significantly higher, as the current study shows. In the hands of inexperienced and inadequately trained surgeons, very serious complications can occur, as recently reported by Nenner et al in a series of 17 patients identified by hospitals because of quality-of-care issues.[16] In this series, laceration of the common bile duct, right hepatic duct, or an intra-hepatic bile duct occurred in nine patients. In one patient, the aorta was lacerated; in two others, the small bowel was either perforated or lacerated; and in one patient, the common bile duct and portions of the right and left hepatic ducts were resected in error.[16] Such case series do not provide a basis for deducing incidence rates for such serious complications. However, they do provide insights into the nature of these complications, and serve to alert the surgical community to the underlying issues of careful patient selection and appropriate training and privileging standards.

The results of the present study indicate a significant level of morbidity when laparoscopic cholecystectomy is performed on a geriatric population group by a diverse group of surgeons. Although some of this morbidity was due to risks associated with age, some of it may have also resulted from factors related to the training, skills, and experience of operating surgeons. On the basis of claims data, many of the surgeons represented in this study were in the early phase of the learning curve when complications are more frequent. The high morbidity rate observed in this study can in part be improved on in the future by standardized training programs that include contemporaneous supervision, by appropriate credentialing and privileging standards, and by careful patient selection.

Acknowledgments. We wish to acknowledge the assistance and suggestions of Florence Kavaler, MD, MPH, Professor of Preventive Medicine and Community Health, State University of New York, Health Science Center at Brooklyn; Harry S. Soroff, MD, Professor of Surgery, State University of New York, Health Science Center at Stony Brook; and Harriet Starr, MA, Vice President, Management Information Systems, Island Peer Review Organization. We also wish to acknowledge the assistance of John Zetzsche, MA, Research Analyst, and H. Peter Lee, SB, Research Analyst, Island Peer Review Organization.

REFERENCES

1. Perissat J, Collet DR, Belliard R: Gallstones: Laparoscopic treatment, intracorporeal lithotripsy followed by cholecystotomy or cholecystectomy—A personal technique. *Endosc* 1989; 21:(suppl)373–374
2. Reddick EJ. Olsen DO: Laparoscopic laser cholecystectomy: comparison with mini-lap cholecystectomy. *Surg Endosc* 1989; 3:131–133.
3. Stoney WS: Laparoscopic cholecystectomy: problems of rapid growth. *S Med J* 1991; 84:681–683.
4. Wolfe BM, Gardiner B. Frey CF: Laparoscopic cholecystectomy: a remarkable development. *JAMA* 1991; 265:1573–1574.
5. Chung RS, Broughan TA: The phenomenal growth of laparoscopic cholecystectomy: a review. *Cleveland Clinic J Med* 1992; 59:186–191.
6. Dubois F, Icard P, Berthelot G, et al: Coelioscopic cholecystectomy: preliminary report of 36 cases. *Ann Surg* 1990; 211:60–62.
7. Wilson P, Leese T, Morgan WP, et al: Elective laparoscopic cholecystectomy for "all comers." *Lancet* 1991; 338:795–797.
8. The Southern Surgeons Club: A prospective analysis of 1,518 laparoscopic cholecystectomies. *N Engl J Med* 1991; 324:1073–1078.
9. Pickleman J. Gonzalez RP: The improving results of cholecystectomy. *Arch Surg* 1991; 121:930–934.
10. Rothman KJ, Boice JD Jr: *Epidemiologic Analysis with Programmable Calculation*, ed 2. Chestnut Hill, MA, Epidemiology Resources Inc, 1982.
11. Zucker KA, Bailey RW, Gadacz TR, et al: Laparoscopic guided cholecystectomy. *Am J Surg* 1991; 161:30–44.
12. Peters JH, Ellson EC, Innes JT, et al: Safety and efficacy of laparoscopic cholecystectomy: a prospective analysis of 100 initial patients*Ann Surg* 1991; 213:3–12.
13. Cuscheri A, Dubois F, Mouiel J, et al: The European experience with laparoscopic cholecystectomy. *Am J Surg* 1991; 161:385–387.
14. McSherry CK: Cholecystectomy: the gold standard. *Am J Surg* 1989; 158:174–178.
15. Easter DW: A surgeon's perspective on laparoscopic cholecystectomy. *AJR* 1991; 157:241–242.
16. Nenner RP, Imperato PJ, Alcorn CM: Serious complications of laparoscopic cholecystectomy in New York State. *NY State J Med* 1992; 92:178–180.

Economic interventions to discourage the illegal sale of cigarettes to minors in New York State

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ABSTRACT. In New York State it is illegal to sell tobacco products to persons under the age of 18 years. In most communities, compliance with this law is poor. This study provides estimates of teenage cigarette smoking and the illegal sale of cigarettes to minors in 1990 in each of New York's 57 counties and in New York City. Results show that in New York State, approximately 135,700 teens between the ages of 12 and 17 years are regular cigarette smokers. Data available on the cigarette consumption and purchasing habits of teenage smokers reveal that nearly 21.9 million packs of cigarettes were sold illegally to minors in 1991 in New York State. This finding underscores the fact that cigarette sales to underage youth in New York is big business, representing $39.5 million in sales annually. Government officials should consider levying an illegal drug profit tax on the cigarette industry to recover the millions in profits derived annually from the illegal sale of cigarettes to children.

(NY State J Med 1992; 92:521–524)

Cigarette smoking kills more than 30,000 New Yorkers and costs the state nearly $4 billion annually.[1] Most tobacco victims are over the age of 35 years. However, the addiction that fosters tobacco use begins for most smokers during adolescence.[2] More than four fifths of smokers born since 1935 started smoking before the age of 21 years, and half started before age 15 years.[3]

Studies of teenage smokers show that most report obtaining cigarettes by purchasing them over the counter or through vending machines.[4–6] In theory, reducing the availability of tobacco products to teenagers should help discourage tobacco use. In an effort to protect children from tobacco addiction, most states have enacted laws to restrict the sale of tobacco to minors.[7] In 1965, New York State passed a law making it illegal to sell cigarettes to anyone under 18 years of age (Penal Law, 260.20). The law was amended in 1983 to include a ban on the sale of chewing and powdered tobacco to minors.

In spite of the existence of laws prohibiting the sale of tobacco to minors, evidence collected in New York State and elsewhere indicates that these laws are rarely enforced.[8–11] Awareness of this problem has stimulated efforts to enforce tobacco sale age-restriction laws.[12] A recent study reporting on an enforcement program in Woodridge, Illinois, provides strong evidence that these laws can work.[13] With continued merchant education, repeated sting operations to catch offenders, and assessment of penalties (including license suspensions), cigarette sales to minors in Woodridge were virtually eliminated. Surveys of Woodridge students indicate that tobacco use has been significantly curtailed as a result of the enforcement program. Unfortunately, the program in Woodridge is the exception rather than the rule.

In an effort to promote a greater awareness of the problem of youth tobacco use and access to tobacco products, this study presents estimates of teenage cigarette smoking and illegal sale of cigarettes to minors computed for each of New York's 57 counties and New York City. Legislative and economic interventions to discourage merchants from selling tobacco products to minors are discussed.

METHODS

Data collected in the 1989 Teenage Attitudes and Practices Survey (TAPS) of youths aged 12–18 years, sponsored by the Centers for Disease Control, National Cancer Institute, and American Cancer Society, were used to generate estimates of the number of teenage smokers in each of New York's 57 counties and New York City.[14] The TAPS survey is based on a representative national sample of 9,965 youths. For this analysis, we have restricted use of the TAPS data to respondents from the northeastern region of the United States ($n = 1,610$) to more closely approximate the cigarette use behavior of teenagers in New York State. Since our main interest is estimating the illegal sale of cigarettes to teenagers, we restricted our definition of a smoker to anyone between the ages of 12 to 17 years who reported having smoked in the last week. This definition provides a conservative estimate of teenage smoking but is more likely to capture minors who purchase their own cigarettes.

Table I shows the age-specific cigarette-use prevalence figures obtained from the TAPS survey for the northeastern region of the United States. Teenagers who reported having smoked in the last week were asked to indicate the number of cigarettes consumed daily. Average daily cigarette consumption by age is also reported in Table I. The TAPS data show small differences in smoking prevalence and amount smoked by gender.[14] Thus, we have chosen to combine male and female data for this analysis. Racial differences in cigarette use are evident in the TAPS data (higher among whites and Hispanics compared to blacks).[14] However, the small number of nonwhites included in the TAPS survey make age-specific prevalence estimates for blacks and Hispanics unreliable. For this reason, we have chosen to ignore race as a factor in generating estimates of teenage smoking.

Census figures from 1990 were used to estimate the number of teens in each age group by county. Multiplying the cigarette

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TABLE I. Reported Cigarette Use by Age* From the Teenage Attitudes and Practice Survey (TAPS) for the Northeastern Region of the United States, 1989

Age Group (yr)	Smoked During Preceding Week % (95% CI†)	Average Number of Cigarettes Smoked Per Day
12–13	1.0 (0.3, 2.3)	5.6
14	7.4 (3.9, 12.5)	6.8
15	12.5 (8.5, 17.6)	10.1
16	15.4 (10.6, 20.1)	10.9
17	22.2 (16.7, 27.8)	10.1

*As of November 1, 1989.
†Confidence interval.

use prevalence figures shown in Table I by the number of teens in each age group yields an estimate of the number of smokers between the ages of 12 and 17 years in each county. Multiplying the average number of cigarettes consumed daily from Table I by the number of teenage smokers in each age group provides an estimate of the number of cigarettes consumed daily by teens in each county.

Teenage smokers get their cigarettes from many different sources including vending machines, over-the-counter sales, friends, and family members.[4–6] To estimate the percentage of cigarettes consumed by teens that represent illegal sales to minors, data from a 1989 study on the cigarette purchasing habits of teenage smokers conducted by the National Automatic Merchandising Association (NAMA) were used.[4] The NAMA is a trade association for companies that sell cigarettes through vending machines. Using a "mall-intercept" technique in 20 US cities, the NAMA study assessed the cigarette purchasing habits of 1,015 teenage smokers. In that study, 72% of teenage smokers said they obtain their cigarettes by purchasing them, and an additional 19% reported that they depend on friends to supply them. Since the friends of most teenagers are themselves minors, the NAMA study indicated that 91% (72% + 19%) of all cigarettes consumed by teens are illegally sold to minors. Multiplying county-specific estimates of the number of packs of cigarettes consumed by teens by 0.91 yields an estimate of the number of packs of cigarettes sold illegally to minors each day. Multiplying this number by 365 provides an estimate of the number of packs of cigarettes sold illegally to teenagers for the year.

RESULTS

Tables II–IV show the estimated number of teenage smokers and illegal cigarette sales to minors in the western New York, capital, and metropolitan regions of New York State. Overall, there are approximately 135,700 teens who smoke an average of 1.3 million cigarettes per day. We estimate that 60,100 packs of cigarettes are sold illegally to underage youth in New York State every day; approximately 21.9 million packs per year.

In 1991, the average retail price for a pack of cigarettes in New York State was $1.80.[15] Multiplying the average retail price per pack by the number of packs illegally sold to minors provides an estimate of the revenue generated from the illegal sale of cigarettes to minors. In New York State in 1991, we estimate the total revenue produced from the illegal sale of cigarettes to minors was $39.5 million.

In 1991, combined state and federal excise taxes accounted for 30.5% of the average retail price of a pack of cigarettes.[15] The state excise tax on cigarettes is $0.39 per pack; the federal tax on cigarettes is $0.20 per pack. In 1991, we estimate that the state government collected $8.5 million in excise taxes from the illegal sale of cigarettes to minors; the federal government collected approximately $4.3 million.

DiFranza and Tye[16] estimate that the profit derived from cigarette sales is 31% of the total sale price minus taxes. Applying this percentage to the revenue generated annually in New York State from the illegal sale of cigarettes to minors, we estimate that tobacco retailers, distributors, and manufacturers shared approximately $8.3 million in profit from illegal cigarette sales to minors last year.

DISCUSSION

Given the addictive nature of tobacco use and the finding that 90% of adult smokers began their smoking careers as children, efforts to stem the epidemic of tobacco-related diseases must focus on preventing children from starting to smoke.[3] Prevention of smoking initiation requires a multi-

TABLE II. Estimated Number of Adolescent Smokers and Illegal Cigarette Sales to Minors in the Western Region (17 counties) of New York State, 1990

County	Estimated Number of Adolescent Cigarette Smokers Ages 12–17	Total Number of Cigarettes Smoked Daily	Number of Packs of Cigarettes Illegally Sold to Minors: Daily	Number of Packs of Cigarettes Illegally Sold to Minors: Annually
Allegany	440	4,273	194	70,963
Cattaraugus	755	7,344	334	121,964
Chautauqua	1,154	11,215	510	186,258
Chemung	752	7,318	333	121,541
Erie	7,090	69,068	3,143	1,147,051
Genesee	483	4,706	214	78,154
Livingston	472	4,585	209	76,139
Monroe	5,179	50,402	2,293	837,054
Niagara	1,709	16,631	757	276,202
Ontario	718	6,978	317	115,880
Orleans	353	3,436	156	57,065
Schuyler	161	1,567	71	26,027
Seneca	285	2,785	127	46,258
Steuben	862	8,375	381	138,090
Wayne	749	7,267	331	120,686
Wyoming	360	3,500	159	58,123
Yates	191	1,855	84	30,808
Total	21,713	211,305	9,613	3,508,263

TABLE III. Estimated Number of Adolescent Smokers and Illegal Cigarette Sales to Minors in the Capital Region (35 counties) of New York State, 1990

County	Estimated Number of Adolescent Cigarette Smokers Ages 12–17	Total Number of Cigarettes Smoked Daily	Number of Packs of Cigarettes Illegally Sold to Minors: Daily	Number of Packs of Cigarettes Illegally Sold to Minors: Annually
Albany	1,963	19,121	870	317,551
Broome	1,474	14,344	653	238,217
Cayuga	656	6,399	291	106,272
Chenango	448	4,334	197	71,979
Clinton	646	6,299	287	104,616
Columbia	515	5,041	229	83,724
Cortland	385	3,748	170	62,248
Delaware	389	3,795	173	63,020
Dutches	1,938	18,862	858	313,252
Essex	292	2,841	129	47,181
Franklin	379	3,686	168	61,211
Fulton	487	4,745	216	78,808
Greene	345	3,367	153	55,910
Hamilton	42	406	18	6,742
Herkimer	558	5,448	248	90,479
Jefferson	871	8,466	385	140,592
Lewis	252	2,449	111	40,679
Madison	545	5,298	241	87,991
Montgomery	418	4,072	185	67,625
Oneida	1,912	18,609	847	309,042
Onondaga	3,383	32,882	1,496	546,096
Orange	2,503	24,276	1,105	403,170
Oswego	1,048	10,173	463	168,956
Otsego	452	4,403	200	73,117
Rensselaer	1,162	11,335	516	188,249
St Lawrence	938	9,119	415	151,440
Saratoga	1,483	14,436	657	239,755
Schenectady	1,068	10,405	473	172,801
Schoharie	268	2,614	119	43,414
Sullivan	518	5,046	230	83,808
Tioga	458	4,451	202	73,921
Tompkins	551	5,350	243	88,849
Ulster	1,190	11,589	527	192,460
Warren	507	4,942	225	82,072
Washington	509	4,958	226	82,345
Total	30,553	297,309	13,526	4,937,592

faceted approach addressing the supply of tobacco, the demand for it, and the environment in which it is used.[12,17,18]

The public supports stronger limits on children's access to and use of tobacco products.[19] Instead of continuing to give lip service to the problem of youth tobacco use, government officials should give serious consideration to enacting laws that would truly limit children's access to tobacco products.[12] As a starting point, the New York State legislature should consider doubling the current $0.39 per pack excise tax on cigarettes as a public health measure to discourage teens from smoking. Raising the price of cigarettes through taxation or other means is one of the most effective ways to discourage teenagers from smoking.[2,20,21] Compared to adults, teens have relatively little disposable income and are therefore more sensitive to increases in the cost of cigarettes. Although cigarette taxes have increased

TABLE IV. Estimated Number of Adolescent Smokers and Illegal Cigarette Sales to Minors in the Metroplitan Region (5 counties and New York City) of New York State, 1990

County	Estimated Number of Adolescent Cigarette Smokers Ages 12–17	Total Number of Cigarettes Smoked Daily	Number of Packs of Cigarettes Illegally Sold to Minors: Daily	Number of Packs of Cigarettes Illegally Sold to Minors: Annually
Nassau	9,597	93,506	4,254	1,552,895
Putnam	695	6,798	309	112,903
Rockland	2,381	23,177	1,055	384,907
Suffolk	11,304	110,216	5,015	1,830,412
Westchester	6,128	59,681	2,716	991,163
New York City*	53,309	518,889	23,609	8,617,442
Total	83,414	812,267	36,958	13,489,722

*New York City includes the following five boroughs: Bronx, Kings, Manhattan, Queens, and Richmond.

over time, the taxes as a percentage of the retail price are substantially lower today compared to 30 years ago (30.5% *v* 50.4%).[15]

The legislature should also consider levying an illegal drug profit tax on the cigarette industry to recover profits obtained through the illegal sale of cigarettes to minors.[16] Eliminating the profit gained from selling cigarettes illegally to teenagers might make the tobacco industry more responsible about keeping their product out of the hands of children. A precedent for this tax already exists in the "windfall profits tax" levied on the oil industry for overcharging customers in response to the energy crisis in the 1970s.

Given the amount of profit made from the illegal sale of cigarettes to minors, one has to question the sincerity of the Tobacco Institute's current campaign (Helping Youth Say No) to discourage young people from smoking.[22–24] The total expenditure for this national campaign has been reported to be approximately $10 million annually.[22] This amount pales in comparison to the profit made by the tobacco industry from the illegal sale of cigarettes to children.[16]

Finally, the legislature should consider earmarking the tax revenue collected from the illegal sale of cigarettes to minors to finance tobacco control intervention, including programs to enforce the laws regulating minor's access to tobacco. In view of the magnitude of the tobacco problem in New York State, it is distressing to observe the low level of tobacco control activities carried out by government agencies charged with protecting public health. A 1990 survey of county health departments revealed that most of those reporting any activity restricted their tobacco control programs to the distribution of literature (unpublished data, Bureau of Adult and Gerontological Health, New York State Department of Health, February 1990). At the state health department level, tobacco prevention and control activities account for only a small fraction of the overall budget (0.031%).[25] Although many nongovernmental agencies sponsor a wide range of tobacco control activities, in all communities the resources devoted to combat the tobacco problem are substantially lower than the resources expended by the tobacco industry to encourage smoking.[2]

REFERENCES

1. Cummings KM, Stiles J, Mahoney MC, et al: The health and economic costs of cigarette smoking in each of New York's 57 counties and New York City: 1987–89. *NY State J Med* 1992; 92:470–473.
2. Centers for Disease Control: Reducing The Health Consequences of Smoking: 25 Years of Progress—A Report of the Surgeon General. Rockville, MD: US Department of Health and Human Services, Public Health Service, 1989, DHHS publication no. (CDC) 89–8411.
3. Centers for Disease Control: Differences in the age of smoking initiation between blacks and whites—United States. *MMWR* 1991; 40:754–757.
4. Findings from the study of teenage cigarette smoking purchasing behavior. Prepared for the National Automatic Merchandising Association. Chicago, IL, Response Research Inc, 1989.
5. Stenwick RS, Fish DG, Manfreda J, et al: Where Manitoba children obtain their cigarettes. *Can Med Assoc J* 1987; 137:405–408.
6. Forster JL, Kunt-Inge U, Jeffery RW: Sources of cigarettes for tenth graders in the two Minnesota cities. *Health Educ Res* 1989; 4:45–50.
7. Centers for Disease Control: State laws restricting minors access to tobacco. *MMWR* 1990; 39:349–353.
8. Office of Inspector General: Youth Access to Cigarettes. Washington, DC: US Department of Health and Human Services, May 1990.
9. Cummings KM, Marshall DR: Awareness of the New York law banning the sale of tobacco to minors. *NY State J Med* 1988; 88:418–419.
10. Skretny MT, Cummings M, Sciandra R, et al: An intervention to reduce the sale of cigarettes to minors. *NY State J Med* 1990; 90:54–55.
11. Hoppock KC, Houston TP: Availability of tobacco products to minors. *J Fam Prac* 1990; 30:174–176.
12. Davis RM: Reducing youth access to tobacco. [editorial] *JAMA* 1991; 266:3186–3188.
13. Jason LA, Py J, Anes MD, et al: Active enforcement of cigarette control laws in the prevention of cigarette sales to minors. *JAMA* 1991; 266:3149–3153.
14. Centers for Disease Control: Teenage cigarette smoking among youth—United States, 1989. *MMWR* 1991; 40:712–715.
15. Tobacco Institute: The tax burden on tobacco. Washington, DC, Tobacco Institute, vol 26, 1991.
16. DiFranza JR, Tye JB: Who profits from tobacco sales to children? *JAMA* 1990; 263:2784–2787.
17. Feighary E, Altman DG, Shaffer G: The effects of combining education and enforcement to reduce tobacco sales to minors. *JAMA* 1991; 266:3168–3171.
18. DiFranza JR. Carlson RR, Caisee RE Jr: Reducing youth access to tobacco. [letter]. *Tobacco Control* 1992; 1:58.
19. Centers for Disease Control: Public attitudes regarding limits on public smoking and regulation of tobacco sales and advertising—10 US communities, 1989. *MMWR* 1991; 40:344–345, 351–353.
20. Cummings KM, Sciandra R: The public health benefit of increasing tobacco taxes in New York State. *NY State J Med* 1990; 90:174–175.
21. Kaiserman MJ, Rogers B: Tobacco consumption declining faster in Canada than in the US. *Am J Public Health* 1991; 81:902–904.
22. Tobacco Institute tells kids: Say no. *Advertising Age* February 3, 1992, pp 1,8.
23. Lipman J: Why activists fume at anti-smoking ads. *Wall Street Journal* February 20, 1992, B9.
24. Warner F: Tobacco takes back the fight. *Adweek's Marketing Week* February 24, 1992, p 16.
25. Centers for Disease Control: State tobacco prevention and control activities: Results of the 1989–1990 association of state and territorial health officials (ASTHO) survey final report. *MMWR* 1991; 40(no R-11):1–41.

Low level and bystander exposure to lead among factory workers

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ABSTRACT. A clinical field survey of 36 workers employed in the manufacturing of leaded products used in radiologic safety was conducted. Although the principal source of lead exposure was limited to an area where two individuals mixed lead oxide and vinyl liquid, increased lead absorption was found in other workers employed in adjacent areas. The mean lead concentration of the plant workers was 29.3 μg/dL (SD ± 9.0), and four workers had blood lead levels of 40 μg/dL or higher. A statistically significant correlation was noted between blood lead and serum creatinine; linear regression analysis demonstrated that cigarette smoking was a significant factor in predicting the blood lead level. The exposure encountered in this plant may be typical of lead exposure in many similar industrial settings. It calls attention to the risk of excessive exposure among workers who are not working at the primary source of exposure, and should be considered in the design of medical surveillance programs for workers employed in small manufacturing plants.

(NY State J Med 1992; 92:525–528)

Lead is a metallic element which is ubiquitous in the human environment. The metal can be found in plants, oceans, rivers, and drinking water, as well as in soil and in various food items. Lead is also present in the air and attaches to dust particles. Consequently, the possibility of exposure to humans in some form or other is great, and the presence of the metal in man is a reflection of environmental contamination. The potential for exposure to humans stems primarily from urbanization and industrial development. The blood lead concentrations of persons living in an industrialized society are much higher than those living in remote areas.[1,2]

It appears, however, that the phasing out of leaded gasoline, one of the most important sources of environmental lead exposure, has had a significant effect on blood lead levels among many segments of North American populations, and has caused a lowering of the mean blood lead concentration during the past decades.[3] This signifies a reversal of a long trend of exponential growth in environmental lead pollution over thousands of years, paralleling man's development and his manipulation of the environment.[4]

The effects of excessive exposure to inorganic lead are of great concern to modern society. The current efforts to control environmental lead exposure have been spurred by investigations suggesting that children may exhibit impaired neurologic development as a result of increased lead absorption from sources in the general environment. The definition of lead toxicity in the community has recently been set at 10 μg/dL for children. It is estimated that some 17% of preschool children in the United States have blood lead concentrations in the toxic range.[5–7] Moreover, lead poisoning is still a common occupational disease and poses a significant occupational health problem in certain areas of the US.[8–10]

The Occupational Safety and Health Administration (OSHA) promulgated a standard for occupational exposure to inorganic lead in 1978.[11,12] The lowering of permissible levels of exposure and mandated surveillance of blood lead concentrations have rendered the diagnosis of acute occupational lead poisoning with extremely elevated blood lead levels relatively uncommon. As additional measures, several states have established heavy metal registries and require reporting of elevated blood and urine levels of metals including lead. The reporting levels in most states with registries (25 μg/dL) is lower than the federal blood lead standard for occupational exposure (40 μg/dL), and the potential for early intervention is thereby improved.

During the past several years we have observed subtle biochemical and symptomatic effects from relatively low-level lead exposure among various occupational groups including firearms instructors, cable manufacturing workers, and cable splicers.[13,14] More recently we had the opportunity to examine a group of workers exposed to inorganic lead during the manufacturing of lead-containing shielding materials used to ensure radiologic safety for personnel employed in hospitals and in nuclear power facilities. The principal items produced were vinyl lead products such as aprons, gloves, and other clothing materials.

The company had provided blood lead screening tests for the employees before this investigation. The mean blood lead levels of workers, who had been tested during a three-year period preceding the current study, had shown an increase from 12.5 μg/dL (range 5–22 μg/dL) to 18.8 μg/dL (range 7.8–34.0 μg/dL). Four workers examined eight months before this examination had a mean blood lead level of 32.5 μg/dL (range 24.0–44.0 μg/dL). The fact that one worker exceeded the current OSHA blood lead standard of 40 μg/dL (1.9 μmol/L) prompted our evaluation of all employees.

METHODS

The study population consisted of 36 workers. They all volunteered for the study and comprised the total active work force at the time of the examination. Informed written consent was obtained for each worker. For purposes of this investiga-

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tion, the plant was divided into five work areas (Fig 1). Area C was the area in which lead oxide in powder form and a liquid vinyl compound were mixed together and baked to form a leaded vinyl compound. This was the area with the greatest potential for significant exposure to airborne lead compounds according to observations made after an industrial hygiene walk-through. Air measurements of lead were not available. Area A was primarily a carpentry shop where wood products were made and lead sheets were incorporated into the various products, such as doors and walls. At one location, a small amount of lead was melted in a pot by one worker using a heat source under a pot. This was done once or twice per week, and there was potential for exposure to lead fumes; the molten lead was poured into small holes at the corners of the wood frame for reinforcement. Grinding of leaded metal surfaces was not performed, although cutting of large sheets of lead was done. The latter operation was not associated with any generation of airborne lead dust.

In Area B, sheet metal work and spray painting of leaded metal cabinets was performed. The cutting and sewing of the vinyl lead fabrics took place in Area D. The office area was designated as Area E. All areas of the plant were essentially continuous, except for the office area, which was a separately ventilated unit. The plant could be reached directly from the office through a door. There was little movement of workers between areas A, B, D, and E because of the relatively specialized operations in the respective areas. A separate lunchroom was not provided. There was no specific ventilation system in the plant.

The evaluations were performed in the office area of the plant. Each individual underwent an interview, including recording of demographic data, occupational history and work practices at the company, a brief medical history and previous history of lead exposure, and lead screening tests. Blood tests consisted of the measurement of lead in whole blood by atomic absorption spectrophotometry performed by the Bureau of Laboratories of the New York City Department of Health, an OSHA approved laboratory, measurement of free erythrocyte protoporphyrin (FEP), complete blood count, and a biochemistry screen that included blood urea nitrogen (BUN) and serum creatinine. Blood samples for lead analysis were taken in beige top Vacutainer tubes (controlled for lead content).

The data were encoded, entered, and analyzed with the assistance of the Statistical Analysis System (SAS) PC software package. Student's t-test and analysis of variance procedure (ANOVA) were performed to compare mean values of blood lead concentration. Pearson correlation coefficients were calculated to evaluate correlations between blood lead level, FEP, hemoglobin, serum creatinine, and location within the plant. Multivariate regression models were also used to identify factors, such as duration of employment, personal industrial hygiene precautions, and smoking habit, which predict the blood lead concentration.

Every worker received a letter with an explanation of the test results, and no information was provided to a third party without the written authorization by the worker. All workers consented to providing the management with the blood lead and FEP data, since this information is essential for both effective industrial hygiene intervention and planning of medical surveillance. Medical follow-up was arranged for all individuals with abnormal test data. Most of the non-lead-related abnormalities, such as abnormalities in serum lipids and blood glucose, were managed by each worker's personal physician, on our recommendation. Follow-up of lead-related findings were managed by us.

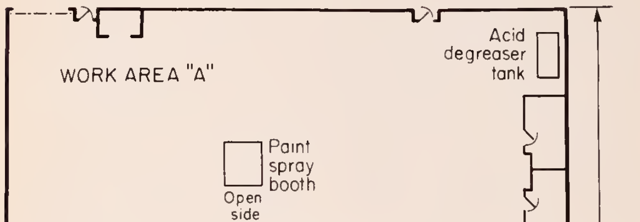

FIGURE 1. Floor plan of the factory. Lead oxide in powder form was mixed with a liquid vinyl compound in Area C.

Results

Demographic Data, Work, and Personal Habits. The examined group of workers consisted of 13 females (36.1%) and 23 males (63.9%). The mean age was 41.8 years, and the mean total number of years of employment at the plant was 12.8 years. With regard to principal work locations, eight employees (22.2%) worked in Area A; six (16.7%) worked in Area B; two individuals (5.6%) worked in Area C; and nine individuals (25%) were employed in Area D. Nine employees worked in Area E, which was the office area (Fig 1). Two individuals (5.6%) performed their work as supervisors throughout all areas of the plant and were present in Area C at some times.

Sixteen workers (44%) reported perceiving exposure to lead dust or fumes at work, whereas one person (2.8%) gave a history of occupational exposure to lead before working at the plant. It should also be noted that three individuals (8.3%) reported past or present exposure to lead through a hobby. Two of these practiced firearms training, one person in the past and the other currently. Their blood lead levels at the time of the examination were 14 μg/dL and 34 μg/dL, respectively. The third individual did autobody work and had a blood level of 37 μg/dL. None of the examined workers reported a history of elevated blood lead levels or lead poisoning before to working in the plant.

In terms of personal work habits, 28 employees (77.8%) reported washing up before leaving work, but only 14 (38.9%) reported changing clothes before leaving work. Thirty-four workers (94.4%) reported washing hands before eating. The majority of workers (72.2%) stated that they ate lunch within the plant. Review of smoking habits demonstrated that 15 individuals (41.7%) gave a history of current cigarette smoking. Among the smokers, 13 (86.7%) smoked within the plant, which was permitted by plant management. Three persons (20%) reported washing hands before smoking, although 80% of the smokers did not.

Indexes of Lead Absorption and Effects. The distributions of the blood lead and FEP levels are shown in Table I. The mean blood lead level of the examined workers was 25 μg/dL (SD ± 10.9) with a range from 9 to 46 μg/dL. The mean FEP concentration was 30.1 μg/dL (SD ± 18.1), with a range from 16 to 119 μg/dL. As expected, there was a significant difference between the blood levels of the office workers and the other plant employees; the mean blood lead concentration for the nine office workers was 12.1 μg/dL (SD ± 2), although the mean blood lead level for the 27 plant workers was 29.3 μg/dL (SD ± 9). It should be noted that one of the office workers had practiced firearms training in the past, and six of the nine office employees were smokers. None of the employees had high enough blood lead levels to meet the criteria (>60 μg/dL) for temporary medical removal from lead exposure established in the OSHA lead standard. However, four individuals had levels at or above the OSHA blood lead standard of 40 μg/dL.

To examine the effects of various personal hygiene precautions taken to minimize lead absorption, a comparison was made between the blood lead levels of (non-office) plant workers who routinely performed hygienic tasks, such as washing hands before eating, eating outside the work place, washing up before leaving work, and changing clothes before leaving work, and those who did not. The only significant difference detected was between those who washed before leaving work (mean blood lead 30.5 μg/dL, [SD ± 8.9] and those who did not (mean blood lead 19.7 μg/dL, [SD ± 2.3; $p = 0.004$]).

Although the principal exposure source in the plant was in Area C, the area where lead dust and fumes were most prominent, there were 15 workers among those not directly employed in Area C, who were found to have blood levels above the mean of 25 μg/dL. Of these workers, eight were smokers and seven were non-smokers; one practiced firearms training. None had worked in Area C, where the mixing of the lead compound took place. Comparisons between mean blood lead levels of workers employed in the other areas (A, B, and D) did not yield any statistically significant differences. The mean blood lead levels for the various areas are shown in Table II.

A statistically significant correlation was found between blood lead concentration and serum creatinine ($p = 0.038$). There was no significant correlation between serum creatinine and age ($p = 0.28$), or between blood lead and age ($p = 0.79$).

It is interesting that the correlations between blood lead and FEP and between blood lead and hemoglobin were not statistically significant ($p = 0.27$ and $p = 0.16$, respectively). Thus, most individuals appeared not to have reached a threshold for enzymatic inhibition of heme synthesis as measured by FEP. When the correlation between FEP and blood lead level was examined with the exclusion of the one single extreme value for FEP of 119 (and a concomitant blood lead concentration of 17 μg/dL), the correlation coefficient was 0.54 ($p = 0.0007$) (Fig 2). When the correlation between FEP and blood lead levels was studied for the 22 individuals with blood lead levels of 20 μg/dL or higher, the correlation coefficient increased to 0.65 ($p = 0.001$).

TABLE I. Distribution of Blood Lead and FEP Concentrations Among 36 Manufacturing Workers

FEP (μg/dL)	Blood Lead Level (μg/dL)					
	<10	10–19	20–29	30–39	40+	Total
<30	2	10	7	3	0	22
30–44	0	2	1	7	2	12
45>	0	1	0	1	0	2
Total	2	13	8	11	2	36

TABLE II. Mean Blood Lead Concentrations by Work Area

Work Area	Number of Workers	Mean Blood Lead Concentration (μg/dL)
A	8	27.1*
B	6	34.0*
C	2	40.5†
D	9	26.7*
E	9	12.1‡

*Not significantly different.
†Significantly different from A, B, D, and E.
‡Significantly different from A, B, C, and D.

The person with the high FEP value was a female with a hemoglobin count of 12.4 g/dL, hematocrit of 36.8%; and serum iron level of 149 μg/dL. Iron deficiency was not the reason for the elevated FEP concentration. She had only worked at the plant for six months. Past blood lead levels were not available; however, she had not reported any lead-related occupation before working in this plant.

Linear regression analysis demonstrated that smoking was a significant factor in predicting the blood lead level ($p = 0.007$). As previously indicated, the use of personal hygiene precautions to avoid lead exposure was negatively associated with blood lead level (regression coefficient = 3.84, $p = 0.039$).

Discussion

This investigation was performed at a plant in which lead exposure had been at a relatively low level, as determined by blood lead levels measured during the three years before this study. Although the principal exposure source was an area in which lead oxide was mixed with a liquid vinyl compound, other production areas were located in the vicinity of this area, suggesting a potential for more widespread lead exposure.

The data indicate that indirect (bystander) exposure did, in fact, occur among these workers and that it was prudent

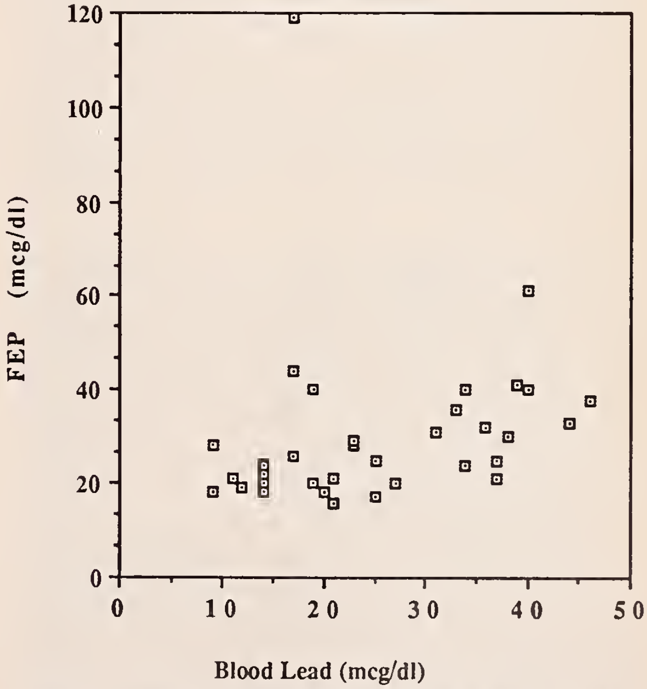

FIGURE 2. Scatter plot of blood lead and FEP levels among 36 factory workers.

to include the total work force in the assessment of degree of lead absorption. Consideration should be given to low level, albeit significant, bystander exposure when regulating work place conditions and designing medical surveillance programs for the protection of the entire work force.

We found an inverse correlation between seemingly prudent personal hygiene precautions and blood lead levels. One possible explanation for this finding is that only three individuals did not wash, and none of them were in the highest lead exposure areas. It is probable that they did not take these precautions because they perceived no risk of exposure. In contrast, those who worked in areas in which lead exposure was perceived as a potential hazard, may have been more observant of personal hygiene measures. Thus, the difference in blood lead levels is more likely a reflection of different exposure situations, rather than the effect of personal hygiene precautions. This observation reinforces a well-known principle in occupational hygiene, namely that sole reliance on personal hygiene precautions is an ineffective way of achieving optimal exposure control, and that ventilation and exhaust of the air pollutant should be the primary control measure. Recommendations were made for improvement of industrial hygiene in the plant.

The results of this investigation also demonstrate that some biochemical disturbances were associated with the relatively low degree of lead absorption observed in this population. Although the correlation between blood lead and FEP was not significant when the entire group was considered, a stronger correlation between blood lead and FEP level was found when the calculations were repeated and limited to those individuals with blood lead levels of 20 μg/dL or higher, and after exclusion of one extreme value.

A statistically significant correlation of creatinine with blood lead level was observed, although creatinine concentration did not appear to be a predictor for blood lead level by regression analysis. Lead-related effects on renal function have been documented, but usually with higher and more chronic occupational exposures. However, a recent study of individuals without industrial lead exposure demonstrated a correlation between serum creatinine and blood lead level among male subjects.[15] Evaluation of the present study population with more sensitive tests, such an *n*-acetyl-glucose-aminidase,[16] would provide more detailed information about renal tubular function, which is of particular interest in the context of the present exposure situation.

Other work sites and occupations associated with high risk for lead poisoning include battery manufacturing plants, smelters, automobile radiator repair shops, flamecutting of metal structures covered with lead containing paint (often performed by ironworkers), the handling of lead containing plasticizers in plastics manufacturing, and firearms instruction, particularly in indoor shooting galleries.

The OSHA standard and a more recent review[17] contain the details of the comprehensive medical surveillance program for lead-exposed workers.

Blood sampling is required every six months in low-risk industries (ie, when repeated blood lead levels have been below 40 μg/dL). If an employee has been found with blood lead concentration above 40 μg/dL but below the obligatory removal level of 60 μg/dL, blood tests must be repeated at least every two months until the blood lead concentration is below 40 μg/dL. If the blood lead concentration exceeds 60 μg/dL, the examining physician must recommend to the employer that the worker be removed from lead exposure, and repeated measurements should be made every month. The worker may be advised to return to lead-related activities only if two consecutive blood tests have been 40 μg/dL or lower.

In addition to laboratory tests, medical surveillance must include a thorough clinical evaluation, as well as supplementary blood tests, such as zinc protoporphyrin (ZPP) or FEP, complete blood count, serum creatinine, and blood urea nitrogen. Semen analysis and pregnancy testing, if requested by the employee, has to be performed. As mentioned above, more stringent regulations are provided by the heavy metal registries in several states, with required reporting of a blood lead concentration of 25 μg/dL. The registries also provide for follow-up of the worker's immediate family members, particularly children who may be at risk for excessive lead exposure in the home environment (the threshold level for children is 10 μg/dL). The administration of both ZPP, FEP, and blood lead tests usually provides sufficient information to characterize both the degree of absorption (blood lead) and the biological response (ZPP and FEB), although the reporting to registries is currently limited to the blood lead levels.[18]

Longitudinal investigations of lead-exposed workers may provide much needed information about the potential impact of low-level, long-term occupational lead exposure on various health parameters.

REFERENCES

1. Fergusson JE: *The Heavy Elements: Chemistry, Environmental Impact and Health Effects*. Oxford, Pergamon Press, 1990.
2. Piomelli S, Corash L, Corash MB, et al: Blood lead concentrations in a remote Himalayan population. *Science* 1980; 210:1135–1137.
3. Annest JL, Pirkle JL, Makric D, et al: Chronological trend in blood lead levels between 1976 and 1980. *N Engl J Med* 1983; 308:1373–1377.
4. Patterson CC: Contaminated and natural lead environments of man. *Arch Environ Health* 1965; 11:344–360.
5. Needleman H, Schell A, Bellinger D, et al: The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. *N Engl J Med* 1990; 322:83–88.
6. Lippmann M: 1989 Alice Hamilton Lecture: lead and human health. Background and recent findings. *Environ Res* 1990; 51:1–24.
7. Agency for Toxic Substances and Disease Registry: The nature and extent of lead poisoning in children in the United States: a report to Congress. Atlanta, US Dept of Health and Human Services, Public Health Service, Atlanta, DHHS document 99-2966, 1988.
8. Rudolph L, Sharp DS, Samuels S, et al: Environmental and biological monitoring for lead exposure in California workplaces. *Am J Public Health* 1990; 80:921–925.
9. Maizlish N, Rudolph L, Sutton P, et al: Elevated blood lead in California adults, 1987: results of a statewide surveillance program based on laboratory reports. *Am J Public Health* 1990; 80:931–934.
10. Papaneck PJ, Ward CE, Gilbert KM, et al: Occupational lead exposure in Los Angeles County: an occupational risk surveillance strategy. *Am J Ind Med* 1992; 21:199–208.
11. US Department of Labor, Occupational Safety and Health Administration: Occupational exposure to lead: Final standard. Fed Reg 43(220):52952–53014 and 43(225):54353–54616, 1978. Fed Reg 46(238):60758–60776, 1981.
12. Code of Federal Regulations, Special Edition of the Federal Register, 1988; 29: ch XVII, July 1 ed, 1910.1025, pp 832–870. Office of the Federal Register.
13. Fischbein A, Thornton J, Blumberg WE, et al: Health status of cable splicers with low-level exposure to lead: results of a clinical survey. *Am J Public Health* 1980; 70:7,697–700.
14. Fischbein A, Thornton JC, Sarkozi L, et al: Subjective symptoms in workers with low-level exposure to lead. *J Applied Toxicol* 1982; 2:289–293.
15. Staessen J, Yeoman WB, Fletcher AE, et al: Blood lead concentration, renal function, and blood pressure in London civil servants. *Br J Ind Med* 1990; 47:442–447.
16. Meyer BR, Fischbein A, Rosenman K, et al: Increased urinary enzyme excretion in workers exposed to nephrotoxic chemicals. *Am J Med* 1984; 76:989–998.
17. Rempel D: The lead exposed worker. *JAMA* 1989; 262:532–534.
18. Surveillance for occupational lead exposure—United States, 1987. *MMWR* 1989; 38:642–646.

A history of Jewish attitudes toward nursing

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The history of nursing is closely bound to the status of religion[1,2] and women[3–5] in the society of their times. One finds in the literature on transcultural aspects of nursing numerous accounts of the religious requirements of Jewish patients[6,7] but not of the social aspects of nursing in this population. There is no entry on nursing in great contemporary Jewish encyclopedias.[8] Relatively few American Jewish women entered this career in the past, a phenomenon not mentioned by feminist historians of nursing. Statistics on this score are scanty. Of 2,353 graduates of the Bellevue and New York Hospital schools between 1875 and 1920, only four were Jewish.[9] Four of the 152 students of the Somerville Hospital in the greater Boston area from 1903 to 1939 were of this faith.[5] There were none among the 203 women entering the Training School of the Pennsylvania Hospital between 1898 and 1909.[10] The paucity is the more remarkable because of a religious obligation for the care of the sick, the particularly honored place of the physician, and the vaunted nurturing nature of the Jewish woman.

The following essay probes this seeming anomaly. It is based in part on historical sources and in part on an anecdotal survey by the author of 15 women who became registered nurses before 1946. Several extracts of their comments are cited in the pages that follow. The situation can be understood only in the context of nursing in the larger American scene of the same era and in Jewish communities of other times and places. From the analysis emerges the idea that cultural determinants were more important than anti-Semitism in accounting for the small number of Jewish nurses. This is in contrast to the situation in medical schools where discrimination had been the driving force in limiting admission of Jews. The phenomena involved reflect to varying degrees the historical evolution of nursing in general. As the social isolation of the immigrants receded and the status of the profession improved, Jewish women entered more fully into nursing careers.

Nursing in Late 19th Century America

In Christian tradition, such as Roman Catholic nursing orders and Lutheran and Anglican deaconesses, nursing was regarded as a holy calling. However, nursing had not been honored in America when the major waves of Jewish immigration from eastern Europe began in the 1880s. It had been an unskilled and menial occupation, often filled in public institutions by women morally unfit to give patient care.[11] Formal training schools did not begin until 1873. They came into being through reform efforts of upper class women appalled by the pitiable state of indigent patients in public hospitals and almshouses. An avowed aim was to give nursing a respectability and so attract women of high character. At first, the schools sought educated, American-born women for training; most were of Anglo-Saxon extraction. The exigencies of the First World War and the pandemic of influenza in 1918 greatly increased the call for student nurses. With this, the social base changed and pupils increasingly came from rural and mining areas to escape poverty. Many developments unfolded over the next period and some still are in flux.

Nursing in the Jewish Tradition

Although Jewish hospitals and their medieval hospice (*hekdesh*) precursors in the cities of Europe long antedated the massive waves of Jewish immigration to America, little is known of nursing care in these institutions. Most illness was tended in the home by the women of the families. Folk remedies were transmitted by word of mouth. Some women acquired special healing skills, such as midwifery. One reads repeatedly of these good souls who dedicated themselves to assistance of ill and poor neighbors.

As in church hospices and public almshouses in Europe and America at the time, the *hekdesh* was a charitable refuge for all unfortunates, not only the helpless ill. It was maintained by a society (*chevra*) called *Bikkur Holim* (literally Visitors of the Sick). This *chevra* was one of several mandatory welfare agencies of the organized Jewish community. In larger cities, the *hekdesh* often was a sophisticated institution; in impoverished areas, the facilities were quite wretched and the term *hekdesh* thus became a Yiddish pejorative for any filthy place. Visiting and nursing the sick was an obligation assigned by the overseer to members of the *chevra*,[12] but inmates were cared for also by permanent occupants known as *Anshey Hahekdesh*. The caretakers lived in the facilities with their charges. Men cared for men; and women for women. The female attendant usually was the wife of the custodian.[13] They fed, washed, gave prescribed medicine to the patients and provided them the comfort of prayer. When, at times, the caseload became too heavy, additional personnel were hired. The *chevra* also was obliged to give home care when one of the family had to leave for work and there was no one else to attend the ill.[14]

The trend toward professionalizing nursing in European hospitals took place more rapidly in countries not governed by the Church than in those that were;[1] hence, changes were seen two generations earlier in Germany than in Poland. As the hospitals took on a modern character in the latter part of the 19th century, there were increasing

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Presented in part at the History of Nursing Day, School of Nursing, State University of New York at Stony Brook, Jan. 28, 1991.

pressures in the Jewish institutions, too, for replacing traditional visitors by trained nurses.

There are records of Jewish nurses working at the Jewish Hospital in Berlin over a century ago.[15] Education of Jewish women as professional nurses was encouraged in Germany before 1900.[16] Nursing, it was argued, would be a gainful occupation, a solace to invalid co-religionists and a beneficence that would gain the approval and admiration of the German public as a whole. Jewish hospitals in Hamburg, Frankfurt, Berlin, Koln, Breslau, and elsewhere created their own training schools. They were patterned after the Lutheran deaconess system. Even the dark garb was adopted, except that the pendant was a Star of David rather than a Cross. Competition for admission was brisk.

According to A. Philips born,[17] the first nursing association (*Verein*) was formed in Hamburg in 1892. The Frankfurt school opened shortly thereafter.[18] Hospital training lasted a year and a half, six months longer than state examinations required. Sister organizations soon formed in other large cities. They federated into a national professional society, Deutschoer Verband Jüdischer Krankenpflegerinnezereine, in 1905. By 1914, they had hundreds of members. At all times, the standards of the schools were high. The practice of nursing was strictly regulated and graduates were effectively indentured to their *Verein*. Hopes that the schools would be populated exclusively by middle class women proved to be unrealistic however.[19] The career militated against marriage, the social desideratum. Anxieties were expressed by families, too, that their daughters would be exposed to infection or worked beyond their strength. Most students came from lower socioeconomic status although educational requirements for admission were higher than those of the Christian confessional schools. They were willing to trade off the regimented life for the financial benefits and security that the training offered.

After the World War, other occupations opened up for women, competed with nursing, and conditions liberalized. An analogous organization for educating Jewish nurses and protecting their professional interests was inaugurated in Prague, Czechoslovakia, in 1889.[20] Jewish Red Cross nurses served in the military hospitals during the first World War. Alumnae later found the education stood them in good stead when they fled the Nazis. In Britain, they were eagerly absorbed to meet the personnel shortage during the Second World War.

The situation was quite different in the Russian sector of Poland whence the bulk of the Jewish immigrants came after 1880. Before the First World War, virtually all hospital nursing in that country, whether Catholic or state-run, was in the hands of the religious.[1] Nuns offered three principal advantages to hospital administrators: They were dedicated, inexpensive, and compliant. They had virtually no scientific training, although some developed authentic bedside skill.[21]

At the turn of the century, there were 130 Jewish hospitals with 3,890 beds in Russia.[22] Professional nursing came to them no later than it did to non-Jewish institutions through several routes. The Jewish Hospital in Warsaw, by 1899, was one of the largest and most advanced in Europe. Even then, there were conflicts between its administration and the more traditional organization of visiting night aides. The latter were untrained laymen accused of being dirty and noisy and doing unwise things (eg, giving patients tea to drink when that was contraindicated medically). The plea was to replace them with trained personnel of the sort already working day shifts.[23] When a nursing school was finally created there many years later, the first director found that there were 350 attending doctors for the 1200 patients in its 12 pavilions. "When I arrived the hospital employed about 30 men and women called 'nurses.' These were regarded as a somewhat higher type of servant than the scrubwoman."[24] The eastern European Jews at the time of emigration to America thus had little exposure to formal nursing. What little they had was characterized by poor quality or tainted with an alien and hostile religion. The double meaning of *Schwester*, nurse and nun, was to be a problem for some of the immigrant girls in America contemplating entering the field. A touching moment is told by one respondent to my inquiry, a Bellevue graduate, class of '26, whose mother had been widowed at an early age:

> When my mother wrote to her parents in Russia that I was going to be a nurse she explained that I was not going to be a nun. There was no term for nurse in Yiddish except "kronken shwester" (Sister) and this work was done by nuns.

The backwardness of nursing in eastern Europe must be regarded as one of the major factors in making it unattractive to the Jewish immigrants.

The recommendation that professional nursing, with a particular orientation to public health, be introduced to eastern Europe was attributed to Herbert Hoover, who headed the American relief mission there after the First War. The area was devastated by epidemics of typhus that fell heavily on the Jews. Two professional nursing schools were opened for the first time in Warsaw in 1921, one under American, the other under Polish Red Cross auspices.[25] The Rockefeller Foundation, as part of its international program to upgrade nursing education, donated generously to the Warsaw State Training School. Jewish applicants were not accepted according to the constitution and bylaws of the Polish schools.[24]

In 1922, at the instigation of a Jewish relief agency, the American Joint Distribution Committee,[26] a third nursing school was created at the Jewish Hospital in Warsaw. The statement by Sellew and Nuesse,[1 (p415)] that the Rockefeller Foundation sponsored the Jewish Hospital and nursing school, is incorrect. The hospital antedated the foundation. Rockefeller support of the school was denied by officials both of the school[24] and the foundation.[27] It perhaps needs to be said that at that time Jews constituted a large minority, in some cities the majority, of the population in Poland. Furthermore, the language they spoke was not Polish, but Yiddish. The Warsaw hospital was now a municipal facility and already the largest in the country. It had, unlike the others, specialty pavilions for obstetrics and pediatrics, in addition to medicine and surgery.

The school opened in July 1923 under the direction of an American nurse, Amelia Greenwald (Fig 1). Its academic program was based on the curriculum of the University of the State of New York. By all accounts, it compared well with the better American schools of the time.[28] Three years later, its supervision was turned over to the school's own graduates.

The students were Jewish.[24,29] All were educated and spoke three or four languages. There were many more applicants than the school was able to accommodate. Of 100 candidates for the first class, 21 were accepted. The majority of the graduates in the first years stayed on to work

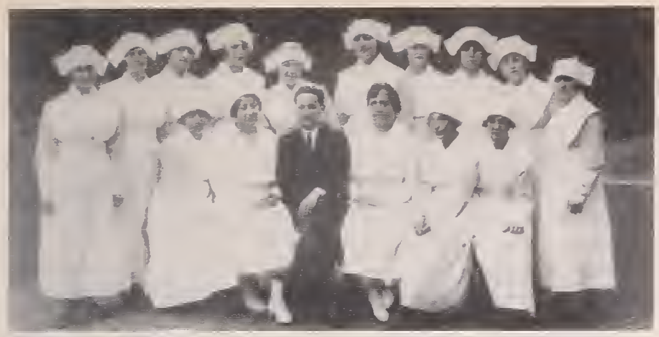

FIGURE 1. Amelia Greenwald and the first graduating class of the School of Nursing of the Jewish Hospital of Warsaw, 1925 (courtesy of the American Joint Distribution Committee Archives).

at the Hospital. By 1937, the school had awarded diplomas to 245 graduates. About one-third were employed there and constituted much of the nursing staff. Increasing anti-Semitism caused about one-sixth to emigrate to Palestine. Some of the trained nurses on the Hospital staff were not Jewish. A larger proportion of the "grey nurses," equivalent to our "practical nurses," as well as ward attendants were Gentile.

There was relatively little objection by Orthodox male patients to physical care by female nurses. A student who attended the school between 1938 and 1940, recalled, however, that an elderly religious man burst into tears when he had to be catheterized by her.[29] In American hospitals of all shades at the time, modesty forbade the catheterization of men by women.

Another training school for Jewish nurses opened in Vilna in 1922.[30] This city had been the center of Jewish learning for centuries. Unlike the Warsaw school, it was sponsored by TOZ, the Polish version of OZE (Obschestvo Zdravookhraneniya Yevreyev, Society for the Protection of the Health of the Jews). This was an enormous umbrella organization formed in Russia in 1912.[31] This school offered a two-year course. The didactics, including laboratories in chemistry and physics, were given during the first six months at night in a rented "gymnasium," a facility analogous to our senior high school (Fig 2). There followed 18 clinical months in the Jewish Hospital. Unlike the Warsaw school, the language of instruction was Yiddish.[32] The teachers were members of the medical staff, and the diploma was recognized by the Lithuanian government.[33] Training schools also came into being in Rovno and at the Poznanski Hospital in Lodz.

There had been, in 1921, a short-lived Jewish Training School for Public Health Nurses in the city of Rovno.[33] Little is known about it other than that it awarded a diploma after six months of study. The students participated in fighting the typhus epidemic. Forty-eight women were graduated in the first class. Then it was disbanded because the American sponsors did not have the resources to sustain it in the face of the commitment to the school in Warsaw. The Joint Distribution Committee also made a small contribution to starting a school at the Jewish Hospital in Krakow but decided that it, too, was not financially viable.[35]

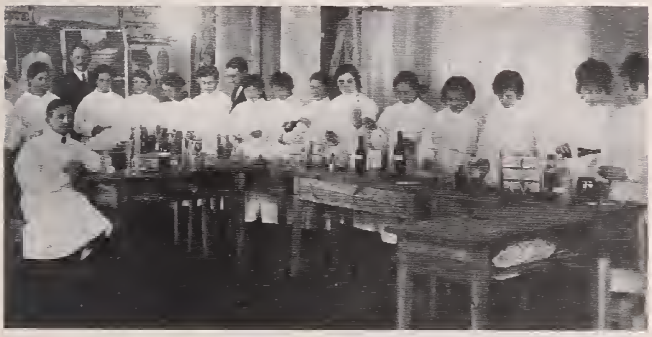

FIGURE 2. Laboratory class of the Nurses Training School of the Jewish Hospital of Vilna (c 1922). Note the wallposters in Yiddish (courtesy of the American Joint Distribution Committee Archives).

Another environment in which nursing by Jewish women thrived was in Palestine, later Israel. It had its start in 1912 with the importation of Jewish nurses from abroad (Fig 3). The American, Henrietta Szold, horrified by the poverty and lack of hygiene of the Jewish inhabitants, recruited two New Yorkers, Rose Kaplan and Rachel Landy, to go to Jerusalem.[36] From this grew the Hadassah Hospital School of Nursing, which became a prized institution. Nursing at the Orthodox hospital in Jerusalem, Shaare Zedek, was organized by a 1913 graduate from Hamburg-Selma Maier. She was known as Schwester (Sister) Selma in the German style.[37] By the time of independence (1948), there were seven Jewish nursing schools in Israel, and their numbers subsequently increased.

The Immigrants and the Hospitals

The immigrant Jews in America congregated in cohesive enclaves in the major cities of the East. Although there was much diversity in their social attitudes, they were bound by strong ties of loyalty to family and community. Fears of

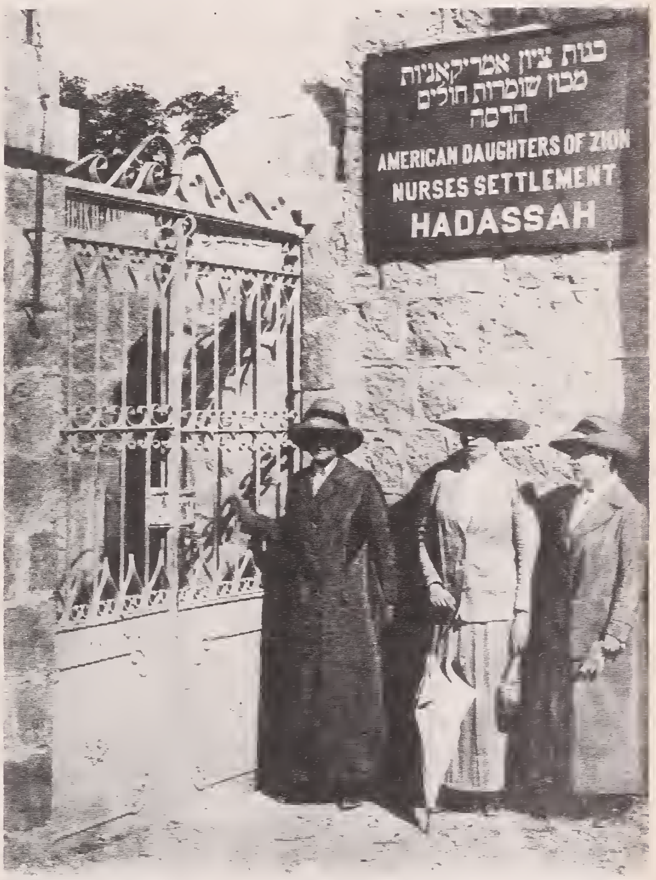

FIGURE 3. Rachel Landy, RN, (left) and Rose Kaplan, RN, (right) at the gate of the Nurses Settlement in Jerusalem, 1913 (courtesy of the Hadassah Archives).

age-old, anti-Semitism and enforced conversion to Christianity were woven firmly into their fabric. Proselytism was an established and open feature of the Protestant and public hospitals here and became a major basis for the creation of the alternative Catholic and Jewish hospital systems.[38] In time, there were to be 62 Jewish hospitals in the United States. Sexual modesty and avoidance of physical contact with men not of the immediate family were mandated by custom and religious precept. Poverty-stricken immigrant women turned primarily to clothing trades for employment; their daughters preferred teaching or social work over nursing.

THE NEGATIVE IMAGE OF NURSING

The reasons for the negative image of nursing appear to have been multiple. Aside from factors already mentioned, they included religious scruples, sexual mores, the role of women in Jewish family life, the menial nature of nursing, the poor reputation for high moral standards.

Seven of 13 nurses who responded on this score acknowledged opposition to their career choice from their families. Another four women were dissuaded from following this course, often to their lasting regret. The negative attitude was strongly felt and clearly expressed in a letter to the author by an 82 year-old religiously observant nurse who told of her experience in 1924:

> At sixteen I told my parents that I intended to enter a nurse's training school. My mother wept—my father slapped me so hard I wince today just remembering it. But I was stubborn, became a student, graduated after 2-1/2 years, and instead of congratulations my parents refused to recognize me. My aunts and uncles felt the same disgust for me—after all, think of all the dirty work I did—and (sh-sh) all the naked men I saw!

Three respondents who enjoyed support at home for their career choice came from families living by idealistic notions of the Enlightenment, variously socialist, Zionist, or intellectual.

ROLE OF WOMEN IN THE JEWISH FAMILY

Among the Jews of Eastern Europe and those who emigrated to the United States, the pivotal social value was the strength of the family and the integrity of the community. In this, the woman had a well-defined role: to be wife and mother, to keep the family together, and to guide the religious development of her children.[39] Assertion of the moral worth of Judaism persisted even after traditional religious observance attenuated. In times of need, which were frequent, women worked to supplement the family income, but this was not a desideratum. The type of work undertaken carried varying degrees of esteem, and determined how the women and men felt about themselves. The most inferior jobs were those in domestic service; they were avoided by those who had other skills, such as sewing or trading. It has been estimated that only 12.5% of Jewish girls worked as servants compared with up to 40% of other immigrant groups.[40,41]

Student nurses had to live in hospital facilities, and this loosened the ties with home. The demands of the hospital schedule would interfere with observation of the Sabbath, a force in home life even when the younger generation may no longer have shared the religious compulsions of its parents. Three of seven respondents who went ahead with their training acknowledged that they were driven by a stubbornness or desire to break from the mold of their families. The Second World War opened opportunities for some to do this by entering the Cadet Nurse Corps. Parents could hardly object to the good cause. A few of the graduates went on to make military nursing their career.

MENIALITY

There was a view widely shared in the country that the respectability so cherished by the reformers of nursing education had declined following the First World War. Training schools commonly went to monastic extremes to prevent the young women from being placed at moral risk. Access to drugs and liquor posed a threat. Like domestic servants or waitresses, young girls were subject to sexual harassment. So, too, however, were garment trade workers, largely Jewish immigrant girls, as vividly portrayed in Abraham Cahan's famous novel, *The Rise of David Levinsky*.[42]

At the time of the immigration, nursing in fact had much in common with domestic occupations. One took directions without recourse was not respected by the physician, worked long hours, had distasteful janitorial functions, earned low pay, and lived away from home. Within one or two generations, the Jews moved upward socially and entered the middle class. Other occupations or leaving the work force altogether then became possible.

CONTACT WITH THE OPPOSITE SEX

It cannot be disputed that perceived prohibitions against physical contact between the sexes militated against a nursing career in observant communities of a past era. Even so, relief of human suffering took priority over doctrine in the minds of some. A 1938 graduate of the school in Lodz, Poland, wrote me:

> I came from a very orthodox family. My grandfather (Layzer Lipschic) was a great "Rav" [rabbi] in Ozorkow, Poland. . .[He] gave me permission to enter nursing. . .Grandpa allowed me to ride the street car on Shabat (Sabbath), even Yom Kippur (Day of Atonement). [He] believed, if I were able to help sick people, I should have all the privileges. . .He taught me humanity—and was my intellectual guide.

In fact, the religious teachings have been interpreted in different ways. The *Code of Laws*[43] compiled in the Middle Ages stated that:

> A male is not allowed to attend a woman who is suffering from abdominal pains, lest he will be overcome by desire since he is capable. But a woman may attend a male who is suffering since he is sick and incapable.

Lord Immanuel Jakobovits,[44] an eminent British Orthodox rabbi, has pointed out that neither the Talmud nor the Codes mention any laws that impose restrictions on the physician in the treatment of female patients. The Responsa were relatively liberal, in comparison with other religions, with respect to this subject. A need for chaste conduct was urged on practitioners. In some instances, physical examination was obligated to women (eg, in infertility and assisting at childbirth). The principle has been explicitly extended by religious law, *Halacha*, for a female nurse to attend a male patient.[45]

Nursing was considered so improper for Jewish women that at the Beth Israel Hospital in New York, observant men at times requested bed care by male orderlies; or, if these were not available, by Gentile nurses.[46] Sex distinctions are still made in ultra-Orthodox communities in

America. Male physicians may attend Hasidic women, but female physicians may not take care of men. A Williamsburg, Brooklyn community was persuaded to participate in a tuberculosis and diabetes survey in 1946,[47] but women were not to draw blood from them! In general, Hasidim take a pragmatic view of health care and the nursing of men in hospitals does not create a problem. For the rebbe, the exalted leader of the community, matters are different. David Rozen relates that when the saintly head of the New Square village was in his final illness, the men of the community gathered to tend his physical needs and spare him the affront of being touched by female attendants.[48]

The first national gathering of Jewish nurses took place in America only in 1989 and under Orthodox auspices.[49] Several major ethical issues, working on the Sabbath, abortion, and sexual morality, were addressed. At the same time, a 220-bed hospital, Ma'ayanei Hayeshua, was dedicated in Israel for the ultra-Orthodox to ensure that patients would be cared for only by nurses of their own sex.[50] Similar sentiments existed in American hospitals at the beginning of professional nursing. N. Tomes[10] recounts the outrage felt at the Pennsylvania Hospital in 1885 when Miss Sharpless, the first woman nurse on the male surgical ward, helped a very sick man to change his outer garments. "A cry of horror" went up from physicians and others.

ANTI-SEMITISM

Six of the 11 nurses who responded to my inquiry concerning anti-Semitism recalled that Jewish girls were not wanted in American nursing schools or were exposed to anti-Semitic expressions. A graduate of the Boston Beth Israel class of February 1929 recounted:

> The only hospital to limit Jews was the prestigious Mass. General. One day a girl appeared in our dorms. She had applied to Mass. General, been accepted because her last name was Greene. When they found out she was Jewish, suggested she apply at B.I. "You'll be happier among your own."

This cannot explain the relative paucity of Jewish nurses because many Jewish hospitals formed schools of nursing that were less restrictive. There were at least 14 of them.[51] According to Bullough and Bullough,[2] these hospitals were in the forefront of the nursing reform movement. There were charges that even some of them, Michael Reese in Chicago and Mount Sinai in New York, also had quotas for Jewish candidates.[52] In the prestigious Jewish hospital schools, the majority of the pupils were for the most part Gentile.

JEWISH HOSPITAL TRAINING SCHOOLS

The Mount Sinai Hospital Training School for Nurses in New York was organized in 1881.[53] The proposal to create it did not gain universal favor initially. The committee of the Hospital Board of Directors, in recommending its establishment, wished to attract Jewish students but anticipated difficulties in accomplishing this. It stated in somewhat tortured language:

> The vital objection and one which your Committee feels, is a great bar to the immediate establishment of such a school, is the difficulty of obtaining nurses to be trained (probation nurses) from among our co-religionists: we have not been educated to feel that a professional nurse is not in a manner, so to speak a position, wanting in dignity, and we fear that the school cannot be filled with Jewish females or that a majority of them will be of our faith, thus affording them a means of livelihood for the future, besides which we feel that great good will be derived from their service by those families who prefer Jewish nurses at the bedside of their sick or dying.
>
> If whilst it is to benefit the sick of Mt. Sinai Hospital it cannot at the same time prove advantageous to our race, we would not recommend its establishment for the results would not recompense the great labor and expense. However on the other hand our directors feel that our female co-religionists would take part by offering their services and accepting the positions assigned them, we heartily recommend the project, and to that end would suggest that the aid of our ministers be called in to speak there of from their respective pulpits and to further the project in other ways as to them seems fit and proper.[54]

The Training School had a separate corporate status but it was represented on the Hospital board. There were no formal stipulations with respect to religion except that a place of worship would be provided for prayer on the Sabbath. The large majority of names in the roster of graduates during the first twenty years were not Jewish. At first, there were no established educational criteria for admission. Examinations were held to affirm the good character of the candidates and their diction in English or German.[52] Later, diplomas or other certificates of education were required. The school lasted until 1971.

The Training School for Nurses of New York's Beth Israel Hospital ran a different course from the preceding in keeping with the divergent constituencies and missions of the two institutions. Its doors opened in 1902. In its goal to have nurses sympathetic to the Orthodox immigrant patients and able to communicate with them in Yiddish, Beth Israel was willing to train promising candidates who did not yet meet the formal language or educational requirements of Mount Sinai. The School was managed by a committee of the Board of Directors rather than by a separate corporation. In explaining the need for the School, the chairman of the committee stated that no other hospital took Jewish girls into their nursing schools.[55] Of the eleven women graduated in the first class in 1904, at least nine had clearly Jewish names. The superintendent was Alice T. Kurtz; her assistants were Sadie C. Sharp and Lena R. Rabinowitz.

There was no flood of applicants. The entrance requirements included a "good English education." The class of 1907 had a predominance of Irish students, and they outshone the Jews by a wide margin. Their command of English must have been an important variable. For many years thereafter, the large majority of Beth Israel's graduates was Jewish. Vigorous efforts were made over the next two decades to attract Jewish women and to upgrade their pre-nursing credentials. Advertisements were placed in Jewish newspapers and magazines. The Hebrew Immigrant Aid Society cooperated in guiding immigrant girls to the School. In 1907, steps were taken to "instruct probationaries to enable those not conversant with English to learn the language sufficiently to pass the examinations." In 1920, the State Board of Nursing found that the educational standards for admission were deficient. To correct this, the School engaged high school teachers to tutor pupil nurses so that they might acquire the requisite State Regents' credits. This included instruction in elocution, a practice that caught the eye of newspapers in 1924.

The programs and facilities of the Beth Israel School for Nursing improved greatly over the next years. After the Second World War, the Jewish character of the student body, like much else of the patient population and staff, attenuated.

The Montefiore Hospital Nursing School in New York was established in 1922 but disbanded nine years later when there was a glut of graduate nurses in the country. At that school, too, non-Jewish students predominated.[56]

The Training School for Nurses of the Jewish Hospital of Cincinnati was established in 1892. The hospital, the second oldest of its kind, by this time was "non-sectarian" although its avowed raison d'etre was the care of the Jewish poor. No mention appears in the minutes of its Board of Trustees about religious concerns in forming the Training School. The first superintendent, Mary Hamer Greenwood, reported the following: "The constant endeavor since the institution of the training school has been to raise the standards of the requirements for admission and by so doing to accept as pupils only women of education and refinement, no distinction as to creed, or country being made."[57] Among the few Jewish trainees in the next years, one went on to a position of prominence: Naomi Deutsch, class of 1912, later became the first Director of Public Health Nursing in the Children's Bureau in Washington.

MIDWIVES

Approximately half of the births in America during the first decade of the 12th century were attended by midwives rather than medical doctors. The newly arrived immigrants followed this practice. It was in keeping with their experience in Europe and was far less expensive. Mother and midwife shared the alien language. The midwife gave support in addition to delivery by providing housekeeping and nursing care; doctors did not. There is a dearth of primary source material on this subject before 1910. What information is available is confounded by the way in which the immigrant data were presented. The newcomers were broken down by the country of their birth. We must assume that "Russian" immigrants to Manhattan or the mills of Lawrence, Massachusetts,[58] at the time were overwhelmingly Jewish.

Midwives from England and northern Europe had a long tradition of disciplined professional training. They were often more skilled at delivery than physicians. In America, obstetrics was recognized as the weakest area of medical training. A study commissioned by the city in 1906 concluded that the New York midwife was "essentially medieval," very different from the European midwife. Fully 90% were "hopelessly dirty, ignorant, and incompetent."[59] There were no legal standards. Less than half had some form of European diploma. More acquired papers from diploma mills on this side.[60]

In 1905, 31% of babies born to "Russian" immigrants in Manhattan were delivered by midwives.[61] Approximately 15% of all the midwives were Russians. In the adjacent Italian neighborhoods, more than 90% of deliveries were attended by midwives. By the 1920s, midwifery had dwindled throughout urban America under the onslaught of the medical profession, and the Jewish midwife disappeared. An important asset of the physician in effecting the switch was his ability to employ anesthesia. In 1906, a Jewish Maternity Hospital opened in New York City; it merged with Beth Israel in 1930.[62] It would take another four decades for the concept of a nurse-midwife to resurface.

SOME LEADING CONTRIBUTORS TO AMERICAN NURSING

Individual Jews left their mark on nursing even before the formal establishment of the profession. Phoebe Yates Pember (Fig 4) was the "legendary matron" of the Chimborazo Hospital in Richmond, the capital of the Confederacy during the Civil War.[63] Up to this time, it was the world's largest military hospital; 76,000 patients were cared for there during the war. Pember had no training in nursing, but in the custom of the time, her early widowhood was taken as an asset. Family obligations, it was believed, might distract the nurse from dedicated ministry to the care of her wards. Pember's appointment, as those of her counterparts in the North, evoked much opposition because she entered into what previously had been regarded as a male domain. One of the ward surgeons commented "in a tone of ill-concealed disgust that 'one of them had come.' " Pember proved, however, to be a tough administrator. She wore a pistol on her hip to protect the rum barrel, the only available anesthetic, from the predations of staff and less needy patients.

FIGURE 4. Phoebe Yates Pember (1823–1913), legendary matron of the Chimborazo Hospital (Portrait by Malcolm Thurgood).[63]

The philanthropist, Adolphus Solomons (1826–1910), was not only a doer of good deeds for Jews, but a close associate of Clara Barton in founding the American Red Cross.[64] He was responsible for introducing the idea that the Red Cross should send trained nurses into areas hit by epidemic disease. This was first tried in the outbreak of yellow fever in northern Florida in 1888. Only in 1912, did the organization create a rural visiting nurse service.

Lillian D. Wald (1867–1940) was born of prosperous German-Polish parents. She was graduated from the New York Hospital Training School for Nurses in 1891. Two years later, she took up residence in a tenement on the lower east side of New York to bring hands-on nursing care to the immigrant poor. With the support of the financiers, Jacob Schiff and Solomon Loeb, a building was obtained at 265 Henry Street to assist their work. From 1893 to 1895, the facility was known as the Nurses' Settlement. Thereafter, a wider base of benefaction expanded the services and the name was changed to the Henry Street Settlement. This was the definitive beginning of public health nursing in the United States and a milestone in the development of social work. Although Wald, Schiff, and Loeb were Jewish, as were most of the clients at the time, a policy decision was made early that the settlement was to have no religious affiliation. It was to be humanitarian rather than sectarian.[65] Unlike Schiff and Loeb, Wald maintained a distinct detachment from Jewish identity. At times her insensitivity to the religious needs of her clients brought her into conflict with Schiff.[66] While decrying Nazi anti-Semitism, she wrote in 1934: "...it is fundamentally not more dangerous than their suppression of Catholic freedom and liberal thought, and their suppression of the rights of women."[67] Even her close friend, Lavinia Dock, criticized Wald as not being sufficiently outspoken on the persecution of the Jews at that time.[66]

Another major Jewish figure in advancing the profession was Josephine Goldmark (1877–1950). Goldmark was the sister-in-law of Louis D. Brandeis. She was a social worker and collaborated with Brandeis on improving the position of women. From 1919 to 1923, she conducted the first national study on the state of nursing education under the aegis of the Rockefeller Foundation.[68] The importance of the Goldmark report in upgrading nursing education has been likened to that of the Flexner report in medicine.

Regina Kaplan (1887–1957) was born of an immigrant consumptive mother in Denver and trained in that city.[69] She became superintendent of nurses at the Leo N. Levi Hospital in Hot Springs, Arkansas, in 1916. This was the country's first hospital for rheumatism, founded by B'nai B'rith. The kitchen remained kosher although most patients were not Jewish. Kaplan was a feisty individual. She was responsible for the idea of creating a national arthritis research foundation. Later, she became vice-president of the American Hospital Association.

Mathilda Scheuer (1890–1974) became a registered nurse in 1910 and soon entered into public health work. She was an activist for the poor and later, as president of the American Nurses Association, an aggressive leader for advancement of the profession.[70]

There was little of Jewish content in these efforts other than asocial conscience that Jewish culture and its coincidence with the reformist political ferment in America at the time dictated.[71] Then revolutionary ideas, public health nursing and feminism, drew major figures such as Naomi Deutsch and Margaret G. Arnstein into Wald's orbit. These well-meant impulses had a wide variety of expression. At one extreme was the anarchist-feminist leader, Emma Goldman, "...one of the most significant women in America in the years before World War I."[72] Goldman was a nurse. The Henry Street Settlement was not sufficiently radical in righting the wrongs of society to her mind.[66] Other Jewish women found an outlet for their social idealism in Zionism. Henrietta Szold, differed from her contemporaries, Wald and Goldmark. As Brandeis, she was of the upper class and a Zionist. She envisioned that the Hadassah nursing resource in Jerusalem would be patterned after the Henry Street Settlement.[36]

Conclusion

There was an old saw that Jewish women did not make good nurses. Exactly what was meant by that is not clear. To some it was that they did not take orders kindly. Rose M. Hauer, supervisor of nursing at New York's Beth Israel Hospital for many years, found Jewish nurses to be as dedicated to their patients as their Gentile peers.[46] Without exception, each of the respondents acknowledged deep gratification with their career choices. One 78-year-old respondent wrote the following:

> My application for nursing school in New York City [in the late 1920s] was completed but I needed to have a physical examination to finalize the papers...The doctor (I cannot even remember his name) laughed when I handed him the form to fill out. He told me that "Jewish women do NOT make good nurses." He dissuaded me by saying that the work was "menial" and not fit for Jewish girls and that the pay was poor and the hours too long.
>
> Years later in the 1940s I trained with American Red Cross and did the "menial" jobs in a hospital in Far Rockaway. I loved it! For years I cheerfully gave back rubs and massaged feet and other limbs, fed patients, carried bed pans and, in those days, gave bed baths. I still volunteer in the Emergency Department of Sacred Heart Hospital. I'll always be sorry I listened to that doctor.

The prejudices against nursing of the immigrant generations were by no means confined to the Jews.[73] They receded as the communities in America became less insular and the character of the profession improved. Academic degrees in nursing surely must have appealed to the intellectualism prized in Jewish culture. In the past, a proportion of Jewish women became nurses because it was their only option for a career in health care when medical education was closed to them. The freer entry of women into medicine at present has drawn away some who formerly would have become nurses. Even so, there are now large numbers of Jewish nurses in America. As this essay is completed, the first call for an organization of nurses affiliated with Hadassah in 1990 attracted over 300 members in Boston alone.[74] Many have become leaders in contemporary nursing administration and education. These include Evelyn R. Benson, Claire M. Fagin, Rose M. Hauer, Adele Herwitz, Jacqueline Hott, Myra E. Levine, Ingeborg Mauksh, Rachel Rotkovitch, and Thelma M. Schorr among others.

Since this manuscript was completed, my attention has been drawn to a recent volume, which contains a wealth of information about the Training School of the Boston Beth Israel Hospital.[75]

Acknowledgments. Many persons have assisted in this study. Their names are listed in alphabetical order: Evelyn R. Benson, Stefani Bloch, Esther Bodkin, Hilda F. Broida, Clara Budd, Hadara E. Chemnick, Edward Cherney, Walter Cherney, Sylvia Derzon, Cornelia K. Fox, Rose Hauer, Paula E. Hyman, Marion Kaplan, Elaine M. Katzman, Helena Kavalier, Chana Kurtzman, Myra E. Levine, Barbara Melosh, Edward T. Morman, Ruth Pally, Miriam Pincus, Harriet Pinkert, Susan Reverby, Lottie Robins, Rachel Rotkovitch, Annette Schildkraut, Ida B. Schneidman, Yemema P. Seligson, Jeannette B. Steinberg, Rivka A. Stockler, Ruth E. Taffim, Judy Travis, Hedy B. Turnbull, Dorothy Welles, Frieda Wohl, Roslyn Yoachim, and Pearl Zele.

It is a pleasure, too, to acknowledge the cooperation of Betty Emilio of the Health Sciences Library at Stony Brook; Janusz Kapuscik of the Glownej Biblioteki Lekarskie, Warsaw; and the following archivists: Alice Cornell, Cincinnati Jewish Hospital, University of Cincinnati; Denise Gluck, Joint Distribution Committee, New York; Lucinda Manning, Teachers College Nutting Collection; Kevin Profitt, American Jewish Archives, Hebrew Union College, Cincinnati; Barbara J. Niss, Mount Sinai Hospital, New York; Melissa Smith, Rockefeller Foundation; Debra Tadevich, Beth Israel Hospital, New York.

I am particularly grateful to Drs Daniel M. Fox, Claudia Prestel, and, Nancy Tomes for critical review of the manuscript.

Abbreviations of Archives. AJA, American Jewish Archives; BI, Beth Israel Hospital, New York; CJH, Cincinnati Jewish Hospital; JDC, Joint Distribution Committee; MSH, Mount Sinai Hospital, New York; RF, Rockefeller Foundation; TC, Teachers College, Columbia University.

REFERENCES

1. Sellew G, Nuesse CJ: *A History of Nursing.* St Louis, CV Mosby, 1946.
2. Bullough VL, Bullough B: *The Emergence of Modern Nursing,* ed 2, New York, Macmillan, 1969.
3. Ashley JA: *Hospitals, Paternalism and the Role of the Nurse.* New York, Teachers College Press, 1976.
4. Melosh B: *The Physician's Hand: Work Culture and Conflict in American Nursing.* Philadelphia, Temple University Press, 1982.
5. Reverby SM: *Ordered to Care: The Dilemma of American Nursing, 1850–1945.* Cambridge University Press, 1987.
6. Boyle JS, Andrews MM: *Transcultural Concepts in Nursing Care.* Glenview Il, Scott Foresman, 1989, pp 405–409.
7. Rotkovitch R: The Jewish heritage, in Carnegie E (ed): *Ethnicity and Health.* New York, National League on Nursing, 1976, pp 37–46.
8. *Encyclopaedia Judaica,* Jerusalem, Keter, 1974; *Jewish Encyclopedia,* Gordon, 1976.
9. Mottus, JE: *New York Nightingales: The Emergence of the Nursing Profession at Bellevue and New York Hospitals, 1850–1921,* Thesis, New York University, 1980, p 215.
10. Tomes N: "Little world of our own": The Pennsylvania Hospital Training School for Nurses, 1895–1907. *J Hist Med* 1978; 33:507–529.
11. Kalisch PA, Kalisch BJ: *The Advance of American Nursing,* ed 2, Boston, Little Brown, 1986, p 95.
12. Baron SW: *The Jewish Community: Its History and Structure to the American Revolution.* vol 2. Philadelphia, Jewish Publication Society, 1942, p 328.
13. Marcus JR: *Communal Sick-Care in the German Ghetto.* Cincinnati, The Hebrew Union College Press, 1947, p 139.
14. Glicksman WM: *Jewish Social Welfare Institutions in the Memorial (Yizkor) Books: Studies in Jewish Communal Activity.* Philadelphia, ME Kalish Folkshul, 1976, p 44.
15. Hartung von Doetinchem D, Winau R: *Zerstörte Fortschritte. Das Jüdische Krankenhaus in Berlin, 1756–1861, 1914–1989.* Berlin, Ed. Hentrich, 1989.
16. Feldmann G: *Jüdische Krankenpflegerinnen.* Cassel, Gebruder Gotthelft, 1901.
17. Philipsborn A: The Jewish hospitals in Germany. *Yearbook,* Leo Baeck Institute 1959; 4:220–234.
18. *XVI. Jahresbericht des Vereins für Jüdische Krankenpflegerinnen zu Frankfurt am Main.* Frankfurt, G. Grunebaum, 1909.
19. Prestel C: Erschliessung neuer Erwerbszweige fur judische Frauen: Das Beispiel der Krankenschwester. *METIS.* Z Hiftor Frauenforsch Fem Prax.
20. *Jahresbericht des Sections zur Heransbildung israel. Krankenpflegerinnen des Central–Vereines zur Pflege Jüdischer Angelegenheiten.* Prag, D. Kuh, 1900–1901.
21. Greenwald A: Nursing education in Poland. Unpublished manuscript, 1925. TC, Microfiche 396.
22. Weissenberg S: Jüdische Krankenhauser in Russland. *Z Demog Statist Juden* 1908; 4:137–140.
23. Kramsztyk Z: O sluzbie szpitalnej w Szpitalu Starozakonnych. [About hospital services for the Old Believers' Hospital.] *Gaz Lekarska* 1899; 45:1194–1195.
24. Greenwald A: Report on nursing in Poland. Unpublished manuscript, 1929. JDC File 368.
25. Knapik AJ: Nursing and education in Poland, 1918–1928; a cooperative venture of Polish and American nurses. *J Nurs Hist* 1985; 1:38–55.
26. Waldman MD: Letter to Isabelle Stewart, Mar 24, 1922. TC Microfiche 395.
27. Beard M: Letter to Bernard Flexner, Jan 23, 1929. JDC Folder 367.
28. Stewart IM: Letter to Stella Hawkins, June 7, 1941. TC microfiche 396.
29. Pozarik-Kent S: Personal communication to author, Jan 4, 1989.
30. Rafes J: Jewish doctors and public health in Vilna. Paper presented at the 62nd Annual YIVO Conference, Oct 21, 1990, New York City.
31. Wulman L: OZE. *Encyclopaedia Judaica,* Jerusalem, Keter, 1972, vol 12, p 1538.
32. Wirszup T: Personal communication to author, Jan 4, 1991.
33. Sedlis A: Personal communication to author, Oct 21, 1990.
34. Summary report for the month of January (1922), Joint Distribution Committee, Warsaw. JDC File 359.
35. Summary of meetings of medical committee held from November 14th to November 29th (1922). JDC file 362.
36. Levin M: *Balm in Gilead: The Story of Hadassah.* New York, Schocken Books, 1971, pp 20–21.
37. Stockler RA: *Hospital Nursing in the Holy Land from the Period of the Crusades to the First Five Years of the State of Israel.* Tel Aviv University Department of Nursing, 1986.
38. Rosenberg CE: *The Care of Strangers: The Rise of America's Hospital System.* New York, Basic Books, 1987, pp 111–112.
39. Hyman PE: The modern Jewish family, in Kraemer D (ed): *The Jewish Family: Metaphor and Memory.* New York, Oxford University Press, 1989, pp 179–183.
40. Glanz R: *The Jewish Woman in America: Two Female Immigrant Generations, 1820–1929.* vol 1, New York, KTAV Publishing House, 1974, p 21.
41. Weinberg SS: *The World of our Mothers: The Lives of Jewish Immigrant Women.* Chapel Hill, University of North Carolina Press, 1988, pp 192–199.
42. Cahan, A: *The Rise of David Levinsky: A Novel.* New York, Harper, 1917.
43. Ganzfried S (ed): Laws concerning one who is sick, the doctor and remedies. *Code of Jewish Laws. (Shulchan Aruch)* vol 4, New York, Hebrew Publishing Co., 1927, p 86.
44. Jakobovits I: *Jewish Medical Ethics.* New York, Bloch Publishers, 1959, pp 111–116.
45. Abraham AS: The nurse. *Medical Halacha for Everyone: A Comprehensive Guide to Jewish Medical Law in Sickness and in Health,* ed 2 revised, Jerusalem, New York, Feldheim Publishers, 1984, pp 130–132.
46. Hauer RM: Interview with author, Mar 29, 1990.
47. Poll S: *The Hasidic Community of Williamsburg.* New York, Free Press of Glencoe, 1962, pp 148–149.
48. Rozen DJ: Personal communication to author.
49. First National Conference for Orthodox Nurses, New York City, Sept 17, 1989. Association of Orthodox Jewish Scientists Raphael Society.
50. An Orthodox prognosis. *Jewish Week (NY)* Mar 23, 1990.
51. Bridge DE: *The Rise and Development of the Jewish Hospital in America.* Thesis, Hebrew Union College, Cincinnati, 1985.
52. Levine M: Myra Levine, in Schorr TM and Zimmerman A (eds): *Making Choices, Taking Chances: Nurse Leaders Tell Their Stories.* St Louis, CV Mosby, 1988, pp 215–228.
53. Nowak JB: *The Forty–Seven Hundred: The Story of the Mount Sinai Hospital School of Nursing.* Canaan, NH, Phoenix Publ, 1981.
54. Report of Committee on the Establishment of a Training School for Nurses. Minutes of the Board of Directors, Mount Sinai Hospital, Nov 28, 1880.
55. Ladinski IJ: Report of the Training School Committee, Beth Israel Hospital, Jan 17, 1905.
56. Levenson D: *Montefiore: The Hospital as Social Instrument, 1884–1984.* New York, Farrar, Straus, and Giroux, 1984, p 132.
57. Greenwood MH: Annual report of the Chief Nurse and Superintendent of the Cincinnati Jewish Hospital School of Nursing, Sept 27, 1896. CJH Box 180.
58. Declercq E, Lacroix R: The immigrant midwives of Lawrence: the conflict between law and culture in the early twentieth century, Massachusetts. *Bull Hist Med* 1985; 59:232–246.
59. Kobrin FE: The American midwife controversy: a crisis of professionalization. *Bull Hist Med* 1966; 40:350–363.
60. Wile IS: Immigration and the midwife problem. *Boston Med Surg J* 1912; 167:113–115.
61. Crowell FE: The midwives of New York, in Litoff JB (ed): *The American Midwife Debate: A Sourcebook on Modern Origins.* New York, Greenwood Press, 1985, pp 36–49.
62. Levitan T: *Islands of Compassion: A History of the Jewish Hospitals of New York.* New York, Twayne Publishers, 1964.
63. Pember PY: *A Southern Woman's Story: Life in Confederate Richmond.* Jackson, TN, McKowat-Mercer Press, 1959; republished, edited by Wiley BI, St Simons Island, GA, Mockingbird Books, Inc, 1974.
64. Goodman AV: Adolphus S. Solomons and Clara Barton: a forgotten chapter in the early years of the American Red Cross. *Amer Jew Hist Quart* 1970; 59:331–356.
65. Silverstein NG: Lillian Wald at Henry Street, 1893–1895. *Adv Nursing Sci* 1985; 7(2):1–12.
66. Siegel B: *Lillian Wald of Henry Street.* New York, Macmillan, 1983.
67. Duffus RL: *Lillian Wald: Neighbor and Crusader.* New York, Macmillan, 1938, p 312.
68. Winslow C-EA: *Report of the Committee for the Study of Nursing Education in the United States.* New York, Macmillan, 1923.
69. LeMaster CG: Rose Kaplan: Arkansas's "Lady with the Lamp." (unpublished paper, 1987.) AJA
70. Benson ER: Mathilda Scheuer (1890–1974): a biographical sketch. *J Nurs Hist* 1986; 2: 36–42.
71. Sorin A: *The Prophetic Minority: American Jewish Immigrant Radicals, 1880–1920.* Bloomington IN, Indiana University Press, 1985.
72. Kaufman M (ed): *Dictionary of American Nursing Biography.* Westport CT, Greenwood Press, 1988.
73. Kernodle PB: *The Red Cross in Action.* New York, Harper, 1949, p 208.
74. Falchuk N: We've come a long way. *Nursing: The Jewish Connection.* 1990; 90:1.
75. Leninthal AJ: *First a Dream: The History of Boston's Jewish Hospitals, 1896–1928.* Boston, Beth Israel Hospital in association with the Francis A. Countway Library, 1990, pp 133–134, 166–167, 467–468.

Bernard John Dowling Irwin, MD

Physician, naturalist, and military hero

Allen D. Spiegel, PhD, MPH

With the blood of his forbearers surging in his veins, it may have been genetically ordained that New York educated physician, Bernard John Dowling Irwin, would become a soldier and a hero. Irwin's lineage traced back to Sir William de Irrwyn, armor bearer to Scotland's King Robert Bruce.[1] Reminiscences of Irwin, by his army colleagues, characterized his personality: "Many people accept danger when it is presented; the Irish seek danger for the love of it"[2] and "An Irishman, red head and hot-tempered" (referring to the new post surgeon.)[3] In view of that background, it is not surprising that Irwin displayed courageous actions with New York's Seventh Infantry Regiment battling hostile Apache Indians. In 1861, he was the first to perform a heroic deed for which he would later be awarded the Congressional Medal of Honor. In addition, he was the first to organize an army field tent hospital that could accommodate up to 2,500 sick and wounded immediately before the Civil War battle at Shiloh, Tennessee, in 1862.[4]

A Brief Biography

Irwin was born on June 24, 1830, at Fort Round House, County Roscommon, Ireland, to James and Sabina Maria (Dowling) Irwin. He and his family emigrated from Ireland in 1845 and settled in New York City. Fifteen-year-old Irwin was educated by his father and private tutors as he studied classical and modern languages along with the usual subjects. In adulthood, he became proficient in five languages.[5(p708)] He also attended the University of New York from 1848 to 1849. At age 18, he enlisted in the New York State National Guard as a private. Beginning at Castleton Medical College (Vermont), he enrolled in the first class of the New York Medical College and earned his medical degree in early 1851. After a few interim medical experiences, he became a career army medical officer who served in the US Army for more than 40 years. He began his service at Fort Columbus in New York harbor before being transferred to the Western states. During the Civil War, he was captured at the battle of Richmond, Kentucky, and remained with the Union wounded for about three weeks. Shortly thereafter, he was exchanged and returned to the Union army. With the end of the war, he successively served in various posts in the Western states, at the military academy at West Point, again at posts in the Western states and was then put in charge of the Army medical purveying depot in New York City from 1885 to 1886 (Fig 1).

Two years after joining the then newly organized Association of Military Surgeons in 1891, Irwin was elected First Vice President. He was a member of the Public Health Congress, Auxiliary to the World's Columbian Exposition, 1893; Vice-President of the Pan American Medical Congress, representing the Medical Department of the US Army, 1893; Delegate of the US to the Eleventh International Medical Congress in Rome, 1894; Commander, of the Order of Indian Wars, 1903–1905; and Vice-President of the American White Cross First Aid Society, 1904.

Irwin retired in 1894 and was promoted to Brigadier General in 1904 by an Act of Congress (Fig 2). Although Irwin died at his summer home in Coburg, Canada, on December 15, 1917, his final resting place is at the West Point military academy in New York. Buried beside him is his son, Brigadier General George LeRoy Irwin, a career artillery army officer from his West Point graduation in 1889 to his death on February 19, 1931. Irwin General Hospital in Fort Riley, Kentucky, is a memorial to Bernard J. D. Irwin, "the fighting doctor"[6]

Irwin Attends the New York Medical College

There were two New York Medical Colleges. Irwin attended the New York Medical College that existed from 1850 to 1864. Today's New York Medical College came into existence in 1936.

This earlier New York Medical College was chartered on April 8, 1850, and opened for its first course of lectures on October 16, 1850. Irwin paid a matriculation fee of $5.00, a fee of $105.00 for the full course of lectures and a demonstrator's fee of $5.00. Students needing room and board in the area paid $3.00 to $4.00 a week. There was also a fee of $30.00 for the final examination. The first commencement took place in March 1851 and the college finally closed in 1864.[7]

In his history of medicine in New York, Walsh[8] said that this earlier New York Medical College "represents a veritable oasis in the desert of low grade medical education in America at this time."

The other New York Medical College was chartered on April 12, 1860, as the Homeopathic Medical College of the State of New York in New York City. About five months later, this medical college initiated its first lecture courses.[9] Poet, journalist, and civic leader, William Cullen Bryant was elected President of the Trustees and served from 1862 to 1872.[10] After several name changes, the college was renamed the New York Medical College in 1936 and exists today in Valhalla, New York.[11]

Medical Graduation in 1851

At Irwin's graduation on February 28, 1951, the prominent lawyer, George Wood, President of the Trustees, bestowed the degree of Doctor of Medicine on the 13 graduates. Professor Green delivered the valedictory address on "The Prospective Progress of Medicine in

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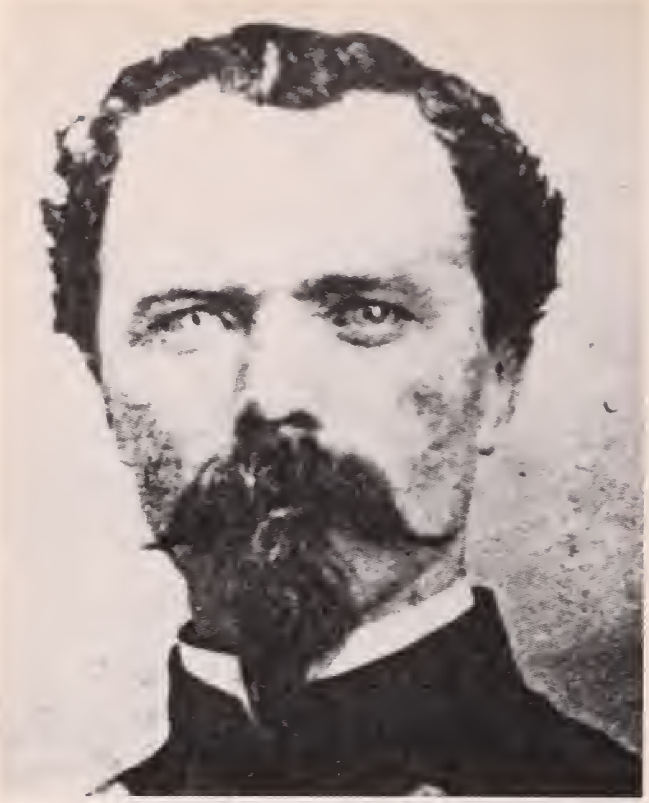

FIGURE 1. Dr Bernard J. D. Irwin as a young Army officer (courtesy of the Prints and Photo Collection, National Library of Medicine).

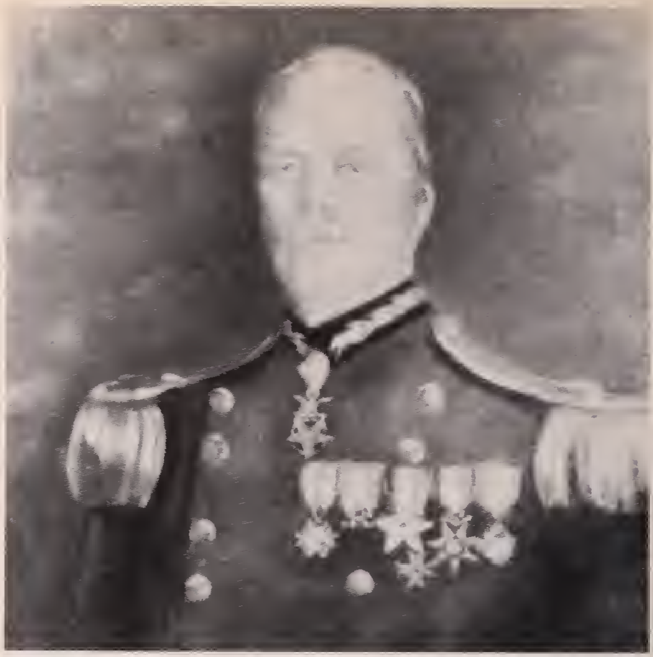

FIGURE 2. Brigadier General Bernard J. D. Irwin as depicted in an oil painting by his daughter Amy (Mrs Robert Rutherford McCormack). General Irwin is shown with the Congressional Medal of Honor at his neck, other medals (the Military Order of the Loyal Legion, the Order of Indians Wars and the Medal of the Association of Military Surgeons), and three badges pertaining to the Civil War (courtesy, Prints and Photo Collection, National Library of Medicine).

America."[12] Comparing American and European medicine, Green remarked: "The healing art will make progress among us, because we are characterized by a fondness for novelty and experiments." Green added: "But let us never forget, that the most glorious, though least gainful triumphs of our art, should be to prevent, not to cure disease." There was sustained and rousing applause after Green welcomed the graduates into a noble profession and declared with obvious sentiment: "You are the firstlings of our hands and hearts." After listening to Green's speech, a *New York Daily Tribune* reporter commented: "It was well received and exhibited a perfect knowledge of the subject."[13]

To celebrate their graduation, Irwin and his friends paid $5.00 for a private box at Niblo's Gardens. They saw a new vaudeville *"LaMaitresse Des Langues"* and the grand ballet of *"Catarina Ou La Reine Des Bandits."*

At a meeting of the graduates and students of the New York Medical College on March 11, 1851, Irwin was selected to chair a committee. He wrote to Dr Green "for the purpose of soliciting for publication a copy of your eloquent and patriotic address delivered at the Annual Commencement."[12]

At the time of Irwin's graduation, the medical column of the newspapers advertised Dr Delany's cures for private diseases in the first stage; a no cure-no pay offer by Dr Corbitt; Dr Glover offered to cure all those difficult and protracted cases that escaped from medical pretenders; Dr T. L. Nichols and Mrs Gove Nichols, Water Cure Physicians, offered consultations from 12 PM to 4 PM; Dr J. S. Fancher described his Grecian Fancheronian Drops to cure dyspepsia, chills and fever, jaundice, diarrhea, and cholera positively; and Horehound Cough Syrup, Bryan's Pulmonic Wafers, Vegetable Pain Killer, and Mrs Burns Antique Ointment all proclaimed their unique benefits.

Did Irwin receive a quality medical education? A *New York Times*[14] editorial lauded the New York Medical College: "The faculty are all young men. There are no old fogies among them. They are men of wide reputation, full of zeal and determined not to be outdone in nothing that can facilitate the acquirement of a thorough medical education."

AN ARMY MEDICAL OFFICER: DUTIES AND EXAMS

Irwin's familial heritage may have subliminally guided him into medical service in the US Army. There is no clear indication of why he chose the Army rather than private practice.

In an April 10, 1849, letter to the AMA Committee on Medical Education, Acting Surgeon General H. L. Heiskell[15] detailed the duties and examinations for admission into the medical staff of the US Army. Regulations stated that the medical officer is required to investigate the physical agents that affect the health of the troops, to make reports on the medical topography of his station and to aid in the selection of military positions. Regarding military hygiene, he is to express his views in respect to the diet, clothing, watering, and exercise of the troops. "From necessity, the Army medical officer is a general practitioner in the most extended sense of the term. . . . From location, he often must rely entirely upon his own resources. Thus he is deprived of the advantage of seeking aid and friendly counsel from professional brethren."[15]

Before Irwin's appointment, he had to pass an examination by the Medical Board of Examiners. Before this, he had to prepare, in writing, a brief extemporaneous description of the causes, symptoms, pathology, and treatment of a disease assigned by the Board. In addition, the applicant had to prepare one or more prescriptions appropriate to the case, written out in the required form for the apothecary. Examiners raised questions on subjects usually taught in medical schools, on literary and scientific achievements, and looked for grammatical knowledge of the English language and knowledge of Latin and natural philosophy (or physics), since those were deemed important to those entering on the study of a liberal profession. Furthermore, the examiners evaluated the applicant's natural endowments, his general professional intelligence, his exact knowledge, his practical ability, and his strength of judgment.

At this time, a medical career in the army seemed to be a highly competitive position. A report from the US Surgeon General noted that only five of 22 applicants qualified for the Army medical corps.[16] Reiterating criticisms of medical education at the time, the Army disclosed the most striking causes of failure: insufficient preparatory education, a hurried course of professional pupilage, and a want of proficiency in practical anatomy, pathology, and clinical medicine.

Apparently, Irwin had no trouble meeting the comparatively stringent US Army requirements. He passed the entrance examination on November 4, 1853, and was number 15 on the list. While waiting for a vacancy, he was a resident physician and house surgeon at the Emigrant's Hospital from 1853 to 1855. From September 1855 to November 1856, he was an Acting (contract) Assistant Surgeon until he accepted an Army commission as an Assistant Surgeon on November 7, 1856.

Medal of Honor Bravery

Reports about the actions for which Irwin earned the Congressional Medal of Honor are conflicting. This occurred because the participants often wrote their reports many years later. Irwin himself did not write his version until 1893, some 32 years later. Yet, there is some consistency.

After brief duty in several New Mexico locations, First Lt Irwin was assigned as Post Surgeon to Fort Buchanan, Arizona in December, 1857. During the latter part of 1860, hostile Indians raided a Sonoita Creek frontier ranch where John Ward lived with his mistress, Jesusa Martinez, and her son Felix.[17] These Indians stole cattle and kidnapped the young boy. Ward traveled the eleven miles to Fort Buchanan to ask for help in recovering the cattle and his foster son. He told the officers that he believed that the Chiricahua Apaches under Chief Cochise were responsible. Colonel Pitcairn Morrison pledged to help, but bad weather and pressing business prevented direct action until February 1861. With Second Lt George N. Bascom in command, about 60 men from the Seventh Infantry Regiment were sent to rescue the boy and to recover the cattle. Bascom found Cochise and demanded the return of the boy and the cattle. Cochise explained that he knew nothing of the raid and offered to help get the boy and the cattle back.

Unyielding, Bascom called Cochise a liar and attempted to hold Cochise and his party as hostages. Acting with lightning speed, Cochise slit the tent with his knife and boldly made a dash for freedom. Although about 50 bullets were fired at him, Cochise escaped and several soldiers were wounded in the melee. However, Bascom did hold three Indians captive, family members of Cochise. Shortly, Bascom was surrounded by hundreds of Apaches. Cut off from Fort Buchanan, Bascom sent a messenger to the Fort under cover of darkness. On hearing from the messenger, Colonel Morrison had few soldiers available and asked Fort Breckenbridge to send support troops. Knowing that Bascom had wounded men, Irwin volunteered to command the relief party. Irwin led fourteen picked infantrymen, mounted on mules, out into a blinding snowstorm on February 13, 1861. At Sulphur Springs Valley, Irwin encountered a band of Coyotero Indians returning from a cattle raid. Irwin led the hard chase of six or seven miles, retrieved 30 ponies and 40 cattle and captured three Indians. Finally, after 100 miles, Irwin entered the long, winding canyon leading to Apache Pass on February 14. At the entrance to the canyon, they found the naked remains of eight people tied to the wheels of their wagon train. These victims had been tortured and burned alive. Fortunately for Irwin's troops, the Apaches sighted the relief column from Fort Breckenbridge under Lt Isaiah N. Moore. Fearing an attack, the Apaches retreated, allowing Irwin to unite with Bascom's troops. Irwin tended to the wounded as troops spent several days finding Cochise's abandoned village and destroying it. On the march back to Forts Buchanan and Breckenbridge, Bascom and Irwin found the bodies of six men who had been tortured and killed by Apaches.

Although a historian branded Irwin and Lt Moore as "blood thirsty,"[18] Irwin's own words[19] may reflect contemporary societal attitudes as well as his own personality:

> I suggested their (the captured Indians) summary execution, man for man. On Bascom expressing reluctance to resort to the extreme measure, I urged my right to dispose of the lives of the prisoners captured by me. After which, he acceded to the retaliatory proposition, and agreed that those prisoners and three of the hostages taken by him, should be bought there and executed. After full and deliberate consideration, it was accordingly done the next day as the troops marched by that point on their return to their Forts. The punishment was an extreme mode of reprisal, but was demanded and justified by the persistent acts of treachery and the atrocious cruelties perpetrated by the most cowardly and intractable tribe of savages infesting the territory.

In a March 17, 1861 message from Departmental Headquarters at Santa Fe to Fort Buchanan, the commanding officer was instructed to commend Irwin and Bascom:[20]

> The Department Commander directs that you will publicly express to Dr Irwin, US Army, and to Lt Bascom, 7th US Infantry, his approbation of the excellent conduct of those officers, and the troops under their command in the operations against Apache Indians during the last month. He emphatically approves of Lt Bascom's decided action in executing the Indian warriors, after the atrocious murders which had been committed by the tribe.

Angered by the hangings, Cochise initiated a bloody 12-year Indian war. Within 60 days, 150 white men traveling the Overland Trail were killed.[21] This war may have begun with or without the Apache Pass incident; there were multiple factors.[22] However, Terrell[21] is caustic and direct about the responsibility for the war being "the stupidity, pigheadedness, and uncontrollable temper of a young shavetail (Bascom), fresh out of West Point." To the point, a historical consensus agrees that Cochise almost certainly did not steal the cattle and kidnap the boy and the Coyoteros most likely were the raiders.[5,23]

Ward's foster son was never returned. He changed his name from Felix to Mickey Free and became a scout for General George Crook in the 1870s and 1880s. Apaches called him "the coyote whose kidnapping brought war to the Chiricahuas."[24]

Thirty-three years later, on January 24, 1894, Irwin was belatedly awarded the Medal of Honor. His citation reads:

> Voluntarily took command of troops and attacked and defeated hostile Indians he met on the way. Surgeon Irwin volunteered to go to the rescue of Second Lt George N. Bascom, 7th Infantry, who with 60 men was trapped by Chiricahua Apaches under Cochise. Irwin and 14 men, not having horses, began the 100-mile march riding mules. After fighting and capturing Indians, recovering stolen horses and cattle, he reached Bascom's column and helped break his siege.[25]

Irwin's heroism occurred before the Civil War began and even before the Congressional Medal of Honor was created. On December 21, 1861, President Abraham Lincoln signed legislation authorizing a Navy medal of honor. A similar bill, on July 12, 1862, created the Army medal of honor for non-commissioned officers and privates. On March 3, 1863, a bill passed making officers eligible. Medals were first awarded on March 25, 1863, by Secretary of War Edwin M. Stanton to six men who participated in the brazen Andrews locomotive raid in enemy territory.[26]

Paradoxically, Irwin earned the Medal of Honor for his heroism. Yet, his intransigence about summarily hanging the captured Indians could have possibly precipitated Cochise's bloody 12-year war with the Americans.[5]

IRWIN'S BELONGINGS DESTROYED BY FIRE

On June 11, 1861, Irwin was transferred from Fort Buchanan to Fort Fillmore, New Mexico. He turned over his baggage and personal effects to the transportation officer for forwarding. En route, on August 3, 1861, the commanding officer, Capt I. N. Moore, received a message from Fort Craig, New Mexico, that the troops at Fort Fillmore had surrendered the post to Texan insurgents. Capt Moore was warned of an impending ambush by the Texan insurgents who were only one day's march away from his position. In preparation for a five- or six-day march over rugged mountain trails to Fort Craig, Capt Moore did not want to be encumbered by baggage and did not want the material to fall into enemy hands. Soldiers were ordered to put the torch to all public and private property, including Irwin's baggage and household effects.

In the 1890s, Irwin instituted a law suit to recover for his losses.[27] His demand was heard by the US Court of Claims. However, the Court declared that the claim was not within their jurisdiction because the events occurred in the war for the suppression of the rebellion.

If a man can be known by the books he reads, can the same be said for the belongings he accumulates? In his claim, Irwin listed $2,286.00 worth of personal items (Table I).

What can be learned from the listing of Irwin's belongings? Army medical officers were usually the best educated men in the service. Although Irwin's life was busy and exciting, there were occasional lulls. In addition to his medical pursuits, he became a naturalist and collector with far-ranging interests including anthropology, ethnology, entomology, herpetology, metallurgy, and ornithology.

Actually, the US Weather Bureau owes its origin to the Army Medical Department. Beginning in 1814, the Surgeon General directed Army medical officers "to keep a diary of the weather, together with an account of the medical topography . . . as the influence of weather and climate upon diseases, especially epidemics, is perfectly well known."[28] Army medical officers cooperated with scientists and institutions absorbed in obtaining specimens of animals, plants, and minerals. Scientists requested data on the various Indian tribal folkways and artifacts such as clothing, hunting equipment, warring weapons, and household implements.

Irwin recalled his "frequent correspondence with Professor Spencer F. Biard."[29] Trained and encourage by Baird, the Smithsonian Institution's Assistant Secretary, Irwin made natural history more than just a hobby. Irwin was among the many medical officers who kept forwarding a constant stream of specimens. Furthermore, Irwin directed other Army personnel to collect specimens. Writing about Maj Charles E. Bendire, Irwin reminisced that "it was I who initiated in him the taste for natural history which he cultivated with so much zeal and advantage as a naturalist."[29] In turn, Bendire wrote the following about Irwin:

> . . . Dr Irwine [sic] is also an excellent man in every way, and in the early days of Arizona in 1857 and 1858 he used to be quite interested in making natural history collections and the Smithsonian is indebted to him for a great many nice things from the Northern portion of the Territory. I am indebted to the doctor for my life and never would have pulled through if it had not been for his unremitting attention to me while at Fort Buchanan.[30]

Many of Irwin's letters are still in the Smithsonian's archives. In a letter to another Army medical officer naturalist, Irwin remembered "a collection of reptiles . . . made by me for the Smithsonian Institute at Fort Buchanan and vicinity in 1858–1860."[28] There are still specimens credited to Irwin in the Smithsonian's anthropology and ornithology collections.[31] Irwin was also well traveled, having made three trips to Europe in five years: 1851, 1854, and 1855.

FIRST ARMY FIELD TENT HOSPITAL

By 1862, with the Civil War in progress, Capt Irwin was Medical Inspector, Army of the Ohio, Fourth Division-Nelson's. Up to this time, there was no ambulance service to speak of and no efficient methods for transporting the wounded. Barely adequate medical personnel administered first aid at the front lines and base hospitals were far in the rear. There was nothing between these two.[28]

Immediately before the battle of Shiloh, Tennessee, on April 6 and 7, 1862, Irwin devised the first tent field hospital for the treatment of the wounded during a war (Fig 3). However, Laffin[32] comments that the Prussians in Europe and the British in India used tent hospitals in 1848. Still, Irwin's was possibly the largest tent field hospital. Actually, his creation was a precursor to contemporary wartime Mobile Army Surgical Hospital (MASH) units. Before Irwin's innovative idea, the wounded were treated in the barns, stables, churches, and any accommodations that might be found in the immediate area of a battle.

Displaying unusual resourcefulness, Irwin[33,34] described how the tent field hospital evolved:

> . . . it became necessary to have one of the operating stations moved forward to a deserted farmhouse, situated on an open piece of level, unbroken ground. The presence of a spring of cool, potable water and the nearness of the building to a small branch of a creek were advantages that were promptly recognized. But a short time had elapsed ere these valuable desiderata were utilized in affording shelter and other comforts. A large number of wounded of our own division and those of the contending forces had been disabled on that part of the battlefield during the conflict of the preceding day and left without assistance some twenty-four to thirty-six hours. Proximity of the operating station to the recaptured camp of a division of our troops, who had been driven therefrom and partly recaptured by the enemy during the preceding day, suggested the utilization of the abandoned tents for the

TABLE I. Irwin's Personal Property and Baggage Which Were Destroyed by Fire in June, 1861

Item	Cost
2 uniform coats, $30, $36	$ 66.00
1 cloth riding jacket, gold-lace embroidery	35.00
1 cloth riding jacket, plain	25.00
1 black cloth frock coat	25.00
1 black cloth dress coat	42.00
1 military overcoat	50.00
1 beaver cloth overcoat	30.00
4 white Marseilles vests	24.00
3 velvet vests	30.00
1 leopardskin vest	20.00
1 large fur cape, otter skin	15.00
15 shirts, $3 each	45.00
2 dozen half hose, $6 per dozen	12.00
2 pair of blue cloth uniform pants at $12 each	24.00
2 pair of plain cloth pants, one black, one blue	18.00
6 pair of white duck pants, at $6 each	36.00
3 linen coats	30.00
2 linen jackets	12.00
3 pair of woolen blankets, at $10 per pair	30.00
6 sheets at $2 each	12.00
1 full-dress military hat with trimmings	19.00
1 fatigue cap	5.00
1 Panama straw hat	20.00
1 felt hat	4.00
1 superfine Navajo blanket	40.00
1 superfine Navajo blanket	35.00
3 small Navajo blankets	15.00
2 pair fur gloves	12.00
2 pair buckskin gauntlets, $2.50 per pair	5.00
Silk and linen handkerchiefs	12.00
2 dozen shirt collars, neckties, silk oravats	10.00
2 pair passants	10.00
1 pair shoulder knots	8.00
6 pairs of boots and shoes	50.00
1 waterproof cape	10.00
1 hunting suit, 2 pairs of pants	12.00
Hunting shirts	16.00
Hunting knife	12.00
1 leather gun case, powder flask, shot pouch, ammunition	25.00
1 bearskin robe	20.00
1 leopardskin robe	30.00
1 hair mattress	20.00
Towels	6.00
1 large sole-leather trunk	40.00
1 valise	10.00
1 carpet	30.00
1 rug	10.00
Window curtains	10.00
Underclothing	24.00
Set of kitchen furniture	50.00
Dining-room furniture	50.00
Sundry engravings, two paintings on cooper, daguerreotypes	50.00
1 portable camp table, oak	10.00
4 portable camp chairs	8.00
Commissary stores	10.00
1 writing desk and case	15.00
About 100 volumes select medical and standard books	200.00
Cabinet collection of Navajo, Apache, Pino, Papago, Zuni, Moigi, and other Indian warlike weapons, bows, quivers, arrows, lances, clubs, etc.	100.00
Cabinet collection of animals, birds, reptiles, insects, and Indian crania, pertaining to Arizona and New Mexico, collected at much labor and expense, during nearly seven years' service there	100.00
Manuscript notes, on the influence of the climate of Arizona on gunshot wounds, illustrated by 43 cases	100.00
Cabinet of minerals, precious and other metals, mines of Arizona, consisting of about three hundred choice specimens collected with great care during a residence of nearly four years from the various mines of that Territory, including many rare varieties of gold, silver, copper, lead, zinc, antimony, bismuth, arsenic, iron, and other metals in various form	250.00
Manuscript notes of three visits to Europe in 1851, 1854, 1855, private and miscellaneous papers, family relics, souvenirs, etc.	100.00
1 mess chest, with complete outfit for three persons	50.00
Total	$2,286.00

Source: US House of Representatives.[27]

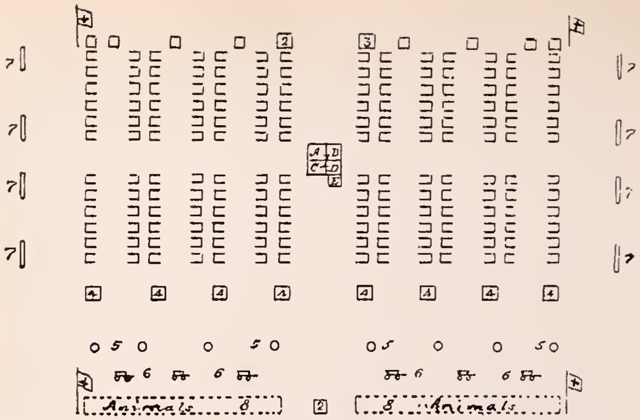

FIGURE 3. Irwin's Plan of the First Tent Field Hospital, Shiloh, TN, April, 1862 Executive Building: (A) Office; (B) Operating Room; (C) Dispensary; (D) Officers Dining Room; (E) Kitchen; (2) Guard; (3) Stores; (4) Officers and Sick Officers; (5) Teamsters; (6) Ambulances and Wagons; (7) Latrines; and (8) Picket Lines (Irwin BJD[34]).

> care of the wounded. As soon as the battle ceased, the regimental hospital tents, commissary tents, and the wall tents were accordingly taken possession of. Within a short space of time, the tents were removed to and pitched in regular camp order on the level ground by which the house was surrounded. That building afforded an operating room, dispensary, office, kitchen, and dining-room for the officers attached to the establishment.
>
> Long into the night the ambulances continued to bring in the wounded. After receiving the necessary professional attendance, the men were made as comfortable as possible by being supplied with an abundance of warm food, good bedding and shelter against the inclement weather. Next day, the camp was enlarged and systematically arranged and the tents increased so as to conveniently accommodate some three hundred patients. All bedsteads, cots, bedsprings, cooking and necessary utensils, hay and straw found in the recaptured camp were appropriated for the use of the hospital. On the evening of the eight, the Division Medical Purveyor reached us bringing our medical supplies and hospital stores. By that time the hospital had already assumed the proportion and discipline of a well-regulated regimental camp, arranged with a view to meet the special wants of those for whom it had been established. Some mattresses had been secured, bed sacks filled with hay or straw were placed on cots or improvised bunks, so that every patient was provided with comfortable sleeping accommodations. A suitable number of attendants of each class were detailed for duty in the establishment, which was placed under the charge of a competent executive medical officer, aided by the necessary number of professional assistants. Invalids were promptly and regularly supplied with abundance of properly prepared, nutritious food. A fair supply of extra diet and delicacies were served to the inmates who needed special attention. It soon became manifest that the wounded who were accommodated in that improvised field hospital were better provided for and more comfortable in every particular than those who were hurried aboard the crowded transports.

Irwin concluded: "One of the most valuable lessons . . . was the demonstration of the inestimable value of the tent as a hospital, and as a reliable substitute for the pick-up structures, obtained by chance, into which the sick and wounded of armies in the field were up to that time huddled."

Union forces suffered more than 13,000 casualties during the battle of Shiloh. Assistant Medical Director Robert Murray issued a commendation:

> Assistant Surgeon Irwin deserves great credit for his admirable management on the field as well as for his promptness and professional skill in the care of the wounded . . . he succeeded in organizing and putting in good working order a hospital for three hundred patients. . . . These were so well taken care of in every way that I designated them as the last to send on board of the boats.

The importance of Irwin's field tent hospital was immediately recognized. Armies of many nations soon adopted this type of modern field hospital. In recognition of his service, Irwin was made a Companion of the Military Order Loyal Legion, US, New York Commandery in December 1867. Today, a memorial tablet on the Battlefield of Shiloh honors Irwin. With a red cross in each corner, the tablet says: "The first tent field hospital ever used for the treatment of wounded on the battlefield, 'the greatest boon in war,' was established here, April 7th, 1986, by Capt B. J. D. Irwin, Assistant Surgeon, USA, Medical Inspector 4th Division, Army of the Ohio."

Chief Medical Officer, West Point

In October 1873, Irwin became the Chief Medical Officer at the US Military Academy at West Point. He conducted physical examinations of the candidates for admission, handled sick call among both civilians and the military, and paid particular attention to public health, sanitation, and the environment.

During Irwin's tenure,[35] a very complete medical evaluation of West Point took place. Irwin was deeply involved in the research for and submission of a Special Sanitary Report in 1874. Recommendations advised the following: construction of new water closets and urinals, screened from view and connected to new sewers; removal of families from quarters in the guard house, discontinuation of the use of dark cells in the basement and abandonment of a ball and chain for prisoners; improvement of the heating system in the cadet barracks; substitution of Java coffee for the type used in the cadet mess; use of boiled instead of cold milk for coffee; addition of much more fresh milk to cadet menus; the rapid construction of a new cadet hospital; and an appropriation of $10,000 to complete a drainage and sewage system for the officers' quarters.

At the same time that Irwin reported a diphtheria epidemic in New York City in January 1875, he also observed a severe form of rampant tonsillitis at West Point. Assistant Surgeon A. C. Gerard, a staff physician, thought the tonsillitis might be linked to the low temperatures and the snow on the ground. Gerard had another theory: ". . . it (tonsillitis) is a modified form of the epidemic diphtheria that has been ravaging the inhabitants of New York City wafted to us by the atmosphere and modified by our pure country air and superior hygienic conditions." A large number of cadets were treated without interference with their classes.

In a space of 48 hours in August of 1875, 65 out of the 205 cadets were afflicted with nausea, fainting, vomiting, gripping pain in the bowels, and violent purging. Irwin and his medical staff diagnosed the illness as "cholera morbus," a term used for any acute gastroenteritis. An investigation attributed the outbreak to a breakfast serving of fish hash prepared with dried cod that was probably contaminated. Following Irwin's advice, the Superintendent prohibited the serving of fish, except for salt mackerel, at cadet meals between June 15 and September 15. Irwin also stopped the commissary from issuing, at one time, a ten-day supply of meat to married solders.

In 1876, a scarlet fever scare developed with six cadets falling ill. A specialist, Assistant Surgeon S. Q. Robinson, was called to West Point to take charge of the outbreak.

Later that year, Irwin tended to a measles epidemic among civilians with a total of 125 cases, 95 occurring during November and December.

After an analysis of the statistics from 1864 to 1873, Irwin found that 20% (3,538) of the 17,442 cases of disease originated "in miasmatic poison, most of which could have been prevented by a proper system of hygienic police. Faulty drainage on the post has been the most potent and constant factor in producing disease, especially those of miasmatic and malarial type." Irwin recommended the early completion of a thorough and perfect system of drainage.

Irwin and the Academy's Board of Visitors continued to urge strongly the completion of the cadet hospital. Each cadet was regularly charged a monthly average of 61¢ to augment the hospital fund. On August 28, 1878, Irwin was relieved from duty at the Military Academy and ordered to report to the Surgeon General.

PROFESSIONAL WRITINGS

Eight of Irwin's articles appeared in the *American Journal of the Medical Sciences.* Traveling 100 miles from Fort Buchanan to treat a patient, Irwin described an amputation of the shoulder joint on April 10, 1859. In graphic detail, he explained his procedure for this serious surgical intervention. He concluded: "On the 24th day after the operation he (the patient) was walking about, and in less than six weeks he started for the Eastern States, restored to perfect health."[36]

Still at Fort Buchanan in September 1860, Irwin made a "proper investigation" of a herb, *Euphorbia prostrata.* This herb was labeled "Gollindrinera" by the Mexican population and used as an antidote to rattlesnake venom. For several months, Irwin experimented on many dogs with satisfactory results. After minutely detailing the herb, the source of its medicinal properties, its physiological effects, and his inability to explain the modus operandi, he described two experiments in detail. In summary, Irwin stated that the herb "never fails to produce a sure and speedy cure." Furthermore, he recommended the herb as being more advantageous than the standard "Bibron's Antidote" then in use.[37]

During the incident at Apache Pass, one of the Indian hostages was bayoneted and transfixed to the ground. When Irwin arrived, he took care of the Indian. His treatment of the puncture wound and recovery of the patient was related in *American Lost Times*[38] the next year.

At Fort Buchanan in May 1861, Irwin treated a soldier who had a gunshot wound of the arm. Since the humerus was splintered, he amputated at the shoulder joint. About two months after being shot, the patient died. However, during the surgery, Irwin pricked the end of his left index finger and apparently was poisoned by absorption of some putrid matter. Suffering pain and throbbing, he administered medications, constant irrigation, and sought consultation with a physician friend who had arrived from Tucson, Arizona. After going through six days of intense pain, intermittent fever, and wild delirium, his finger was incised to relieve the pain. Noting the destruction of muscular tissue at the first metacarpal, Irwin commented that the "condition of the first phalanx is irremediable by any other than surgical means."[39]

In 1874, Irwin described a case of osteophytic inflammation of the right radius of a young woman. She was going East to have her arm amputated, but Irwin's examination convinced him that such an extreme measure was unnecessary. With impressive surgical skill, he did a resection and was able to preserve a useful hand. Citing a few earlier examples, he remarked that the "exsection of any extensive part of the lower extremity of the radius does not appear to have been performed except in the instances already mentioned."[40]

Irwin recounted an 1863 case dealing with the immobility of the tempero-maxillary articulation after a gunshot wound. This patient was unable to separate his jaws for more than a year. Using a "unique and entirely original" approach combining wooden wedges and leather straps, Irwin relieved the problem by novel mechanical means. Irwin was able to pry open the man's jaws without harming him.[41]

Other professional articles dealt with extensive laceration of the hand from an explosion,[42] three cases of penetrating gunshot wounds of the thorax with perforation of the lungs,[43] concussion of the brain,[44] fracture of the sternum with dislocation of the fragments,[45] and his introduction of tent field hospitals in war.[34]

At West Point, Irwin published a seven page pamphlet: *Catalogue of the Library Pertaining to the Cadet's Hospital* United States Military Academy, West Point, NY, 1877, arranged by B. J. D. Irwin, MD, Surgeon and Brevet Colonel, US Army, Post Surgeon. A total of 524 volumes were listed alphabetically by author including 179 books, 259 volumes of periodicals, and 86 miscellaneous volumes.[46]

In 1857, Irwin discovered the remains of a meteorite in an alley in Tucson, Arizona. He presented the "Irwin Meteorite" to the Smithsonian Institution[29] and published a seven page booklet on the history of the great "Tucson Meteorite."[47]

Written in 1887, but not published until 1928, Irwin[48] recalled his heroic encounter with Cochise and the Chiricahua Apaches in *The Infantry Journal.* This article was Irwin's response to a newspaper letter to the editor that he felt did not do justice to the severity of the Apache atrocities.

In a political remonstration in 1890, Irwin sent a seven page letter to the Military Committee of the US Senate and to the Adjutant General of the Army. He protested the appointment of Col Edward P. Vollum as Chief Medical Purveyor for the Army. He contended that as he was Vollum's senior, the appointment, by law, should have gone by seniority and not by selection.[49] However, Vollum was appointed in spite of Irwin's efforts.

EPILOGUE

Bernard John Dowling Irwin combined the adventure and travel of a military life, while making significant medical and scientific contributions. This New York educated physician applied his intellectual abilities in the variegated locations where he served with undiluted gusto. His special achievements included his winning the Congressional Medal of Honor, his creation of the first army tent field hospital during wartime, his numerous contributions as a naturalist and as a medical practitioner. In Surgeon General Ireland's[50] Presidential Address to the Association of Military Surgeons in 1927, he referred to General Irwin: "Irwin was a very distinguished man . . . a man of great energy and character, and he saw to it that the Medical Department received whatever was coming to it in his department. He used to say: 'The doctor first and last is a medical officer.' "

Acknowledgments. Gracious help from the following individuals filled in the gaps and supplied accurate material for this article. Philip Angle, William Deiss, and James Krakker of the Smithsonian Institution, Washington, DC; Ellen F. Carr, Director of Public Relations, New York Medical College, Valhalla, NY; US Senator Alfonse D'Amato, Washington, DC; Donald H. Dederick, Interlibrary Loan Librarian, State University of NY Health Science Center at Brooklyn, NY; Carol Downey, Reference Librarian, Arizona Department of Library, Archives, and Public Records, Phoenix, AZ; Mary C. Gillett, PHD, Historian. Conventional War Studies Branch, the Center of Military History, Department of the Army; Brian Gray, Ranger, Shiloh National Military Park; Bonnie J. Greer, Archivist, Arizona Historical Society, Flagstaff, AZ; Andrea R. Hamburger, Deputy, Academy Relations Division, US Military Academy, West Point, NY; John Hendrickson, Alexandria, VA; John F. Jansson, Director, Editorial Information, *Chicago Tribune*, Chicago, IL; Brig Gen Charles W. McClain, Jr., US Army Chief of Public Affairs, Washington, DC; Edward F. Murphy, President, Medal of Honor Historical Society, Mesa, AZ; BJ Shelton, Arizona Historical Society, Tucson, AZ; Fred E. Schwartz, Executive Director, Chicago Medical Society, Chicago, IL; Gerard F. White, Director, Congressional Medal of Honor Society, New York, NY.

REFERENCES

1. Thompson JH: First medal of honor won by Chicagoan. *Chicago Tribune* March 8, 1959, p 14.
2. Crimmins ML: Recollections of the old medical officers: Brigadier General BJD Irwin. *Mil Surg* 1928; 62:361–365.
3. Hamlin PG: *Making of a Solder*. Richmond VA, Whillet and Shepperson, 1935, p 87.
4. Deaths. *JAMA* 1917; 69:2136.
5. Thrapp DL: *Encyclopedia of Frontier Biography*. Glendale, CA, Arthur H. Clark Co., 1988, p 707,708.
6. *Who Was Who in America, Vol 1. 1897–1942*. Chicago, IL, AN Marquis Co., 1942, p 620.
7. Davis EH: *A Short Sketch of the New York Medical College*. New York, Gillis Press, 1883, p 7.
8. Walsh JJ: *History of Medicine in New York, Vol 2*. New York, National Americana Society, Inc., 1919, p 375.
9. Carr E: Public Information Director, New York Medical College. Personal phone conversation: January 27, 1992. Information came from Wershub LP: *One Hundred Years of Medical Progress: A History of the New York Medical College*., Springfield, IL, Charles C. Thomas, 1967.
10. Packard FR: *History of Medicine in the US, Vol 2*. New York, Paul B. Hoeber, Inc., 1932, p 1219.
11. Kaufman M: Homeopathy in America: the rise and fall and persistence of a medical heresy, in Gevitz N (ed): *Other Healers*, Baltimore, MD, Johns Hopkins Press, 1988, p 113.
12. Green H: *The Prospective Progress of Medicine in America: A Valedictory Address Delivered Before the Graduating Class at the First Annual Commencement of the New York Medical College*. New York, Baker, Goodwin and Company, Printers, 1851.
13. Commencement. *New York Daily Tribune*, March 3, 1851, p 4.
14. Medical Schools in New York. *New York Daily Times*, Oct. 5, 1852, p 4.
15. Heiskell HL: United States Army. *Transactions AMA* 1849; 2:312–315.
16. Kaufman M: *American Medical Education: The Formative Years 1765–1910*. Westport, CT, Greenwood Press, 1976, p 52.
17. Haley JL: *Apaches: A History and Culture Portrait*. New York, Doubleday and Co., 1981.
18. Adams AB: *Geronimo*. New York, GP Putnam's Sons, 1971, p 18.
19. Irwin BJD: *Memorandum to Headquarters Department of the Missouri*. Chicago, IL, April 13, 1893.
20. Ashburn PM: *A Medical History of the Medical Department of the United States Army*. Boston, Houghton Mifflin Co., 1929, p 64.
21. Terrell JU: *Apache Chronicle*. New York, World Pub., 1972, p 219–221.
22. Sacks BH: New evidence of the Bascom affair. *Arizona and the West* 1962; 4:261–278.
23. Utley RM: The Bascom affair: a reconstruction. *Arizona and the West* 1961; 3:59–68.
24. DeBo A: *Geronimo. The Man, His Time, His Place*. Normal, University of Oklahoma Press, 1976, p 176.
25. US Senate Committee on Veterans' Affairs Print No. 3: Medal of Honor Recipients 1863–1978. Washington, DC, Government Printing Office, February 14, 1979, p 292.
26. US Army, Public Information Division: The Medal of Honor of the US Army. Washington, D.C., Government Printing Office, 1948, p 5.
27. US House of Representatives, 52nd Congress 2nd Session, Mis. Doc. No. 18: Report from the US Court of Claims, Washington, DC Government Printing Office, November 7, 1892.
28. Hume EE: *Victories of Army Medicine*. Phil, JB Lippincott Co., 1943.
29. Irwin BJD: A biographical and autobiographical letter to Major Mearns, September 21, 1901. *The Auk* 1902; 19:116–117.
30. Bendire CE: Letter to Edgar A. Mearns, March 7, 1895. Washington, DC, Library of Congress Manuscript Division.
31. Deiss WA: Associate Archivist, Smithsonian Institution. Personal communication May 8, 1992.
32. Laffin J: *Surgeons in the Field*. London, JM Dent & Sons, Ltd., 1970
33. Pilcher JE: The first tent field hospital. *J Assn Milt Surg* 1905; 17:63–66.
34. Irwin BJD: Notes on the introduction of tent field hospitals in war. *Proc Assn Mil Surg* 1894; 4:108–136.
35. Data on Irwin's activities taken from Draft of History of Army Medical Service at United States Military Academy, West Point., New York, typed and undated.
36. Irwin BJD: Amputation at the shoulder-joint. *Am J Med Sci* 1859; 38: 350–353.
37. Irwin BJD: Notes on Euphorbia prostrata as an antidote to the poison of the rattlesnake. *Am J Med Sci* 1861; 41:89–91.
38. Irwin BJD: A case of puncture wound: body transfixed by a bayonet: recovery. *Am Med Times* 1862; 4:274.
39. Irwin BJD: Gunshot wound of arm; amputation at the shoulder-joint. Severe effects to operator from inoculation with matter from wound. *Am J Med Sci* 1861; 41:337–340.
40. Irwin BJD: Case of osteophytic inflammation of the right radius: Resection; Preservation of useful hand. *Am J Med Sci* 1875; 68:131–133.
41. Irwin BJD: Extensive laceration of the hand, caused by the explosion of a bottle of white gunpowder while held in the hand. *Am J Med Sci* 1875; 69: 439–444.
42. Irwin BJD: Three cases of penetrating gunshot wound of the thorax with perforation of lungs; recovery. *Am J Med Sci* 1875; 70:404–407.
43. Irwin BJD: Concussion of the brain with cases illustrative of different degrees of severity. *Am J Med Sci* 1876; 71:399–407.
44. Irwin BJD: Fracture of the sternum with dislocation of the fragments, treatment and the results. *Med News* 1888; 3:691[6]93.
45. Hume EE: *Ornithologists of the United States Army Medical Corps*. Johns Hopkins Press, Baltimore, MD, 1942, p 239.
46. Irwin BJD: History of the Great Tuczon Meteorite. Memphis, Blelock & Co., 1865.
47. Irwin BJD: The Chiricahua Apache Indians: a thrilling incident in the early history of Arizona territory. *The Infantry Journal* 1928; 32:368–375.
48. Irwin BJD: Protest against the appointment of Edward P. Vollum as medical purveyor, USA. Sent to the military committee of the US Senate, September 19, 1890.
49. Ireland MW: Presidential Address, *Milt Surg* 1927; 41:701.

Judicial ruling on a physician violation of New York State law protecting the confidentiality of HIV-related information

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A recent ruling by State Supreme Court Justice Bernard L. Regan of Onondaga County[1] held that a Syracuse physician violated the New York State law protecting the confidentiality of HIV-related information when the physician revealed under subpoena from the Pennsylvania Workers' Compensation Board that the patient was HIV-positive. Justice Regan dismissed the physician's defense of legal immunity based on her compliance with a subpoena holding that a subpoena does not provide sufficient justification to disclose HIV-related information. The statute, Article 27-F of the Public Health Law prohibits physicians and other health-care providers from disclosing HIV-related information except under specific circumstances enumerated by the statute. The statute does not include release of HIV-related information pursuant to a subpoena as an exception to the rule against disclosure. Although the plaintiff's primary complaint of breach of Article 27-F was upheld by Justice Regan, other complaints, including a complaint based on breach of oral contract were dismissed. In dismissing the complaint based on oral contract, Justice Regan ruled that an HIV-infected patient has a legal duty to disclose that fact to the treating physician before undergoing any "bodily invasive physical examination."

BACKGROUND

The patient, identified as John Doe, was tested positive for HIV infection in 1987. In April 1989, John Doe consulted the physician, identified as "Jane Roe, MD"* for treatment related to sinus and ear problems. During the course of treatment, the plaintiff informed Dr Roe that he was HIV positive and described his HIV history so that Dr Roe could take "proper precautions" for any intrusive tests, and because the HIV information might be relevant to the treatment for his sinus and ear problems. The plaintiff also informed Dr Roe that he did not want the HIV-related information to be disclosed to his insurance company. In May 1989, when the sinus and ear problem became aggravated, John Doe filed a Workers' Compensation claim in Pennsylvania. Workers' Compensation hearings were held beginning in November 1989 and continuing up to May 1990 and in this regard, Dr Roe was subpoenaed to submit copies of John Doe's treatment records to the Pennsylvania Workers' Compensation Board. Dr Roe complied with the subpoena and forwarded John Doe's entire chart, including information that Doe had tested positive for HIV. In August 1990, the Pennsylvania Workers' Compensation Board determined that John Doe's alleged injury was not work related. The report made no mention of Doe's HIV status.

THE LAWSUIT

In March 1991, John Doe instituted a lawsuit against Dr Roe for the disclosure of the HIV information. Among the legal grounds claimed by Doe for recovery of monetary damages included breach of statutory duty under Article 27-F of the Public Health Law, breach of the physician-patient confidentiality relationship recognized by 4504(a) of the Civil Practice Law and Rules (CPLR) and breach of oral contract or promise not to reveal the HIV-related information. Dr Roe made a motion to dismiss the lawsuit on the basis of a number of defenses. In requesting that the contract claim be dismissed, Dr Roe stated that there was no enforceable contract because of a lack of "consideration." Generally, for an agreement or promise to be enforceable as a legal contract, there must be "consideration" between the parties, which is the price bargained and paid for in exchange for the other party's promise. To constitute consideration, something must be promised, done, forborne or suffered by the party to whom the promise is made. The plaintiff contended that the consideration given by him in return for Dr Roe's promise not to disclose HIV-related information was his disclosure of HIV-related information to Dr Roe so that Dr Roe could take precautions to prevent infection to herself and her staff. In finding that there was no consideration in return for a promise, and hence, no enforceable contract, Justice Regan held that the patient had a legal duty to inform Dr Roe of his HIV status. According to Justice Regan, the legal duty "arises out of not only moral and ethical considerations, but out of logic, common sense and medical evidence as well, with regard to the general health of society and its physician caretakers." Since Doe was legally obligated to disclose his HIV status to Dr Roe, such disclosure did not constitute consideration or something of value that was given in exchange for Dr Roe's promise not to disclose HIV-related information. Having agreed with the defendant Dr Roe that there was no consideration given in exchange for the oral promise, Justice Regan dismissed the oral contract claim.

Regarding Doe's claim based on breach of statutory duty, Dr Roe claimed that Pennsylvania law and not New York

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*Both John Doe and Jane Roe, MD, are pseudonyms used by the parties in the legal action.

law should control because the action initially arose out of a Workers' Compensation claim and subpoena filed and issued in Pennsylvania. Justice Regan held, however, that New York law and more particularly Article 27-F of the Public Health Law applied because the physician-patient relationship was entered into in New York and the alleged improper disclosure occurred in New York.

Responding to the claims based on breach of statutory duty, Dr Roe contended that she has judicial immunity because she was required under subpoena to release the information. As a preliminary matter, Justice Regan stated that the extra state service of the Pennsylvania subpoena was not a valid subpoena because New York State does not recognize the validity of a subpoena served out-of-state. Had Dr Roe elected not to comply with the out-of-state non-judicial subpoena, the State of Pennsylvania would have been required to seek an order in a New York Court to compel compliance with its subpoena. Inasmuch as the subpoena was "presumptively valid" and Dr Roe did not know that the subpoena was not valid, Justice Regan dismissed portions of the plaintiff's lawsuit as it related to the disclosure of confidential medical information other than HIV-related information.

THE COURT'S RULING

In reference to the release of HIV-related information, Justice Regan held that such disclosure raises different legal issues and policy considerations. The reason for this, according to Justice Regan is that Public Health Law §2780 expressly provides that "no person who obtains confidential HIV-related information in the course of providing any health service or pursuant to a release of confidential health-related information may disclose or be compelled to disclose such information except to certain persons, governmental agencies or private entities further specified by Public Health Law §2782." Section 2782(1)(k) authorizes a physician to disclose HIV-related information when such disclosure is ordered by a "court of record of competent jurisdiction"[†] upon application to certain grounds contained in Public Health Law §2785(2).[‡] The exception provided by §2782(1)(k) was not applicable in the case because the Pennsylvania Workers' Compensation Board is not a "court of record of competent jurisdiction," and made no application for a such a court order.

Justice Regan further ruled that the Workers' Compensation Board was an insurance institution that could legitimately be informed of a patient's HIV status under Public Health Law §2782(1)(i) [for purposes to reimburse health care providers for health services] or §2782(1)(j) [for purposes other than making reimbursement for health services]. To submit HIV-related information to an insurance institution under §2782(1)(j), the insurance institution must first secure a dated and written authorization that indicates the health-care providers and other persons who are authorized to disclose the information, the nature of the information to be disclosed, the purposes for which the information is to be disclosed, and which is signed by the protected individual or a person authorized by law to consent for the protected individual. No such express written authorization was issued in this case, according to the judge.

In ruling that Dr Roe's disclosure was not justified as either in compliance with a court order pursuant to §2782(1)(k) or in accordance with an express written authorization to provide an insurance institution with HIV-related information pursuant to §2782(1)(j), Justice Regan held that the exceptions to the general rule requiring the confidentiality of HIV-related information must be "strictly construed." Even had the subpoena been a valid one, the release of confidential HIV-related information pursuant to subpoena would not be justified.

The remaining argument of Dr Roe was that Article 27-F provides a specific civil penalty of $5,000 and/or criminal misdemeanor prosecution for willful acts, and accordingly, because the statute does not create a private right of legal action, the plaintiff could not maintain a private lawsuit. Justice Regan ruled, however, that in addition to the civil penalties provided under Article 27-F, a private right of action for damages exists under Article 27-F. Because Dr Roe did not act either intentionally or with reckless indifference, stated the Judge, the claim for punitive damages for $50,000 was dismissed. Essentially, in upholding John Doe's claim that Dr Roe was without legal justification to disclose HIV-related information to the Pennsylvania Workers' Compensation Board, a jury is free to decide whether compensatory damages should be paid to the plaintiff, and if so, the amount of such damages.

COMMENTS

The ruling of Justice Regan may seem harsh because it appears to force physicians to risk the legal penalties for non-compliance with a subpoena to avoid the more potentially serious risk of civil and/or criminal penalties and possible legal action under Article 27-F. The case illustrates, however, that courts are likely to be very strict in construing the exceptions to the rule requiring confidentiality of HIV-related information, and physicians should be familiar with the enumerated exceptions to the general rule of confidentiality and the circumstances in which HIV-related information may be disclosed.[**] A physician who is subpoenaed to disclose confidential HIV-related information should consult an attorney, who may seek to have the subpoena quashed.

It appears that the decision in *Doe v Roe* is the first court decision in this State that squarely addresses the question of whether an HIV-infected patient has a legal duty to disclose the HIV status to his or her treating physician. This question was answered affirmatively in *Doe v Roe* in the context of dismissing the patient's complaint based on contractual promise. It will be interesting to see whether other courts will follow this precedent if and when the question is presented in a different context, such as the case where a physician is exposed as a result of treating an HIV-infected patient and the physician seeks damages for the failure of the patient to reveal to the physician the patient's HIV status before undergoing invasive procedures.

REFERENCE

1. *Doe v Roe,* Misc 2d, US, Onondaga County, August 1992.

[†] "Courts of record" are enumerated by §2 of the Judiciary Law.

[‡] Subdivision 2 of §2785 provides that the application for the court order must show: (a) a compelling need for discolosure of the information for the adjudication of a criminal or civil proceeding; (b) a clear and imminent danger to an individual whose life or health may unknowingly be at significant risk as a result of contact with an individual to whom the information pertains; (c) upon the application of a state, county, or local health officer, a clear and imminent danger to the public health; or (d) that the applicant is lawfully entitled to the disclosure and the disclosure is consistent with Article 27- F of the Public Health Law.

[§] Public Health Law 2783.

[**] The Office of General Counsel of the Medical Society of the State of New York has published a summary of the confidentiality requirement of Article 27-F.

W. Somerset Maugham, Capgras syndrome, and aging

JEROME M. SCHNECK, MD

W. Somerset Maugham (1874–1965), the famous English author of novels and short stories, chose writing as a profession after having qualified as a physician. He joined the ranks of other great literary figures who had also qualified in medicine, among them John Keats, Oliver Wendell Holmes, S. Weir Mitchell, Arthur Conan Doyle, and William Carlos Williams. Details of his medical training were incorporated by Maugham in his classic autobiographical novel, *Of Human Bondage*.[1] His opinions about writing and literature appear in *The Summing Up*.[2] *The Moon and Sixpence* and *The Razor's Edge* are among his other widely read books, and his short stories include "Rain" and "The Letter." In addition, many successful plays attest to his productivity and abilities. All of the aforementioned works are discussed in a biography of Maugham by Ted Morgan.[3]

Maugham's nephew, Robin Maugham (1916–1981), was the son of his elder brother, Lord Maugham (Frederic Herbert Maugham), a distinguished lawyer and Lord Chancellor of England.[3] Robin, like his uncle, was a best-selling novelist and a writer of plays, although he did not attain the stature of Somerset Maugham. He qualified first as an attorney. His most widely known novel is *The Servant*, which was made into a film (as were many of his uncle's books).

In 1978, Robin Maugham published a small book, *Conversations With Willie: The Recollections of W. Somerset Maugham*, centering on his visits to this uncle's Villa Mauresque on the French Riviera where, at the age of 90, Somerset Maugham displayed on three or more occasions the symptoms now recognizable as Capgras syndrome.[4] These symptoms involve misidentifications of persons, in this case Robin himself, and it is reasonable to suggest that are probably more significant than heretofore recognized in relation to evaluations of memory loss in the elderly.

CAPGRAS SYNDROME

In 1923, Capgras and Reboul-Lachaux reported the case of a 53-year old woman who suffered from the delusion that persons significant to her had been replaced by impostors.[5] Similar reports began to appear afterwards with increasing frequency, but the phenomenon is still not widely known and diagnosed. At first the reports appeared mostly in British and French psychiatric literature. Now, they are also to be found in American journals. The descriptions are almost invariably termed Capgras syndrome.

Several variants of the syndrome have been described by Atwal and Khan.[6] One example is the autoscopic type in which patients see doubles of themselves in persons or objects nearby, as noted also by Signer.[7] There is, in addition, a hallucinatory autoscopic variant; this phenomenon served as a major theme in stories by Edgar Allan Poe published in 1839, Charlotte Perkins Gilman in 1891, and in the novel, *The Double*, by Fyodor Dostoyevsky published in 1846.[8–13]

Capgras syndrome has often been associated with schizophrenia and affective psychoses, but it has also been encountered in patients with varying types and degrees of brain pathology. Lewis,[14] for example, described a girl aged 19 years in whom magnetic resonance imaging revealed bilateral subcortical lesions in the occipitotemporal and frontal regions. During a six month follow-up a concurrent diagnosis of "schizophreniform psychosis" was made as well. Koritar and Steiner[15] noted differences of opinion about the etiologic factors in Capgras syndrome. They described two patients diagnosed as suffering from paranoid schizophrenia in whom general physical and neurologic examinations revealed normal findings. Pelletier, Bartolucci, and Wallace[16] regarded the syndrome as a nonspecific symptom seen in both functional and organic conditions. It seems reasonable to believe that an either/or approach is unnecessary because a combination of psychopathologic and neuropathologic etiologies may be present.[17]

The most widely accepted psychodynamic hypothesis in the evaluation of the Capgras delusion is the "ambivalence theory" according to Fishbain and Schiffman.[18] The theory notes that the patient harbors strongly ambivalent feelings toward a person significant to him which are not adequately repressed, and the anxiety resulting from this conflict induces "a psychotic solution based on denial and displacement."[18] This view is consistent with the findings reported in a patient described by the present author.[17] It applies also to the episodes experienced by Somerset Maugham.[4(pp172–183)]

MAUGHAM IN OLD AGE

In his 80s, Maugham grew increasingly frail and suffered hearing loss and failing sight. He had cataracts and said, "My favorite pastime, reading, is now denied to me." His mental faculties became significantly impaired. He had memory lapses and episodes of confusion, and he displayed abnormal behavior. Morgan described a lunch to which Maugham invited a man named Eric Dunstan whom he disliked but whom his secretary, Alan Searle, suggested he entertain. They chatted and laughed amiably. "After lunch, as they sipped their coffee, Maugham gave Dunstan a piercing look and said, 'Tell me, whatever happened to that swine Eric Dunstan?' "[3(p610)] On another occasion a member of Parliament and an art critic visited the Villa Mauresque. "As they walked into the drawing room,

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Maugham popped up from behind the sofa adjusting his trousers. He had defecated on the rug, and scooped up a handful of feces like a guilty child."[3(p610)] Once, when Maugham's secretary returned to the Villa Mauresque after an evening out, he found the house in a turmoil. "Maugham had disappeared. They found him walking on one of the roads in his stockinged feet."[3(p610)]

In 1964, when Maugham was 90-years old, Robin visited his uncle at the Villa Mauresque and found him suspicious of all the servants, feeling they were stealing from him at the same time. Maugham claimed, "I am one of the most horrible old men alive today. I've been so wicked, I've been so cruel."[4(pp172–173)] Invited to lunch with an old friend, he said to his hostess, "This is a very indifferent meal." Later, he commented, "As a thoroughly second-rate writer there's nothing very much I deserve. But if I hadn't had a stammer, I could have been a great man."[4(pp177–178)] Maugham had been troubled by a stammer throughout his life. In his autobiographical novel, *Of Human Bondage*, he described his protagonist as suffering from a club-foot instead of a stammer.[1]

Robin found his uncle often irritable and angry. "He would sit like a malignant crab on the first-floor gallery above the patio."[4(p178)] He would scowl and glare with loathing. He would often be unpleasant and highly critical. He complained of being old and tired and miserable. "I want to die so much."[4(p179)] Robin observed, "His grief was heart-rending. I felt the tears scudding into my eyes. It was anguish to see a person one had once respected and loved suffering such despair."[4(p179)]

Robin also noted, "But at times he now began to dwell more and more on the happier side of his past and tell stories about the kings, queens, and princesses he had known and the splendors of his life."[4(pp179–180)] Yet Maugham also said, "I don't care any more. It all doesn't amount to anything. No one in the world cares about me anymore . . . It's true, you know, I'm quite forgotten. No one thinks about me or cares about me not a row of beans. And anyhow, kings, queens, and princesses, the whole bloody royalty, they don't really amount to anything. I led a very grand life in the days of my success. But it didn't last for long. I used to drink with kings and queens. But I can't remember their names. I have to speak very quickly these days because no sooner have I opened my mouth than the matter flies from my mind."[4(p183)] On the occasion when Robin asked his uncle, "What is the happiest memory of your life?" Maugham replied, "I can't think of a single one."[4(p182)] During his last visit to the Villa Mauresque, when Maugham was in bed, Robin approached him. "He was asleep. He looked like a shriveled doll."[4(p183)]

MAUGHAM AND CAPGRAS SYNDROME

When Maugham was 90 years old he had at least three episodes of Capgras' syndrome in his home, Villa Mauresque on Cap Ferrat in the South of France. Maugham's episodes of Capgras Syndrome were witnessed and recounted. Maugham was querulous, displaying marked mood swings, anger and affection, bitterness, sweetness, charm, and hostility.[4(pp172–183)]

On one occasion in June 1964, Maugham had cocktails and dinner with Robin. His secretary-companion, Alan Searle had a long needed night out alone. Maugham asked Robin the time and was told, "About nine." He asked for Alan. Robin said, "As you know, he's gone out for a while." Five minutes later, Maugham urgently asked the time again and wanted to know where Alan had gone. He began to tremble and repeated the same questions. Robin did not know exactly where Alan had gone, although it was probably to Nice. "Then Willie turned on me. His hands clawed frantically at the air. 'I know you are the stupidest member of your family, but perhaps you can answer a simple question. What time is it?'" Twenty minutes had past. Maugham inquired acidly about arrangements Robin had made with Alan. He had made none. "'Where has he gone?" Willie now addressed me as if I were a complete stranger. 'When will Mr Searle be back,' he demanded." Alan Searle was of course long well known to Robin, and Maugham, addressing Robin and Robin speaking with his uncle, never referred to Alan as Mr Searle. Robin did not know when the secretary-companion would return to the villa. "Then I shall wait here until Mr Searle does return,' Willie announced."

"Suddenly he turned on me once more. His face was contorted with hatred. 'Where do you live," he asked, in a voice quivering with rage. 'In Brighton.' 'Don't be willfully stupid,' he said. 'I mean, where are you staying tonight?' 'Here in your villa,' I replied firmly. 'I'm your guest.' 'No, you're not,' he answered. 'There's no room for you. Who are you? What room do you think you are going to sleep in?' "

Maugham tottered out of the room and screamed at the staff in the kitchen. When he returned, he asked again where Alan Searle had gone. "'I'm asking you, when is he coming back? When will Mr Searle be here? What are you doing in this house? What is your name?'" Maugham rushed out again and returned with a kitchen assistant. "'Dans quelle chambre est-ce-que monsieur va se coucher?' 'Mais en haut, monsieur,' Marie answered. 'Dans quelle chambre?' Willie demanded. 'Dans la chambre a lui.' 'Montre la moi.' So upstairs we trooped. Marie, terrified, opened the door of my bedroom. Willie glared into the empty room. Then he flicked the light switch. 'I'm not going to pay for any more electricity for you than that.' "

Robin felt he should say something pleasant. He thanked his uncle for an excellent dinner. "Willie tottered toward me, shaking with rage. I thought he would strike me. 'That's the very least you should say to me,' he shouted. 'I expect more than that.' And he shuffled angrily from the room. Marie, in tears, gave me a look of commiseration before she left. Willie slammed the bedroom door shut. I could hear him screaming and shouting along the patio."

"Alan returned about ten thirty. 'The staff are in tears,' he reported. 'I haven't had an evening out for a whole year, and now I shall feel I can never go out again.' "

Robin wrote in his diary, "I'm sure that if Willie had a knife or gun, he would have killed me. He is, I'm now convinced, a maniac. It is his quality of malignancy that pierces me with fear."

The next morning Maugham had completely forgotten the events of the previous night. Robin, wearing a "toweling gown" belonging to the villa, was on his way to the pool. His uncle greeted him affectionately: "You're looking very smart."

On another occasion, Maugham was reminiscing. Robin gave him a cigarette and lit it up for him. Maugham said "That's extremely kind of you. You know, I've always wondered how it would be to have a relationship with someone who had lived two or three thousand years before you. Even if it were a brief relationship. Speaking of relationships, do you know my nephew Robin? He's a very pleasant person. He's promised to visit me. I wonder if on your travels you ever met him?" "I am Robin." "No." "I am Robin Maugham." "That is as it may be. But the Robin to whom I am referring was quite different . . . Did I tell you that I think my brother Freddie (Robin's father) may have been a lord? But he never told me. If I'd asked him when he was in a good humor he might have said he was the duke or something. But otherwise he'd have told me to go to hell."

The following morning Robin was in his bath when Willie shuffled through his bedroom and stood over him. Robin said Good morning and Maugham demanded "What was that?" Maugham was silent for a moment, glowering down at Robin without recognition. He then asked suddenly, "Do you know who that bath you're in belongs to?" "Yes." "To whom does it belong?" "You, Willie." "And who is going to use it when you've finished with it?" "I simply don't know," Robin replied. Maugham shook his head and shuffled out.

Later that morning, Maugham asked Robin, "Does my nephew look like you?"

"Precisely," Robin replied. Whereupon, Maugham observed, "Then he must be a charming person."[4(pp172–183)]

Maugham, Robin, and the Ambivalence Theory of Capgras Syndrome

Details of Maugham's contacts with his older brother Frederic reflect ambivalent feelings on the part of both toward each other. It is not unreasonable to suggest that Maugham's ambivalence toward Frederic's son Robin may have been in part an extension of his attitude toward Frederic. Robin wrote about his father and uncle in *Somerset and All The Maughams.* Biography was one of his interests.[19] Several events illustrating Maugham's ambivalence toward his nephew have been described by Robin in his book about conversations with his uncle.[4] This is consistent with the ambivalence theory of Capgras syndrome.[17,18]

An amusing episode reflecting Maugham's behavior toward his nephew occurred when he anticipated a visit to the Villa Mauresque by Dr Felix Martí-Ibáñez.[4(pp105–106)] Martí-Ibáñez was a psychiatrist, medical historian, fiction writer, and the internationally known editor-in-chief of *MD* magazine. He was long a friend of Maugham, who had once asked him to edit, for historical accuracy, his novel about Spain, *Catalina*. Martí-Ibáñez also wrote short stories which he sent to Maugham for criticism.[3(p493)] Maugham, when in his eighties, told Robin that Martí-Ibáñez and his wife were coming to lunch the following day.[4(pp105–106)] "And they are coming to see me, not you. So you can keep quiet and speak only when spoken to." The guests arrived and greeted Maugham warmly. "'And this,' Willie said, indicating me with a quick nod of his head, 'is my nephew Robin.' Dr Ibáñez stared at me. 'Not Robin Maugham?' he blurted out. 'Yes,' I said, dreading what was to come. 'The Robin Maugham who wrote *The Servant, Come To Dust, Behind the Mirror*. . . ?" The list seemed to me interminable. Meanwhile, Willie had begun to scowl. 'My wife and I have read all of your books,' Dr Ibáñez continued. 'We admire them very greatly.' Willie's scowl grew heavier. But he made no mention of the conversation later."[4(pp105–106)]

On another occasion, Maugham, in front of our dinner guests told Robin, "You'll never be a writer. I see you as an aging, impoverished viscount on the fringes of literary society." Robin had inherited the title from his father. Afterward Maugham said to his nephew, "I must tell you here and now that you'll never make a writer. I want you to give up the attempt before you make a complete fool of yourself."[4(pp96–97)]

Robin wrote that when *The Servant* was published a reviewer for the *New York Times* "hailed it as a 'masterpiece of writing'; unfortunately, he added the fatal words 'written with a skill and speed the author's uncle might well envy.' My publisher used this quote in their advertisements. This enraged Willie, and for two or three years I knew I was out of favor."[4(p95)]

At another time, Maugham complimented Robin on his writings but advised him against writing about homosexuality because it would ruin his reputation in England. It would outrage the public. "'Don't put your head in a noose, even though it may give you a tawdry success with some of your friends.' Suddenly, he bent down and kissed me on the head. 'Don't be angry with what I've said; it's all for your own good.'" Maugham, who once married and had a daughter, was always greatly concerned about his homosexuality becoming known to the general public, and he refrained from dealing with the subject in his books.[4(p118)] "Thus," Robin observed, "he never wrote a homosexual novel as such, although the discerning reader can detect a trace of homosexuality in his excellent novel, *The Narrow Corner*."[4(pp35–36)] Robin did not have his uncle's need for concealment either in his personal life or in his own novels. He revealed at one point that his uncle was angry because "I was living openly on my little yacht with a boy, and far worse, I was writing novels which sold well."[4(p96)]

Although Maugham could be scathingly critical about Robin as a writer, as Robin himself revealed, he managed also, as Maugham's biographer Ted Morgan commented, to compliment him and give him advice. Whereas Robin was able to write about the harsh behavior Maugham displayed toward him, Morgan tends to highlight positive attitudes.[11] "Maugham saw in Robin the son he never had. He befriended him, gave him advice, took an interest in his work, and settled a trust fund on him. Maugham played the role of benevolent uncle so well that young Robin preferred him to his father."[3(p380)] Lord Maugham disapproved of his brother's life as a writer and his life-style in general. He was also critical of his son Robin, who preferred his uncle's profession to that of his father.[3(pp379-390)]

During World War II, Robin was a tank commander in the African campaign. His uncle wrote, in 1942, "I hope you have got through this present fighting unscathed and are successfully smacking German and Italian tanks as you have done in previous battles." He was proud of Robin's gallantry in action to which his brigadier attested. Then Robin was wounded in the chest by shrapnel and soon thereafter in the head. He was released from a hospital near Suez "with a piece of shrapnel still lodged in his brain," and he suffered "blackouts and fits of vomiting and headaches."[3(p469)] Subsequently, in one of his critical moods, Maugham told Robin that he was stupid enough to let himself get shot in the head.[4]

When Robin was in the Army, Maugham set up the aforementioned trust fund for him. Subsequently, when annoyed with Robin and recognizing that the fund's value had increased considerably, Maugham reduced the amount and set a limit to what Robin should receive. He said he felt Robin was frivolous and scatterbrained and hadn't grown up, throwing money about recklessly, mixing with disreputable people, and failing to make a success at living, farming, and writing.[3(p548)] However, Robin did establish his own reputation as a novelist even though, as mentioned, he declined to hide his homosexuality as his uncle had done throughout his life.

Maugham envied Robin's youth, alternating admiration for his handsome appearance and personality with bitter criticism, and deploring at the same time his own aging and deterioration. He often enjoyed Robin's pleasant company, yet often as well, when unhappy and displeased, lashed out critically and unwarrantedly. He harbored a love-hate relationship with his nephew, a type of relationship that intensified as he became a bitter, very unhappy, old man.

In his old age, Maugham became increasingly querulous, stingy, and paranoid, and as Robin observed, he believed his servants were stealing his money. Sweetness and charm became repeatedly overshadowed by vitriolic anger. His mood swings were drastic. He deplored the eventual loss of all his material possessions through death. His senile functioning also consisted of lapses in memory, mild to severe, recent and past, and unpredictable with the increasing psychopathological and neuropathological alterations associated with his aging. Maugham's death at age 91 occurred after a fall in his villa. He became unconscious and comatose and soon died in a nearby hospital.[3(p617)]

CONCLUSIONS

Capgras syndrome occurs in the elderly as well as in persons who are young and in their middle years. It is best known to some neurologists and psychiatrists, but not as much to other physicians. The occurrence of the syndrome in Somerset Maugham, aside from intrinsic interest because of his fame, prompts the suggestion that clinicians treating the elderly might, with greater awareness, be capable of differentiating frequent neurologically and psychologically determined memory lapses and states of confusion from the less frequent misidentifications of the Capgras phenomenon. Awareness of the ambivalence factor could aid in recognition of the syndrome. Assessing patients' interpersonal relations, especially within family settings, could yield more effective treatment of the elderly when family dynamics are taken into consideration.

REFERENCES

1. Maugham WS: *Of Human Bondage.* New York, Doubleday, 1915.
2. Maugham WS:*The Summing Up.* New York, Doubleday, 1938.
3. Morgan T: *Maugham.* New York, Simon and Schuster, 1980.
4. Maugham R: *Conversations With Willie: The Recollections of W. Somerset Maugham.* New York, Simon and Schuster, 1978.
5. Capgras J, Reboul-Lachaux J: L'illusion de "sosies" dans un delire systematise chronique. *Bull Soc de Med Ment* 1923; 2:6–16.
6. Atwal S, Khan MH: Coexistence of Capgras and its related syndromes in a single patient. *Aust NZJ Psychiatry* 1986; 20:496–498
7. Signer SF: Capgras syndrome: the delusion of substitution. *J Clin Psychiatry* 1987; 48:147–150.
8. Poe EA: William Wilson, *The Works of Edgar Allan Poe,* vol 3, part 1. New York, Leslie Judge Company, 1912, pp 143–174; *The Tell Tale Heart and Other Writings.* New York, Bantam Books. 1982, pp 182–201.
9. Schneck JM: Edgar Allan Poe's "William Wilson" and Capgras syndrome. *J Clin Psychiatry* 1990; 51:387–388.
10. Gilman CP: *The Yellow Wallpaper and Other Writings by Charlotte Perkins Gilman.* New York, Bantam Books, 1989, pp 1–20.
11. Gilman CP: *The Living of Charlotte Perkins Gilman: An Autobiography.* New York, D. Appleton-Century Company, Inc, 1935.
12. Schneck JM: S. Weir Mitchell, Charlotte Perkins Gilman's "The Yellow Wallpaper," and Capgras syndrome. *NY State J Med* 1991; 91:445–449.
13. Dostoyevsky F: *The Double* (1846). London, Penguin Books, 1972.
14. Lewis SW: Brain imaging in a case of Capgras syndrome. *Br J Psychiatry* 1987; 150:117–121.
15. Koritar E, Steiner W: Capgras syndrome: a synthesis of various viewpoints. *Can J Psychiatry* 1988; 33:62–66.
16. Pelletier JR, Bartolucci G, Wallace J: Capgras syndrome: the many avenues of delusional misinterpretation. *Can J Psychiatry* 1985; 30:225–227.
17. Schneck JM: Capgras syndrome. *NY State J Med* 1990; 90:470–471.
18. Fishbain DA, Schiffman J: The daughter as the principle "double" in a Capgras syndrome: psychodynamic correlates. *Am J Psychiatry* 1986; 40:607–611.
19. Maugham R: The art of biography. *MD* 1980; 13–15.

Osler-Weber-Rendu disease

Diagnosis and management of spontaneous hemothorax during pregnancy

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Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome) is a relatively rare, inherited disease transmitted via an autosomal dominant gene.[1] It is characterized by multiple arteriovenous malformations mainly situated in the skin and mucous membranes. Accordingly, epistaxis is highly characteristic of the disease.[2] However, there have been many cases in which patients had lesions in several organs of the body including the lung.[3,4] These aneurysms have thin walls and may spontaneously rupture.[4–6]

The association of pulmonary arteriovenous fistulas with this syndrome makes it important that such patients be routinely evaluated. These fistulas often give rise to symptoms and physical findings such as dyspnea and cyanosis;[4,6] polycythemia and clubbing may also be seen.[4,6] Cerebrovascular accidents can occur, possibly related to thromboemolism.[4,6] Auscultatory findings may include a bruit on chest examination with diastolic accentuation during inspiration and a systolic murmur.[4,6] Rupture is usually accompanied by hemoptysis, but on rare occasions hemothorax can occur.[4–6] Pregnancy has been implicated as a cause of deterioration in patients with arteriovenous fistulas: however, the mechanism remains unclear.[7,8]

Case Report

A 24-year-old woman, who was six months pregnant, was admitted to the labor and delivery suite of New York University Medical Center with the acute onset of left-sided pleuritic pain and shortness of breath. Her pregnancy had been uneventful until that point. On examination, she had absent breath sounds over the left hemithorax, was slightly cyanotic, and had clubbing of her fingertips. The remainder of the examination was unremarkable.

Blood gas studies on admission showed a pH of 7.50; Pao_2, 60 torr; and Pco_2, 30 torr on room air. She denied any significant medical history. A chest film showed complete opacification of the left hemithorax, and a thoracentesis yielded non-clotting blood. A pulmonary angiogram showed a large, bleeding arteriovenous fistula in the left lower lobe (Fig 1).

The patient underwent an embolization procedure with two medium Gianturco stainless steel coils, which successfully occluded the arteriovenous fistula. A chest tube was inserted to drain the hemothorax. After these procedures, the patient's clinical status greatly improved, and her arterial blood gas measurements returned to normal (pH 7.40; Pao_2, 90 torr; and Pco_2, 40 torr).

On further questioning, the patient admitted to having a history of frequent epistaxis, and an examination of the nasopharynx revealed numerous telangiectasias. Although she was vague about her family medical history, she did note that a grandfather had been hospitalized several times for bleeding before he died of a massive hemorrhage secondary to Osler-Weber-Rendu syndrome.

The patient continued her pregnancy without any further difficulty and gave birth to a healthy baby boy at term. She has not had any other episodes of bleeding except for occasional epistaxis. A follow-up pulmonary angiogram has shown persistent occlusion of the embolized arteriovenous fistula, and an arterial blood gas on 100% oxygen has shown a normal shunt fraction. Abdominal and head computed axial tomography (CAT) scans have not demonstrated any other vascular abnormalities.

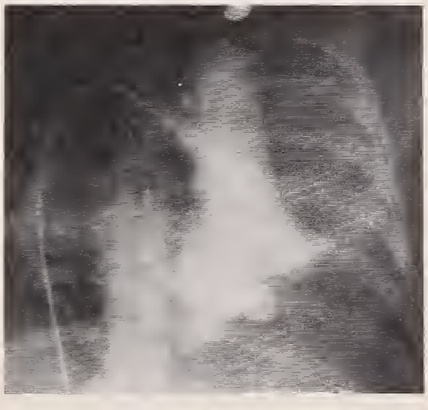

FIGURE 1. Pulmonary angiogram showing large bleeding arteriovenous malformation in the left lower lobe and associated hemothorax.

Discussion

Hereditary hemorrhagic telangiectasia has been known as a disease entity for more than 100 years. It was initially known as "hereditary epistaxis."[9] At the turn of the century, Rendu,[10] Osler,[2] and Weber[11] independently described the condition as mainly affecting the skin and mucous membranes. Over the succeeding years, additional cases were described, and the pattern of inheritance as an autosomal dominant characteristic was established. Later, the pulmonary manifestations of this disease became known. Approximately 50% of patients with pulmonary arteriovenous fistula have hereditary hemorrhagic telangiectasia.[4]

These vascular malformations occur most often singly but may also be multiple.[4–6] They have thin walls and may rupture spontaneously.[4–6] Anatomically, the pulmonary arteriovenous aneurysm is a pathologic connection between an afferent pulmonary artery and one or more efferent veins.[5] It may take the form of a fistula composed of dilated irregular vessels, or it may be a smooth aneurysmal, thin-walled sac as in the case described here.[5] In about 20% of cases, the aneurysms may be bilateral or occur in ipsilateral lobes.[4–6] Rarely, the afferent artery may arise from the aorta or from an intercostal artery.[4,5] Variations in the size of a pulmonary arteriovenous malformation may be induced by the Valsalva or Muller maneuvers.[4,6] A bruit is occasionally audible over the aneurysm.[4,6] Sometimes the aneurysms are quite small and may be overlooked on routine radiographic examination. Angiography may be needed for complete evaluation.[5]

Since the number and size of telangiectasias in the skin and mucous membranes increase with age, it is likely that pulmonary arteriovenous malformations do as well. Most occur in patients under 40, and they are rare in children.[13] Aneurysms may be present in the lungs even when no telangiectasias are noted in the skin or mucous membranes.[4,6]

When pulmonary symptoms are present, they are usually due to the hypoxia that occurs as a result of shunting through the

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arteriovenous malformation or rupture.[4,6] Intrabronchial rupture producing hemoptysis occurs in about 25% of reported cases, but intrapleural rupture is extremely rare and frequently fatal when it does occur.[4–6] The bleeding is usually rapid and massive, requiring urgent intervention. For unclear reasons, most episodes of hemothorax have been reported as occurring on the left side. Occasionally, neurologic symptoms such as hemiplegia or loss of consciousness may occur. These episodes are presumably due to emboli (either thrombus or air) arising from the arteriovenous malformation.[4,6] Cerebral abscesses have also been noted.[4,6] Pregnancy has been associated with the deterioration of arteriovenous malformations.[7,8] The increased blood volume occurring in pregnancy may be a predisposing factor placing additional stress on a thin-walled aneurysm. Vascular changes related to hormonal levels during pregnancy may also play a role in further weakening the aneurysm wall.

Previously, surgical resection was the only treatment available for symptomatic pulmonary arteriovenous malformations. Now, embolotherapy is the treatment of choice.[17,18] Silicone balloons and coil springs have been used most often. This patient underwent a successful total occlusion of the left lower lobe arteriovenous malformation as demonstrated on follow-up angiography. Her shunt fraction also returned to normal.

Pregnancy seems to be a significant risk factor for the worsening of arteriovenous malformations in patients with Osler-Weber-Rendu syndrome. The proper management of life-threatening pregnancy complications in pregnant patients requires angiography with emergency embolotherapy, which can be done safely and effectively with proper radiation shielding of the fetus.

REFERENCES

1. Hones FM: Multiple hereditary telangiectases causing hemorrhage (hereditary hemorrhagic telangiectasia). *Bull John Hopkins Hosp* 1909; 20:63–73.
2. Osler W: On a family form of recurring epistaxis, associated with multiple telangiectases of the skin and mucous membranes. *Bull Johns Hopkins Hosp* 1901; 12:333–337.
3. Grung P: Telangiectasia hemorrhagica hereditaria Osler with arteriovenous aneurysms of the lung and with hepatosplenomeglia. *Acta Med Scand* 1954; 150:95–100.
4. Hodgson CH, Kaye RL: Pulmonary arteriovenous fistula and hereditary hemorrhagic telangiectasia: a review and report of 35 cases of fistula. *Dis Chest* 1963; 43:449–455.
5. Sammons BP: Arteriovenous fistula of the lung. *Radiology* 1959; 72:710–715.
6. Dines DE, Arms RA, Bernatz PE, et al: Pulmonary arteriovenous fistulas. *Mayo Clin Proc* 1974; 49:460–465.
7. Swinburne AJ, Fedullo AJ, Gangemi R, et al: Hereditary telangiectasia and multiple pulmonary arteriovenous fistulas: clinical deterioration during pregnancy. *Chest* 1986; 89:459–460.
8. Gammon RB, Miksa AK, Keller FS: Osler-Weber-Rendu disease and pulmonary arteriovenous fistulas: deterioration and embolotherapy during pregnancy. *Chest* 1990; 98:1522–1524.
9. Babington BG: Hereditary epistaxis. *Lancet* 1865; 2:362.
10. Rendu M: Epsitaxis repetees: chez un porteur de petits angiomes cutanes et mugueux. *Bull Soc Med Hop Paris* 1896; 13:731–734.
11. Weber FP: Multiple hereditary developmental angiomata (telangiectases) of skin and mucous membranes associated with recurring hemorrhages. *Lancet* 1907; 2:160–164.
12. Hoffman R, Robins R: Evolving pulmonary nodules: multiple pulmonary arteriovenous fistulas. *AJR* 1974; 120:861–1[8]64.
13. Shumacker HB Jr, Waldhausen JA: Pulmonary arteriovenous fistulas in children. *Ann Surg* 1963; 158:713–720.
14. Kintzer JS, Jones FL, Pharr WF: Intrapleural haemorrhage complicating pulmonary arteriovenous fistula. *Br J Dis Chest* 1978; 72:155–157.
15. Hodgson CH, Burchell HB, Good CA, et al: Hereditary hemorrhagic telangiectasia and pulmonary arteriovenous fistula: survey of a large family. *N Engl J Med* 1959; 261:625–635.
16. Dalton ML Jr, Goodwin FC, Bronwell AW: Intrapleural rupture of pulmonary arteriovenous aneurysm: report of a case. *Dis Chest* 1967; 52:97–100.
17. White RI, Lynch-Nyhlan A, Terry P, et al: Pulmonary arteriovenous malformations: techniques and long term outcome of embolotherapy. *Radiology* 1988; 169:663–669.
18. Keller FS, Rosch J, Barker AF, et al: Pulmonary arteriovenous fistulas occluded by percutaneous introduction of coil springs. *Radiology* 1984; 152:373–375.

Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids

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In the setting of inflammatory bowel disease, adrenal hemorrhage has been reported with the use of adrenocorticotropic hormone (ACTH).[1] Adrenal hemorrhage, when accompanied by more than 90% glandular destruction, may present with symptoms of adrenal crisis or with findings of an acute abdomen.[2] We report what we believe to be the first case of unilateral adrenal hemorrhage in a patient with inflammatory bowel disease treated with corticosteroids.

CASE REPORT

A 45-year-old woman presented as an outpatient with complaints of nausea, vomiting, and epigastric pain. The patient had enjoyed good health until three months earlier when she experienced diarrhea and epigastric pain. The patient underwent sigmoidoscopy, which was normal. The patient's symptoms continued, and she began to experience fatigue, anorexia, and had a 12-pound weight loss over the next three months. An upper gastrointestinal series with small bowel follow-through showed antral and duodenal thickening, and also demonstrated a string sign of the terminal ileum consistent with the diagnosis of Crohn's disease.

She was started on ranitidine, 300 mg daily, and sulfasalazine 1,000 mg twice daily. However, the patient was unable to take the medications because of persistent vomiting, and she was admitted to the hospital. Physical examination revealed mild epigastric and right lower quadrant tenderness. The admission hemoglobin count was 11.5 g/dL and the erythrocyte sedimentation rate (ESR) was 55 mm/hr. The patient underwent upper endoscopy, and an inflammed duodenum and antrum were noted. Colonoscopy showed marked inflammatory changes in the right colon, and a biopsy showed inflammatory cells and granuloma formation consistent with Crohn's disease. A computed tomography (CT) scan confirmed right colonic wall thickening with no other abnormalities.

The patient was started on intravenous hydrocortisone, 70 mg every eight hours. In addition, she was given metronidazole, 500 mg every six hours, plus cimetidine, 1,200 mg daily by infusion, and she was kept on intravenous nourishment. Her condition improved, and she was started on clear liquids. Four days later, she had sudden onset of right lower quadrant pain radiating to the right flank. The pain progressively worsened throughout the day. Plain abdominal radiographs and urinalysis were normal. The pain persisted, and an intravenous pyelogram was performed and was found to be normal. Fever developed to 101.4°F, with a fall in the hemo-

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globin to 8.5 g/dL from 10.2 g/dL. There was no change in blood pressure. A repeat CT scan of the abdomen revealed a new right adrenal hemorrhage. The left adrenal gland showed normal morphology. The patient remained on steroids without further problems and was discharged several days later.

DISCUSSION

Adrenal hemorrhage can present with abdominal, flank, or chest pain, in addition to abdominal tenderness, hypotensive shock, nausea, vomiting, confusion, and disorientation. Subsequently, severe hypotension, volume depletion, dehydration, hyperpyrexia, cyanosis, coma, and death may ensue. The diagnosis of acute adrenal hemorrhage should be considered in a patient with unexplained abdominal or flank pain, vascular collapse, hyperpyrexia, or hypoglycemia.[1] The most impressive laboratory finding is a sharp drop in the hemoglobin and hematocrit levels.[2] Confirming the diagnosis requires hormonal proof of adrenal failure, or anatomic evidence of hemorrhage.

The CT scan is an accurate method for evaluating the morphologic status of the adrenal glands.[3] The mechanism of adrenal hemorrhage can vary according to the clinical setting. The three major risk factors for acute adrenal hemorrhage appear to be thromboembolic disease, coagulopathy (spontaneous or iatrogenic), and the postoperative state.[2] Adrenal hemorrhage can occur in patients with overwhelming infection and life-threatening illnesses. Lundstrom et al[3] reported a case of bilateral adrenal hemorrhage after surgical distress in a patient who had been taking steroids for eight years. The probably pathogenic mechanism was surgical stress superimposed on subnormal adrenal function, secondary to the long-term glucocorticoid treatment.

Metastases to adrenal glands are common, but adrenal hemorrhage resulting from these metastases is uncommon.[4] There have been reported cases of massive adrenal hemorrhage secondary to metastatic lung carcinoma and metastatic gastric carcinoma.

Dunlap et al[1] reported two patients on intravenous ACTH therapy for active inflammatory bowel disease who sustained the complication of bilateral adrenal hemorrhage.[1] They were subsequently changed to intravenous corticosteroids. The presumed mechanism of adrenal hemorrhage in this setting is stimulation of glandular growth causing avascular necrosis.

The patient presented here had been on steroids for only five days before to her onset of acute right-sided abdominal and flank pain. She presented with atypical symptoms of acute adrenal hemorrhage and CT scan findings consistent with a new right adrenal hemorrhage. With the use of ACTH, we can postulate that increased stimulation leads to an increase in gland size, which may cause avascular necrosis. Treatment with corticosteroids should have the opposite effect, and hence hemorrhage must be considered unusual.

In 1955, Owen and Coleman[5] reported a case of massive adrenal hemorrhage in a severe case of ulcerative colitis. In 1950, Rice-Oxley and Truelove[6] reported a similar case with thrombus noted in the suprarenal vein. Both of these earlier reports were autopsy based and characterized by extensive bilateral hemorrhage. Sepsis may have been a factor in both of these cases.

Adrenal stimulation is a natural consequence of stress. Coagulation anomalies are well established in inflammatory bowel disease and may also play a role.

This is the first case to our knowledge of non-fatal adrenal hemorrhage in inflammatory bowel disease without the use of ACTH. Physicians should be alerted to this possible complication in view of the widespread use of corticosteroids in treating inflammatory bowel disease.

REFERENCES

1. Dunlap SK, Meiselman MS, Breuer RL, et al: Bilateral adrenal hemorrhage as a complication of intravenous ACTH infusion in two patients with inflammatory bowel disease. *Am J Gastroenterol* 1989; 84:1310–1312.
2. Rao RH, Vagnucci AH, Amico JA: Bilateral massive adrenal hemorrhage: early recognition and treatment. *Ann Intern Med* 1989; 110:227–235.
3. Lundstrom GM, Chen PS: Bilateral adrenal hemorrhage associated with prior steroids use: CT diagnosis. *J Can Assoc Radiol* 1985; 36:58–60.
4. Shah HR, Love L, Williamson MR, et al: Hemorrhagic adrenal metastases: CT findings. *J Comput Assist Tomogr* 1989; 13:77–81.
5. Owen GD, Coleman PN: Spontaneous adrenal hemorrhage in ulcerative colitis. *Bri Med J* 1955; 1:88.
6. Rice-Oxley JM, Truelove S: Complications of ulcerative colitis. *Lancet* 1950; 1:607–611.

Myxedema coma

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At a meeting of the Northumberland and Durham (Great Britain) Medical Society on February 12, 1891, George Bedmayne Murray, MD, presented the interesting case of Mrs S, a 46-year-old woman who had gradually become slow in her actions and speech. She complained of a great sensitivity to cold, her temperature was low, and her heart rate 60–70/min. Her features were thickened; she had marked periorbital edema, dry skin, and fine hair; all of the classical features of advanced hypothyroidism, or myxedema. Dr Murray proposed that Mrs S be treated with a "liquor thyroidei," a glycerin extract of sheep thyroid he had concocted, beginning with an injection of "25 minims" (about 25 drops) of extract twice weekly. After three months of treatment, her symptoms had all but resolved, and she was switched to an oral regimen, on which she lived in excellent health for 28 years.[1]

Had it not been for Dr Murray's careful observations and bold treatment plan, Mrs S's myxedema would surely have progressed to the severe end stage of myxedema coma, still a relatively rare condition with only about 200 cases reported between 1953 and 1986.[2]

CASE REPORT

A 72-year-old man who had not been to a physician in 40 years presented to this institution because he had fallen to the floor and was unable to get up. During the year before this hospital admission, his family reported that he had been gradually slowing down. Over several weeks, he had become progressively edematous, short of breath, and lethargic. He had no earlier medical history and took no medications.

On physical examination, he was found to be obese, unkempt, and only responded intermittently to yes/no questions. His blood pressure was 126/60 mm Hg; pulse rate was 64/min; respirations was 28/min; and temperature was 32.4°C. He had peri-orbital edema, a non-palpable thyroid and no scar on his neck, distant heart and lung sounds, ascites, and massive edema in his mid-chest. His stool was positive for occult blood. Deep tendon reflexes were sluggish, with a markedly prolonged relaxation phase.

Initial laboratory studies revealed the following: a hematocrit of 36.1%, white blood cell count, 6,900/mm^3; normal sodium and glucose levels, with a bicarbonate of 36

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mmol/L. His arterial pH was 7.22; P_{CO_2} was 99 mm HG; and P_{O_2} was 48 mm Hg. A chest radiograph showed cardiomegaly and small bilateral pleural effusions. The electrocardiogram was only remarkable for sinus bradycardia.

A presumptive diagnosis of myxedema with respiratory failure and impending coma was made because of the findings of lethargy, hypothermia, delayed tendon reflexes, and severe respiratory acidosis. He was placed on a regimen of intravenous l-thyroxine (l-T_4), 500 μg, initially, then 100 μg every day, and intravenous hydrocortisone, 100 mg every 8 hours, which was then tapered over a week. He required mechanical ventilation. A pulmonary artery catheter was placed to help manage an infusion of 5% dextrose in normal saline.

Over the next several days he became normothermic, his weight decreased by 40 kg (with the addition of l-T_4 alone, no diuretics were given), his pulmonary function improved such that his pH and P_{CO_2} were within normal limits, and extubation was successful. The thyroid-stimulating hormone (TSH) level was 44 μU/mL, with a free thyroxine level of less than 0.05 ng/dL. Soon after, however, bibasilar pneumonia developed followed by increased gastrointestinal bleeding, the source of which could not be determined by either upper or lower gastrointestinal endoscopy; his condition did not improve with H_2 blocker therapy and transfusions. One month after admission, following an episode of brisk hematemesis, he expired.

At autopsy, he was found to have an atrophic, fibrosed, thyroid gland, bilateral pneumonia, diffuse cardiomegaly, and multifocal ischemic necrosis of the cerebral cortex. The site of his intestinal bleeding could not be determined.

DISCUSSION

This patient presented with coma and hypothermia, two key features of severe myxedema. Myxedema coma (MC) most commonly occurs in the winter months in elderly women who have a long history of hypothyroidism.[3] It is usually precipitated by a stressful event, most commonly a pulmonary infection. Symptoms are those of progressive hypothyroidism, leading to a severe mental slowing, stupor, and eventually, coma and death. In rare instances a paradoxical psychosis, "myxedema madness," may be seen.

As is evident in the patient reported here, the physical examination classically reveals bradycardia, hypotension, hypothermia, and tachypnea. The skin may be dry, scaly, and slightly yellowed or even orange due to hypercarotenemia. A goiter is rare, but a surgical scar on the neck is common, as is sparse or coarse hair, a puffy, edematous face with a thick tongue, a hoarse voice, evidence of pleural or pericardial effusion, ascites, ileus, fecal impaction, and urinary retention. The deep tendon reflexes are absent or slowed with a prolonged relaxation phase, the sensorium are depressed, and generalized seizures may occur in 25%–50% of patients.[4]

Laboratory evaluation of the patient should include a complete blood cell count, serum chemistries, arterial blood gas analysis, electrocardiogram, chest radiograph, bacterial cultures, cortisol level, and TSH, triiodothyronine (T_3) and thyroxine (T_4) levels. Some investigators have also recommended checking antithyroid antibody levels.

Hyponatremia frequently occurs due to decreased delivery of water to the distal nephron, or less often, due to the syndrome of inappropriate antidiuretic hormone production (STADH) or adrenal insufficiency, which occurs in 5%–10% of patients in MC and may cause significant hypoglycemia.[2] The mechanism of such adrenal insufficiency is thought to involve a sluggish response of the pituitary-adrenal axis, characteristic of myxedema, exacerbated by the increased metabolism of cortisol once thyroid hormone replacement is started, or to primary adrenal failure associated with Hashimoto's thyroiditis, the so-called Schmidt's syndrome. Muscle enzymes creatinine phosphokinase and lactic dehydrogenase are often elevated due to muscle cell membrane leaks and delayed clearance. Arterial blood gas studies may show hypoxia due to congestive heart failure or pneumonia, and CO_2 retention with respiratory acidosis due to alveolar hypoventilation, which is a function of both a decreased hypoxic ventilatory response and diminished response to hypercapnia seen in myxedema.[5] Typical electrocardiographic findings are sinus bradycardia, low voltage caused by obesity or pericardial effusions, and non-specific ST- and T-wave changes. The chest film is particularly important, as many patients with MC have occult pneumonia.

TSH will be high in cases of primary hypothyroidism and low in secondary causes, whereas thyroid hormone levels will be low in both. This patient actually had normal electrolyte, glucose, and muscle enzyme levels, but did have a sinus bradycardia and respiratory acidosis.

MC is a true medical emergency and requires prompt therapy. Treatment can be broken down into supportive care, hormonal replacement, and treatment of precipitating events.

The initial management of severe myxedema includes the establishment and maintenance of a proper airway, assurance of adequate ventilation, and treatment of hypotension and shock. The myxedematous patient may have an excess of total body water and extracellular fluid but may have intravascular volume depletion. Fluids may be given cautiously and vasopressors used, often aided by the placement of a pulmonary artery catheter, although the hypotension of myxedema often responds to replacement of thyroid hormone alone. Hyponatremia in MC usually corrects with simple fluid restriction, although if symptoms are severe, hypertonic saline solutions may be needed. The hypothermic patient should be rewarmed passively, as active rewarming may promote peripheral vasodilation and circulatory collapse.

Hormonal management of MC can be divided into treatment of adrenal insufficiency and replacement of thyroid hormone. Although adrenal insufficiency is estimated to occur in only about 5%–10% of patients with MC, it must be presumptively treated in all cases, as the patient cannot await a formal evaluation of adrenal function. Glucose-containing solutions and corticosteroids should be given, most commonly hydrocortisone: 100 mg intravenously every six hours (some investigators recommend a "loading dose" of 300 mg), which is later tapered as the patient's condition improves.

Thyroid hormone replacement is essential and must be instituted as soon as the clinical diagnosis is made. It should not be delayed pending laboratory confirmation. Most endocrinologists recommend l-T_4 because it can be given intravenously; although T_3 has the theoretical advantages of rapid onset of action, heightened metabolic activity, and shorter half-life, it must be given orally, for example, by nasogastric tube. Normal conversion of T_4 to the active hormone T_3, which occurs in a healthy individual may be impaired in the myxedematous patient. Thus, some physicians recommend giving a combination of T_4 and T_3 in MC. The standard approach to giving T_4 is to begin with a loading dose, most often 400–500 μg intravenously, although some studies suggest that the higher dose may be more dangerous. Werner[2] recommends a loading dose of 4 μg/kg T_4 (200–300 μg for most patients) followed by 100 μg more in 24 hours. From there, the dose is tapered to 50–100 μg intravenously each day for the next several days and finally to an oral maintenance dose, as the patient's condition allows. The usual dose of T_3 when given in combination with T_4 is 25 μg orally or via a nasogastric tube every 12 hours.

Any precipitating factors, such as infection, must be meticulously diagnosed and treated. As demonstrated by this patient, in spite of our vastly improved diagnostic techniques and methods of medical support, the diagnosis of MC still carries a mortality rate of 50%–60%, caused by the frequent complications of infection, heart failure, gastrointestinal bleeding, and hypoxic brain injury.[6] As new discoveries are made, we hope to reduce this number, but it is unlikely that any one person will have as great an effect on the treatment of myxedema as did Dr George Redmayne Murray with his "liquor thyroidei" during an English winter 100-years ago.

REFERENCES

1. Major RH: *Classic Descriptions of Disease*. Charles C Thomas, 1939, pp 289–293.
2. Ingbar SH, Braverman LE: *The Thyroid*, Philadelphia, JB Lippincott Co., 1986, pp 1227–1233.
3. Hamburger S, Collier RE: Myxedema coma. *Ann Emerg Med* 1982; 11:156–159.

4. Sanders V: Neurologic manifestations of myxedema. *N Engl J Med* 1962; 266:547–552, 599–603.

5. Massum RA, Winnacker JL: Severe depression of the respiratory center in myxedema. *Am J Med* 1964; 36:876–882.

6. Lemarquand HS, Mausmann W, Hemstead EH: Myxedema as a cause of death. *BMJ* 1953; 1:704.

Occult breast cancer

A case report and review of the literature

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In 1907, Halsted first described three patients with extensive involvement of the axillary nodes by tumor with no apparent primary.[1] All three patients subsequently developed a cancer of the ipsilateral breast more than two years later. In 1909, Cameron[2] described additional cases and concluded the following:

> Every surgeon ought, therefore, to operate as soon after the discovery of a mammary tumor as he can, removing the whole contents of the armpit and if there be no serious reason to the contrary, one or both pectoral muscles as well. But it is equally his duty to remove the entire mamma when operating upon an axillary tumor which appears to be cancerous. . .even though he can discover no tumor in the breast.

More than 80-years later, this approach is still considered by many the standard treatment of this disease. Since these initial descriptions, this problem has only rarely been reported. In 1954, Kaplan reviewed 43 such cases from the literature,[3] although in 1975 only approximately 100 cases had been reported.[4]

This entity represents a diagnostic and therapeutic challenge. We describe a case recently seen at our institution and review the current literature on this problem.

CASE REPORT

A 48-year-old, white premenopausal female was admitted to the hospital complaining of a right axillary mass. Routine laboratory tests revealed only mild microcytic anemia (hematocrit 38%). A two-view mammogram and a chest roentengram were normal. An excisional biopsy was performed. Frozen section examination of the specimen revealed an axillary lymph node with poorly differentiated adenocarcinoma. The patient underwent a level I, II, and III axillary node dissection during the same operating session. Metastatic cancer was found in an additional two of the 15 resected lymph nodes. Final pathological examination confirmed the diagnosis of a poorly differentiated metastatic adenocarcinoma, consistent with a breast primary (Fig 1). The estrogen and progesterone receptors were negative.

The patient underwent a work up outside our institution including a computed tomography (CT) scan examination of the chest, abdomen and pelvis, an upper and lower endoscopy, a bone scan, and a dilatation and curetage, which were all negative.

Subsequently, the patient underwent a right total mastectomy at our institution. Histological examination of the specimen failed to demonstrate a focus of carcinoma.

Postoperatively, the patient has been treated with a six-month course of chemotherapy (cyclophosphamide) 100 mg/m^2, methotrexate 40 mg/m^2, 5-fluorouracil 600 mg/m^2. She is well without evidence of disease 12 months after surgery.

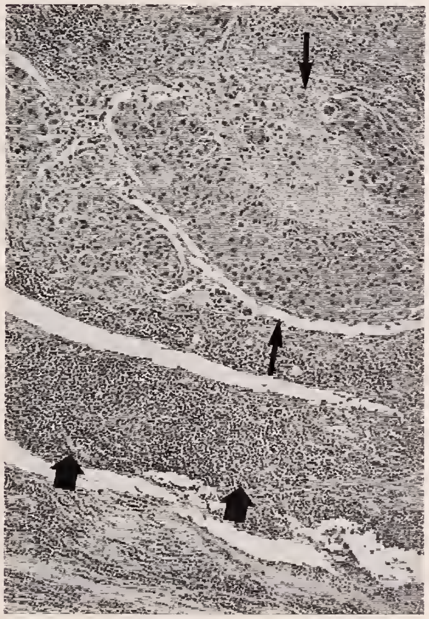

FIGURE 1. Metastatic poorly differentiated adenocarcinoma (long arrows) to axillary lymph node (short arrows) (hematoxylin-eosin; original magnification × 125).

DISCUSSION

Axillary lymphadenopathy is a common problem. In most cases, however, the etiology is benign.[5] Lymphoma is the most common malignancy found in the axillary nodes,[5] but breast cancer as well as other malignancies, such as carcinoma of the lung, thyroid, stomach, colon and rectum, pancreas, in addition to sarcomas and melanomas, may also metastasize there.[6]

Breast cancer presenting as an axillary node metastasis from an occult primary is uncommon, representing less than 1% of all breast cancer cases.[7,8,9,10,11] Most patients are postmenopausal[10,11] and about 20%–30% of them are associated with a positive family history of breast cancer.[10]

Pathologic studies have revealed three histological patterns.[12] Type I is composed of sheets of large pleomorphic cells, with large nuclei and prominent nucleoli. This is the most common type and the most difficult to differentiate from non-mammary adenocarcinoma, malignant melanoma, and undifferentiated lymphoma, requiring special staining techniques. Estrogen and progesterone receptors analysis may confirm the diagnosis. However, their absence does not rule out a breast primary source, because in 40% of the cases, these studies are negative.[10,13] Type II is easily recognized as typical of breast cancer, demonstrating glandular, cribriform and papillary patterns, and comedonecrosis. Type III shows a mixture of the two previous patterns. One occasionally perplexing problem is the differentiation of primary cancer of the tail of the breast from a metastases to a lymph node.

After a mastectomy, a primary breast cancer will be found in the specimen in about two thirds of the cases (Table I). In the remaining one third, no other primary site is revealed on subsequent work-up and after follow-up. However, many of these series include patients from an era before the advent of routine mammography; therefore, some of these cases may not represent truly occult breast cancers by today's standards. In approximately 10%–20% of the cases, the only lesion found is an in situ cancer.[10] This is in contrast to the reported 1% incidence of axillary metastases in series of nonpalpable in situ carcinoma of the breast.[17]

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TABLE I. Incidence of Cancer in Mastectomy Specimens

Author		*n*	Positive (%)
Owen[7]	1954	27	20(74)
Feuerman[14]	1962	10	7(70)
Fitts[8]	1963	13	11(85)
Westbrook[15]	1971	18	9(50)
Ashikari[4]	1976	34	23(68)
Patel[19]	1981	29	16(55)
Kemeny[16]	1986	11	5(45)
Baron[10]	1990	33	22(67)
Ellerbroek[11]	1990	13	1(8)
Total		188	114(61)

Once the diagnosis of occult breast cancer has been made, the question is what to do next. Since breast cancer is both the most likely and the most treatable primary, an extensive work-up is unnecessary, expensive, and confusing, unless specific concerns are raised after a thorough history and physical.[16,18,19] This is well demonstrated by the case we report here. Our patient underwent a full battery of diagnostic tests for a total charge of $8,170. The only result was a false-positive CT scan which lead to an unnecessary D & C. Kemeny et al[16] reported 20 women with this problem who underwent 74 radiographic tests. Only 7% were considered truly positive, whereas 15% were false-positives leading to the diagnosis of a colon cancer in one patient. Therefore, the only useful radiologic investigation in this setting is mammography, even though its limitations have been well stressed by Baron et al, who reported a sensitivity of 29%, a specificity of 73%, and an overall accuracy of 44%.[10] However, in our present medical-legal climate, most clinicians find it difficult to perform a mastectomy without absolute proof of the source of the metastatic disease.

The treatment of occult breast cancer remains controversial. Since the original report by Halsted,[1] patients have been assumed to have an ipsilateral breast primary and have therefore been treated with mastectomy. The survival has approximated that of other stage II breast cancers, even when no primary is found in the mastectomy specimen. However, in the last decade, as the psychosocial impact of mastectomy has become more apparent, this approach has been challenged by proponents of more conservative surgery.

Radical radiotherapy as treatment for occult breast cancer was first reported by Vilcoq et al from the Institute Curie, Paris, in 1982.[9] The treatment consisted generally of node resection or axillary dissection plus radiotherapy. This experience, which included 31 patients has been recently updated.[20] After a median follow-up of nine years, loco-regional failure was unfortunately noted in 25% of patients. Half of these recurrences were in the breast alone and were salvaged by secondary mastectomy in three out of four patients. The other half recurred in regional lymph nodes. These patients usually recurred earlier (mean time to relapse 23 months versus 112 months) and died with disease. Nine patients developed distant metastases. The overall five and 10 year survival was 76% and 71%, respectively. Cosmetic results were reported to be good in 79% of cases. Similar results have been published recently.[11,22] In the MD Anderson series of 42 patients,[11] 69% did not undergo mastectomy. The risk of breast relapse at five years was 17% and 57% in the irradiated and observed groups, respectively. There was no evidence of survival difference between patients who had primary radiation and those who had mastectomy. Further surgical treatment satisfactorily dealt with seven of ten local recurrences in the breast conservation group (Table II).

TABLE II. Mastectomy *v* Breast Conservation

Treatment		*n*	Percentage Survival†
Mastectomy	1988	11	55
Kemeny[16]			
Baron[10]	1990	28	77
Ellerbroek[11]	1990	13	65
Breast Conservation			
Kemeny[16]	1988	7	55 (AND ± RX)
Campana[20]	1989	31	76 (AND ± RX)
Baron[10]	1990	6	65 (RX ± AND)
Whillis[21]	1990	20	66 (RX)
Ellerbroek[11]	1990	29	75 (AND ± RX)

* Five-year survival.

† AND indicates axillary node dissection; RX indicates radiation therapy.

The overall ten year survival for patients with occult breast cancer is 50%–71%.[10,11,16,20] Survival has not been shown to be dependent on the primary cancer being found in the mastectomy specimen.[10,19] Both the nodal and the estrogen receptor status have been shown to be two major prognostic variables.[10,11,19]

The role of adjuvant systemic therapy in this setting has never been investigated in a prospective randomized fashion. There are two retrospective studies that have looked at this issue.[10,11] In both of them, the number of patients was small and no statistically significant advantage was noted with chemotherapy. However, the most reasonable recommendation is still that these patients be treated like other stage II breast cancer patients, with adjuvant chemotherapy. Furthermore, since patients with negative ER-receptors generally have a poor survival, this prognostic factor, along with others like flow cytometry, may be useful in determining the need for adjuvant therapy.

In summary, occult breast cancer is a rare problem, representing less than 1% of all breast cancers. A woman with this problem should be considered to harbor an occult primary in the ipsilateral breast unless proven otherwise. However, in this clinical setting further diagnostic workup is unnecessary. After mastectomy, a primary cancer is found in only two thirds of the specimens.

The management of occult breast cancer is controversial. Primary radiation therapy to the breast with axillary node dissection is an effective method of treatment if one is willing to accept a high local recurrence rate, since survival is comparable to that achieved by mastectomy. However, mastectomy remains an effective way to attain good local control, while obtaining survival comparable to other stage II breast cancers.

Survival has not been shown to be dependent on whether the primary cancer in the mastectomy specimen is found. The nodal and estrogen receptor status are the major prognostic variables. In the absence of good prospective randomized data, it is reasonable to treat these patients similarly to other patients with stage II disease, with adjuvant chemotherapy.

Acknowledgment. This paper is dedicated to Manuela Fortunato born at Flushing Hospital Medical Center on September 30, 1991.

REFERENCES

1. Halsted WS: The results of radical operations for the cure of carcinoma of the breast. *Ann Surg* 1907; 46:1–19.
2. Cameron HC: Some clinical facts regarding mammary cancer. *Br Med J* 1909; 1:577–582.
3. Kaplan IW, Rhinestine H: Occult carcinoma of the breast. *Am Surg* 1954; 20:575–582.
4. Ashikari R, Rosen PP, Urban, et al: Breast cancer presenting as an axillary mass. *Ann Surg* 1976; 183:415–417.

5. Pierce EH, Gray HK, Dockerty MB: Surgical significance of isolated axillary adenopathy. *Ann Surg* 1957; 145:104–107.

6. Copeland EM, McBride CM: Axillary metastases from unknown primary sites. *Ann Surg* 1973; 178:25–27.

7. Own HW, Dockerty MB, Gray HK: Occult carcinoma of breast. *Surg Gynecol Obstet* 1954; 98: 302–309.

8. Fitts WT, Steiner GC, Enterline HT: Prognosis of occult carcinoma of the breast. *Am J Surg* 1963; 106:460–463.

9. Vilcoq JR, Calle R, Ferme F, et al: Conservative treatment of axillary adenopathy due to probable subclinical breast cancer. *Arch Surg* 1982; 117:1136–1138.

10. Baron PL, Moore MP, Kinne DW, et al: Occult breast cancer presenting with axillary metastases. *Arch Surg* 1990; 125:210–214.

11. Ellerbroek N, Holmes F, Singletary E, et al: Treatment of patients with isolated axillary nodal metastases from an occult primary carcinoma consistent with breast origin. *Cancer* 1990; 66:1461–1467.

12. Haupt HM, Rosen PP, Kinne DW: Breast carcinoma presenting with axillary lymph node metastases. *Am J Surg Path* 1985; 9:165–175.

13. Grundfest S, Steiger E, Sebek B: Metastatic axillary adenopathy: use of estrogen receptor protein as an aid in diagnosis. *Arch Surg* 1978; 113: 1108–1109.

14. Feuerman L, Attie JN, Rosenberg B: Carcinoma in axillary lymph nodes as an indicator of breast cancer. *Surg Gynecol Obstet* 1962; 114:5–8.

15. Westbrook KC, Gallager HS: Breast carcinoma presenting as an axillary mass. *Am J Surg* 1971; 122:607–611.

16. Kemeny MM, Rivera DE, Terz JJ, et al: Occult primary adenocarcinoma with axillary metastases. *Am J Surg* 1986; 125:43–47.

17. Rosen PP: Axillary lymph node metastases in patients with occult noninvasive breast carcinoma. *Cancer* 1980; 46:1298–1306.

18. Osteen RT, Kopf G, Wilson RE: In pursuit of the unknown primary. *Am J Surg* 1978; 135:494–498.

19. Patel J, Nemoto T, Rosner, et al: Axillary lymph node metastases from an occult breast cancer. *Cancer* 1981; 47:2923–2927.

20. Campana F, Fourquet A, Ashby MA, et al: Presentation of axillary lymphadenopathy without detectable breast primary (TON1b breast cancer): experience at Institut Curie. *Radiat Oncol* 1989; 15: 321–325.

21. Whillis D, Brown PW, Rodger A: Adenocarcinoma from an unknown primary presenting in women with axillary mass. *Clin Oncol* 1990; 2:189–192.

FROM THE LIBRARY

BACTERIAL ALLERGY, A MISNOMER

Dr Marion T Davidson, of Birmingham, tells us that bacterial products are not allergens and that bacterial allergy is a misnomer.

This is agitation in a very dusty corner indeed and leads to the stirring of old bones and some very sharp searching. But we doubt that Dr. Davidson has found much of value.

His discussion is limited to definitions. Thus, allergy is defined as "a condition of unusual hypersensitivity to a substance which is harmless in similar amounts to the majority of members of the same species." Anaphylaxis, on the other hand, is defined as "a changed reaction capacity which the human or animal gains through treatment with foreign substances." These definitions do not differ basically and accumulated evidence indicates that probably the same reaction mechanisms in the animal body are responsible for specific reactions however classified.

Nevertheless, the characteristic symptoms of human allergy may justify their separate classification. These symptoms are related to the character of the antigens involved and the path of entry into the animal body. Unfortunately, the borderline between the allergic state and anaphylaxis is not always sharply defined, particularly in food allergy where sensitization occurs through the digestive tract.

Bacterial antigens certainly do not ordinarily give rise to symptoms characteristic of those commonly encountered with allergens.

But a hypersensitive state with characteristic local symptoms may be induced in animals by bacterial products in the absence of detectable circulating antibodies. It seems likely that in various infectious diseases some of the symptoms and possibly the variations in susceptibility to infection may be associated with a hypersensitivity fundamentally identical with the common allergies.

Certainly, existing evidence does not warrant the conclusion that bacterial allergy is a misnomer.

SIDNEY W. GROSS, MD
(*NY State J Med* 1946; 46:2025)

LETTERS TO THE EDITOR

Address correspondence to Editor, New York State Journal of Medicine, *420 Lakeville Road, Lake Success, NY 11042. Letters should be typed double-spaced and include the signature, academic degree, professional affiliation, and address of each author. Preference is given to letters not exceeding 450 words, and every effort will be made to assure prompt publication after editorial review. All letters are personally acknowledged by the Editor.*

In support of the *New York State Journal of Medicine*

TO THE EDITOR: I write to defend the integrity of the *New York State Journal of Medicine.*

For personal background, I am a long-time member of the Medical Society of the State of New York (MSSNY), including the Otsego County Medical Society (President 1977–1978) and have been active in MSSNY regularly through membership in the Continuing Medical Education Committee (1978–1989), the Educational Program Committee (1980-present), and as a member of the Associate Editorial Board of the *Journal* since 1984. I am a clinician (gastroenterologist) and medical educator involved in the teaching of medical students and house staff, with an aim and a hope of convincing at least some of these young physicians to remain in New York State.

I believe that to change the near-century old, peer-reviewed, respected *Journal* to a "glossy" publication related principally to medical economic and other non-academic issues is ill-advised, for whatever current economic expediency exists.

In my view, the *Journal* represents the heart and conscience of what the Society should continue to stand for—proof to the public that physicians are scholarly and thoughtful and not simply out to protect themselves. In addition, the *Journal's* current format also provides such a model for the aspiring, not-yet-jaded, young physicians mentioned above. I suspect that the House of Delegates also thought along similar lines when it called for continuation of the present format of the *Journal* under Dr Imperato's outstanding guidance. I suspect that a high proportion of the MSSNY membership (who are all yearly told that $30 of their annual dues are specifically slated for the *Journal*) would express similar views if given a proper format for their expression, views which are presumably and democratically, mirrored by the House of Delegates.

I believe that, rather than to destroy the scientific validity of the *Journal*, MSSNY should constructively seek to assist the editor and his staff in mitigating the financial burden which the *Journal* is said to impose on the Society.

In any event, I do hope that the Council will make no attempt to shortcut the will of the House of Delegates in this important matter which surely needs continued, wide, and open discussion before changes are made, changes which could reflect poorly on the Society long after the present leadership has passed.

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A plea for keeping the *New York State Journal of Medicine* alive

TO THE EDITOR: The recent article written by Dr Gordon[1] reported important information concerning the future of the *New York State Journal of Medicine.* On behalf of many other readers, I would like to express my own views on the importance of the *Journal.*[2] Since my first publication in the *Journal*, I have been a regular reader. Over the years, I have published a few more articles in the *Journal.*[3–6] The published articles represent a wide variety of medical specialties. Therefore, any New York physician can find a column interesting to read regardless of his or her primary specialty. In addition, being listed in the *Index Medicus* has made the *New York State Journal of Medicine* the second leading journal supported by a state medical society, after the *New England Journal of Medicine*.

Ceasing publication of such a reputable journal would be a great loss and would certainly be unacceptable to most readers. Replacing the journal with a throw-away tabloid, focusing on political, regulatory, and government issues, would not serve the needs of most society members who want to read a high-quality, scientific, and peer-reviewed publication. Those members who value the *Journal* as a major benefit of society membership may in fact resign in response to receiving a throw-away tabloid in return for their dues. This would have serious financial and other consequences for the society.

In summary, I submit a plea to support the conclusions of the Committee on Publications, Library, and Archives and the *Journal's* superb editorial staff and to help them continue their efforts to publish a high-quality, peer-reviewed scientific and clinical journal in an efficient and cost-effective manner. Any departures from the current style of the *Journal* will cause a great loss of readers and would fail to serve the needs of the members of the Medical Society of the State of New York. Efforts should be directed toward improving the current journal, but not to eliminating or downgrading.

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1. Gordon M: An update on the *New York State Journal of Medicine. NY State J Med* 1992; 92:293–294.
2. Canver CC: Chlorpromazine in experimental gastric ulcers induced by cold-restraint stress. *NY State J Med* 1985; 85:101–102.
3. Canver CC, Upson JF: Gastrointestinal hemorrhage secondary to colonic varices. *NY State J Med* 1985; 85: 604[6]05.
4. Canver CC, Upson JF: Gastrointestinal hemorrhage caused by colonic varices. *NY State J Med* 1986; 86:207.
5. Canver CC: Spontaneous esophageal perforations: "Boerhaaves's syndrome." *NY State J Med* 1986; 86:310–311.
6. Canver CC, Sekovski B, Taheri P, et al: Acute valvular incompetence secondary to blunt trauma. *NY State J Med* 1992; 92:445–446.

Memories of an internship

TO THE EDITOR: Dr Leo Koven's reminiscences[1] of his internship year at the Jewish Hospital of Brooklyn in 1943 is profoundly evocative and moving to someone who preceded him by somewhat less than a year and shares many of those same memories.

I would like to contribute a few footnotes to Dr Koven's memoir. He makes reference to Dr Rudolph Nissen's illustrious patient, Albert Einstein. As it happened, I was a rotating intern on surgery during Einstein's hospitalization. We were told that Einstein had chosen Dr Nissen in preference to any and all the better known (to us Americans) US surgeons, on the basis of their shared background and language. Patient and surgeon reportedly had an understanding between them preoperatively that if Dr Nissen encountered a problem that might entail substantial morbidity (as was the case with abdominal aneurysm in those days of limited technology for the repair of vascular lesions), Dr Nissen would elect not to do a definitive repair. Such indeed turned out to be the case, and Einstein left the hospital with his aneurysm uncorrected. We were all charmed and awed by his modest, self-contained, undemanding nature. We marvelled that he chose the smallest private room with the fewest amenities and was often seen walking down the corridor to take his bath. In person, he looked exactly like his photographs except for the fact that he was dressed in pajamas.

Another memorable encounter for me, as for Dr Koven, was my month's rotation on neurosurgery. Like everyone else, I was totally overwhelmed by Leo Davidoff: his aloof, austere demeanor; his awesome surgical skill; his profound knowledge of his specialty. Bedside rounds were conducted daily according to his strict protocol: He walked at the head of a long, orderly procession of neurosurgeons, neurologists, chief residents, and interns, each ones place in line fixed by rank. I, the lowliest intern, would normally have been last, except that because of my highly legible handwriting, I was commanded to walk alongside Dr Davidoff at the head of the line so that he could dictate and I could record the progress notes on each patient.

My years as an intern, resident, and fellow in pediatrics led to an extended professional relationship with Dr Benjamin Kramer. He had come to the Brooklyn Jewish Hospital from Johns Hopkins, having already acquired a national reputation for his seminal work in Vitamin D and calcium metabolism. Dr Kramer not only schooled us well in clinical pediatrics, but also encouraged some of us to choose a subspecialty and spend an additional year or more exploring our research capability before venturing into the larger world of clinical pediatrics.

These men, along with pathologist Max Lederer (Lederer's anemia), pathologist/hematologist Alexander Wiener (associate of Landsteiner in the original identification and discovery of the genetic transmission of the major blood groups) brought great distinction to the institution and genuine depth to our post-graduate education.

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[1] Koven JJ: Memories of an internship in 1943. *NY State J Med* 1992; 92:307–312.

TO THE EDITOR: I enjoyed reading Dr Koven's article "Memories of an Internship in 1943," published in the July issue of the *Journal*.[1]

It is with nostalgia that I think of my residency in anesthesiology at the Brooklyn Jewish Hospital in 1942. Bert Hershenson, an early diplomate of the American Board of Anesthesiology, was in charge. He was an outstanding scientist, who in 1943, accepted an appointment to Harvard University and as Director of Anesthesiology at the Boston Lying In Hospital. There is where he published his book, *Obstetrical Anesthesia*. His warning to surgeons against administering their own spinals, seemed timely, so was his plea for a team approach in the operating room.[2]

The mustachioed William Linder was awe-inspiring to us residents. He was a skillful surgeon and like others of his era, he administered spinal anesthesia on his own patients. His skill was quite remarkable, since he was born in 1873 and was ready to retire.

The Second World War caused shortages and dislocations of families. A feeling of patriotism infused all of us, especially those on the staff who were recent immigrants. We were anxious to join the Army. After obtaining my citizenship certificate in 1943, I was inducted into the Army Medical Corps and spent the rest of the war in the Pacific Theater, in charge of anesthesia for the 98th Evacuation Hospital. My most memorable event occurred when Hideki Tojo, the former Japanese Prime Minister, was put in front of me in the operating room in Tokyo after his suicide attempt.[3]

Leo M. Davidoff, MD, an outstanding neurosurgeon, an expert on acromegaly and a scholar, was also a warm human being. As I walked with him frequently from the subway to the hospital, I found out that his walking through wintery snow banks was inspired by the need to conserve gasoline to help with the war effort.

The ravages of tuberculosis in the pre-war population from Europe gave rise to surgeons who became expert in its surgical management. Rudolph Nissen was a born surgeon. He was extremely talented, as evidenced by some procedures which still bear his name. His expert use of local anesthesia for thoracic procedures was often welcome in an era when hopelessness often surrounded tuberculosis.

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[1] Koven L: Memories of an internship in 1943. *NY State J Med* 1992; 7:307–312.
[2] Hershenson BB: *Obstetrical Anesthesia*, Springfield, IL, Charles C. Thomas, 1955.
[3] Spielberger L: Razor or a rogue, *Survey of Anesthesiology*, 1983; 27:255–257.

Waiting for the doctor

TO THE EDITOR: I enjoyed reading Jacqueline Galloway's article, "Waiting for the Doctor," which appeared in the June issue of the *Journal*.[1] She certainly writes well. Her comments about the John Wayne Cancer Institute are very kind, but we have taken to heart the last part of her article describing how to avoid waits.

Waiting is a terrible problem at the Institute and at other cancer centers. Part of the problem is caused by how busy we all are, part by poor scheduling, and part by the emotional nature of cancer and of breast problems that often requires considerably more time and attention than anticipated. We have taken Ms Galloway's suggestions seriously and are attempting to improve our scheduling and may be able to have more time to deal with our patient's emotional concerns. We have initiated a psychosocial support service that addresses individual needs and crises as they arise.

I further appreciated Ms Galloway's perceptive observations of the ambiance at the John Wayne Cancer Institute. We have tried very hard to have our clinical area reflect our concern for our patient's total well being and offer a sense of security and warmth. Her comments are also most timely. We are designing a new Comprehensive Breast Center and are currently budgeting for decor and interior design. Ms Galloway's article is helping me to show the importance of health-care facilities' ambiance and decor to those who control the budget. Her writing will affect the care of other women treated here.

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[1] Galloway J: Waiting for the doctor. *NY State J Med* 1992; 92:262–263.

History: The great reminder

TO THE EDITOR: In the September 1992 issue of the *New York State Journal of Medicine*, Dr Edson reviewed the history of the Long Island Hospital and State University of New York Health Science Center at Brooklyn.[1]

His references to Austin Flint, MD, LLD, Professor of the Theory and Practice of Medicine, and Frank Hamilton, AB, AM, MD, LLD, Professor of the Principles and Practice of Surgery, remind us of the connection of these distinguished clinicians and teachers with upstate New York. They were both active in establishing the University of Buffalo College of Medicine in 1846.

The first we learn of Frank Hamilton was as a member of the faculty of the College of Physicians and Surgeons of the western district of New York at Fairfield, which was founded in 1809, the second medical school in New York State and the sixth in the country at large. When it closed, Hamilton joined the medical faculty at Geneva, New York, in 1840. In 1846, he went on to Buffalo to become one of the founders of the College of Medicine, and then in 1858, he became Dean and the first Professor of Surgery at Long Island College Hospital Medical College.

During Fairfield Medical Department's existence, 555 received the MD degree. As centers of population moved westward, student enrollment declined, and in 1840 with removal of John Delemater, James Hadley, and Frank Hamilton to Geneva Medical College, the College of Physicians and Surgeons of the western district of New York closed. In 1871, the Geneva Medical Faculty transferred to Syracuse and became a nucleus of Syracuse University College of Medicine, now the State University of New York Health Science Center at Syracuse.

I want to thank Dr Edson for recalling past events and people. Frank Hamilton might be likened to peripatetic professors described by Harvey Cushing as pioneers.

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[1] Edson JN: History: The great reminder–The relationship between the Long Island College Hospital and State University of New York Health Science Center at Brooklyn. *NY State J Med* 1992; 92:392–394.
[2] Cushing H: Pioneer medical schools of central New York. An address delivered at the centennial celebration of the College of Medicine of Syracuse University. Syracuse University Press, 1934.

In reply. I was delighted to have Dr Kenneth W. Wright respond to my brief historical article about the Long Island College Hospital which appeared in the September issue of the *Journal*.[1]

Of further interest is the fact that in 1861, while still teaching at The Long Island College Hospital. Dr Austin Flint joined the staff of the Bellevue Medical School and taught there as well.

In fact, the fascinating history of The Long Island College Hospital, the first hospital in this country from which a medical school developed, and my long association with this institution has led me to document its course through the years in book form, soon to be published. In it are biographical sketches of both Dr Austin Flint and Dr Frank Hamilton.

Thank you, Dr Wright, for your additions to our history.

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1. Edson JN: History: The great reminder–The relationship between the Long Island College Hospital and State University of New York Health Science at Brooklyn. *NY State J Med* 1992; 92: 392–394.

ERRATA

In the November 1992 issue, the artwork on pages 499 and 500 and 501 were inadvertently transposed. Figures 1, 2, and 3 on page 499 should be Figures 1, 2, and 3 on pages 500 and 501, and vice versa.

January 1, 1993, Deadline For Reregistering Medical Licenses

The deadline for reregistration of physician licenses is January 1, 1993. Physicians who have not yet received reregistration applications should immediately contact the Division of Professional Licensing, New York State Education Department, (518) 474-4234. Proof of having completed an approved child-abuse training course must be submitted by those who did not do so the last time they registered. Exemptions from this requirement can be obtained by those physicians who do not treat children. A late fee will be charged to physicians who reregister after the January 1, 1993, deadline.

ESSAY REVIEW

William Alexander Hammond, MD

A 19th-century physician-scientist and entrepreneur

Preserve Your Love For Science. Life of William A. Hammond, American Neurologist. By Bonnie Ellen Blustein. 289 pp, illustrated. New York, Cambridge University Press, 1991. $54.95 (hardcover)

William Alexander Hammond, MD (1828–1900), was a leading 19th-century physician who simultaneously achieved both scientific renown and great financial success within the solo practitioner framework that characterized the medicine of his times. He continues to intrigue historians not least of all because he fell short of achieving greatness. Although he had the vision, energy, dedication, and drive to be a great man, he remained the prisoner of attitudes, weaknesses, and antiquated scientific theories and methods which progressively eroded his reputation even while he was still alive.

Bonnie Ellen Blustein has written the first comprehensive study of Hammond's complicated and intriguing professional life. This was clearly an enormous undertaking given the fact that Hammond left no neat archive of primary source materials that could be easily assessed. Rather, what is known about him had to be quarried in diverse archival holdings and from a broad range of secondary sources. Blustein's meticulous study of a wealth of primary and secondary sources, as well as her superb ability to analyze and interpret Hammond against the changing canvas of 19th-century American medicine places her study in a superior class.

Hammond was born in Annapolis, Maryland, the son of a physician, John Wesley Hammond, and grew up in Pennsylvania. Educated by private tutors and at St John's College in Annapolis, he became an apprentice to a Dr William Holme Van Buren in New York City at the age of 16. He later matriculated at New York's University Medical School from where he graduated in 1848 at the age of 20. After serving at the Pennsylvania Hospital in Philadelphia, he entered the army as an assistant surgeon in 1849. He remained with the army for 11 years, primarily serving in remote posts in Kansas and New Mexico.

As Blustein underscores, his assignments on the western frontier placed him in the favorable position of participating in two leading projects of American science, the natural history surveys of the Academy of Natural Sciences of Philadelphia and the Smithsonian Institution. Always interested in science, he conducted original experiments in physiological chemistry while in Kansas which gained him international respect.

Hammond resigned from the army in 1860, primarily because of financial reasons. He accepted a position as professor of anatomy and physiology at the University of Maryland Medical School and opened a private practice in Baltimore. However, six months later he re-enlisted in the army at the bottom of the seniority list. A short time after that, he was appointed Surgeon General of the US Army (Fig 1) over the wishes of Secretary of War, Edwin M. Stanton. This meteoric rise from low-seniority assistant surgeon was by no means serendipitous as Blustein explains in detail. Hammond's continuous scientific output, energy, and vision, as well as his close connections to the scientific establishment brought him to the attention of the US Sanitary Commission, a group of prominent New York civilians investigating the medical care provided to Union troops. They came to the conclusion that the Medical Department required new leadership and a complete reorganization. Hammond, they believed, was the ideal man to set things straight. He did not disappoint them. He completely reorganized the department, made it more efficient, and encouraged and supported able subalterns, such as Dr Jonathan Letterman. He established the Army Medical Museum and initiated the *Surgical Directory of the Rebellion*.

Hammond was an imposing physical presence, standing over six-feet in height. He had a commanding voice and was not only bright, and energetic, but also pompous, brash, and arrogant. His promotion over others produced immediate enemies within the Medical Department who were further galvanized by his reforms. Hammond, however, was not deterred by the opposition. He enjoyed the full support of the powerful Sanitary Commission, organized medicine, and medical scientists. While bringing increased efficiency to the Medical Department, he also fostered and promoted medical research directly related to the provision of better care for the troops.

Stanton, who had appointed Hammond under duress and out of political necessity, was abrasive, opinionated, and at times unjust. It was Hammond's circular number 6, removing calomel from the army drug list, which gave Stanton his chance. This order infuriated a broad constituency of physicians, manufacturers, and distributors, and placed Hammond in the position of challenging an enshrined component of standard care adhered to by many physicians. Encouraged by the growing ranks of those chaffing under Hammond's reforms, Stanton resolved to rid himself of the Surgeon General by resorting to customary bureaucratic maneuvers aimed at encouraging him to resign. In July 1863, he appointed a commission headed by an old Hammond enemy from Kansas to examine the affairs of the Medical Department. There was little doubt that the commission would find Hammond guilty of something. Eventually they came in with flimsy charges of no merit which Stanton greatly exaggerated in importance in order to get rid of Hammond. Some might have resigned before this point was reached. However, Hammond demanded and got a court martial. With a vindictive enemy as the star witness and Stanton pushing for conviction, the guilty verdict came as no surprise to most.

Dismissed from the army, Hammond returned to New York City where he set up a private practice, specializing in neurology. This was a bold move for its time as few physicians were able to survive economically by limiting themselves to a specialized practice. Hammond, however, had an international reputation as a medical scientist, was widely respected, and had no difficulty quickly building a consultant practice from referrals. His dismissal from the army inflicted virtually no harm on his reputation as most rightly saw him as the victim of a gross injustice.

For almost a quarter of a century, from 1864 until 1888, Hammond combined a private practice in nervous diseases

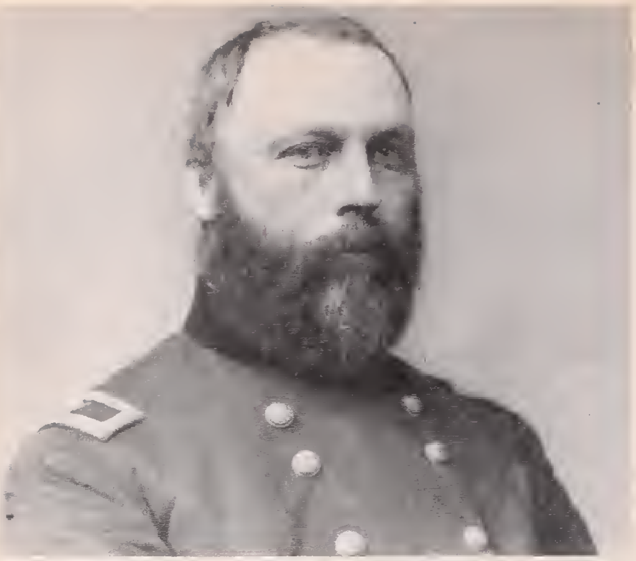

FIGURE 1. William Alexander Hammond at the time that he was Surgeon General of the US Army (courtesy Public Affairs Office, US Army Surgeon General's Office).

with scientific research, the latter largely based on what today would be characterized as anecdotal clinical observation. His annual income rose from $12,225 in 1867 to over $60,000 in 1877. He became a towering figure in American medicine, exerting his influence through scientific organizations, hospitals, and through the medical literature. He founded the *Quarterly Journal of Psychological Medicine and Medical Jurisprudence* and the *New York Medical Journal*. The latter, along with the *Medical Record*, enjoyed a status in the late 19th-century equal to that of the *New England Journal of Medicine* today. His book, *A Treatise On The Diseases of The Nervous System*, first published in 1871, went through several editions until 1898 and became the standard neurological text of its time.

In 1888, Hammond returned to Washington, DC, where he opened Dr William A. Hammond's Sanitarium for Diseases of the Nervous System. There he treated patients, experimented with new surgical procedures and enjoyed a financial reward of some $70,000 annually. By the early 1890s, he was promoting the use of animal extracts such as "cerebrine," "cardine," "ovarine," and others. His basic theory was that these extracts contained the ingredients necessary for the health of the corresponding organs of the body. Isopathy, as this theory came to be known, enjoyed initial support from many scientifically oriented physicians. However, in time Hammond's extracts came to be regarded by many as little more than worthless nostrums. This scarcely deterred him. He established a company to market them and soon became more of an entrepreneur than a medical scientist.

When Hammond died in 1900, Frank Pierce Foster, the revered editor of the *New York Medical Journal*, remarked that "he came very near being a great man."[1] This terse comment perhaps best sums up this unusual physician whose scientific work, as Blustein observes, came to be seen not as mistaken, but as beside the point. Yet, Hammond continues to fascinate medical historians. What rivets many is his court martial and dismissal from the army, more a comment on Stanton's character than a blemish on Hammond's.[2–6]

Flawed as he was by anti-feminist sentiments, a disdain for anti-sepsis, and a willingness to be expedient in order to succeed, Hammond nevertheless represented the best in the intellectual and research traditions of 19th-century American medicine. These traditions were rooted in private consulting practices, specialty societies, and post-graduate schools. They were later displaced by laboratory and clinical research based in medical schools, hospitals, and research institutes. Blustein's excellent study of Hammond's professional life affords insight into this larger canvas of late 19th-century American medicine.

Although Hammond has been the subject of numerous articles, most have focused on his army career and his role in establishing neurology as a specialty.[7] Blustein has now produced the first extensive portrait of his diverse professional life. Derived from a doctoral dissertation produced at the University of Pennsylvania in 1979 under the supervision of Charles A. Rosenberg, she not only chronicles the details of his career, but more importantly places them within the larger context of his times. This was an enormous task, given the dynamic changes surging through medicine in the latter half of the 19th century. She meticulously documents his army career, his tenure as Surgeon General, his dismissal from the army, and his exoneration (1877) by President Rutherford B. Hayes. This fascinating phase of Hammond's career saw him at his best, when he placed medical science above all else in the service of humanity. Yet his real influence on American medicine came during the later clinician-entrepreneur phase of his career when he forcefully projected himself as a scientific organizer, teacher, author, and editor. He was clearly adept at self-promotion, given his rapid and enormous success. As Blustein shows, his reputation as a clinician capable of diagnosing, treating, and curing a wide range of ailments rested not only on his scientific research, but also more importantly on his skills and his use of then novel technologies such as electricity.

Blustein succeeds in presenting Hammond's professional life in great detail and in using it to chart the changing character of medicine at the time. Little is said about his personal life and his family, perhaps because as she observes, "little has been recorded." Colleagues who wrote about him did so with customary 19th-century circumspection. As Frank Pierce Foster said in his obituary tribute in the *New York Medical Journal*, "He was a charming companion, faulty, but it is our conviction, guileless."[1] One can read between Foster's lines, but in the end we are left with an incomplete personal portrait. Thus, the subtitle of Blustein's book, *Life of William A. Hammond, American Neurologist*, speaks primarily to his professional and not to his personal life.

Hammond also exerted enormous influence because he had access to the printed page. Although Blustein covers this in considerable detail, she does not discuss his role in the founding of the *New York Medical Journal* in 1865 (No association with the *New York State Journal of Medicine*).[8] In the bibliography, she lists this journal as having been edited by him between 1865 and 1867 but there is no elaboration on this in the text. This is disappointing given the great importance of this journal to American medicine at that time and later. The bibliography presents Hammond's writings under several headings: monographs, pamphlets and broadsides, works edited, articles in professional journals, translations of works by Hammond, articles written for the general public, and fiction (he wrote eight novels and co-authored a ninth with his daughter). Within these groupings, works are arranged alphabetically. However, in the view of this reviewer, a chronological listing would have been more useful.

From a certain perspective, Hammond is a tragic figure. His scientific thinking became an anachronism even in his own lifetime. Yet, linked as it was to his financial success, he was unable to give it up. Toward the end of his life, medical science passed him by, and as Blustein observes, his animal extracts came to represent not only bad science, but also worse commercialism. Blustein has eminently succeeded in weaving together the complex threads of this physician's fascinating professional life. In so doing, she has produced a meticulously documented and highly readable biography that stands as the definitive study.

PASCAL JAMES IMPERATO, MD
Editor

1. Foster FP: William A. Hammond, MD. *NY Med J* 1900; 71:64.
2. Duncan LC: Days gone by: the strange case of Surgeon General Hammond. *Milit Surg* 1929; 64:98–110, 252–262.
3. Miller MD: William A. Hammond: restoring the reputation of a Surgeon General. *Milit Med* 1987; 152:452–457.
4. Friend HC: Abraham Lincoln and the court martial of Surgeon General: William A. Hammond. *Comm Law J* 1957; 62:71–80.
5. Freeman FR: Lincoln finds a Surgeon General: William A. Hammond and the transformation of the Union Army Medical Bureau. *Civil War Hist* 1987; 33:5–21.
6. Klawans HL: Court martial of a surgeon general. *MD* 1992; 36:103–116.
7. Heck AF: William Alexander Hammond: 1828–1900. *JAMA* 1963; 183: 466–468.
8. Imperato PJ: A history of the *New York Medical Journal. NY State J Med* 1989; 89:403–414.

FROM THE LIBRARY

THE INFLUENCE OF THE ERIE CANAL ON MEDICAL EDUCATION AND PRACTICE IN UPSTATE NEW YORK

The beginnings of modern medicine, modern science, and modern political economics are traced to the nineteenth century. Many factors, such as political and social revolutions, technologic inventions, and scientific discoveries, which had their influences on the rest of the world also had their influences on the State of New York, but the greatest single factor in the development of this State was the building of the Erie Canal.

The need for more physicians kept pace with the growth of settlements into towns and then into cities. Physicians faced with the task of furnishing medical service in these expanding communities followed the traditional custom of training apprentices in their own homes and offices. The shortcomings of this method of training were flagrant and were denounced by Drake and others who, although they had received their own early training under this system, led in the establishment of medical schools with comprehensive curricula. As towns grew, physicians pooled their skills and attempted to organize medical schools. The proprietary medical school became characteristic of nineteenth-century American medicine; it was established not only to meet the needs of the community but for profit to the faculty, who received the fees paid by the students. Competing for larger enrollments of students, the medical schools offered many inducements such as less stringent entrance requirements and shorter terms of instruction. Many of these schools were so transient in their existence they left no records by which to trace their history. The effort to regulate medical schools and licensure was begun by the Medical Society of New York State, and the movement for the organization of the American Medical Association was initiated by that Society in 1847.

IRVING WOLFSON, MD
(*NY State J Med* 1955; 55:2524–2527)

THE ETIOLOGY OF BULIMIA NERVOSA: THE INDIVIDUAL AND FAMILIAL CONTEXT

By Janis H. Crowther, Daniel L. Tennenbaum, Stevan E. Hobfoll, and Mary Ann Parris Stephens. 252 pp. Bristol, PA, Hemisphere Publishing Corp, 1992. $49.50 (hardcover)

These impressive research papers on bulimia nervosa were all previously presented at the Second Annual Kent Psychology Forum, sponsored by the Applied Psychology Center of Kent State University. The participants were educators, psychologists, and research scientists prominent in the field of eating problems and eating disorders. The specific focus was on the etiology of bulimia nervosa. Although many of the questions continue to be unanswered, the book does advance our understanding of this perplexing problem.

The editors have organized the material into three sections: "Developmental and Familial Factors," "Individual Factors," and "Future Directions," reflecting the format of the conference. It should be mentioned that the Applied Psychology Center accepts the American Psychiatric Association's 1987 criteria for bulimia nervosa. This requires the presence of the following symptoms: recurrent episodes of binge eating (two binge episodes a week for at least three months), self-induced vomiting, as well as the use of laxatives and diuretics, vigorous exercise, and a persistent overconcern with body shape and weight.

The consensus of thinking reaffirmed the belief that the cause of bulimia nervosa is multifactorial, consisting of sociocultural, familial, and individual elements. However, the researchers did differ on their emphasis of the causal elements and also recognized that further longitudinal studies are still needed. Although it has been recognized that young women are predominantly at risk, there is now the awareness that men may also be vulnerable. These maladaptive eating attitudes, we learn, already exist in prepubertal school-age children of both sexes.

Although it was stated earlier that many questions concerning bulimia nervosa still remain unanswered, the preventive measures recommended deserve our attention and should not be disregarded. The public health prevention programs to foster more healthy attitudes regarding eating should be instituted today. Also to be considered is stricter monitoring and regulating of the diet and beauty industries. These measures do not have to be postponed until we have achieved a complete understanding of bulimia nervosa.

The editors, Crowther, Tennenbaum, Hobfoll, and Stephens are to be commended on this book. The body of research material is lucidly presented and the appropriate questions raised and discussed. This volume will be a valuable addition to libraries and is recommended reading for the pediatrician, psychiatrist, and public health physician.

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AIDS AND THE LAW: A BASIC GUIDE FOR THE NONLAWYER

By Allan H. Terl, JD. 180 pp. Bristol, PA, Hemisphere Publishing Corp, 1992. $19.95 (softcover)

This book has been published to fill what appears to be a critical gap in the burgeoning acquired immunodeficiency syndrome (AIDS) literature. It attempts to review the legal aspects of human immunodeficiency virus (HIV) disease and AIDS and describe the impact of the disease on many aspects of law.

The author, a lawyer, first began to work on AIDS issues when, as an American Civil Liberties Union (ACLU) chapter leader, he fought the termination of an employee who apparently was discharged because he was diagnosed with AIDS. The author has since devoted almost all of his time to consulting on AIDS-related issues. In the preface, he states that this book is written in the hope "that an understanding of the legal issues relating to HIV disease will help those with specific rights to be aware of them, to muster the courage to insist on them, and if necessary, to pursue them through the courts." The statement outlines the tenor of the book. The author clearly takes an advocacy approach to AIDS within the context of the legal system. Indeed, the pain this epidemic has caused both the author as well as countless others is well detailed throughout this text. It is this same pain, and resultant anger, that diminishes its value as a guide.

The book is divided into 22 chapters. The topics covered include issues of confidentiality, torts, housing, free speech, and prisoner issues. Each chapter is presented in a similar fashion. A series of cases are cited, with a cacophony of editorial comments.

Occasionally, chapters conclude with a series of guidelines from the American Bar Association or some other recognized body. More often, the author simply moves on to the next topic.

Health and social service professionals caring for HIV-infected patients are being confronted by legal issues raised by this disease on a daily basis. Beyond the obvious issues of confidentiality and access to entitlement programs, we are now being challenged by issues related to tuberculosis, AIDS orphans, as well as the challenges raised by concern over transmission of HIV in the health-care setting. These professionals need a clearly written and dispassionate guide to these issues. Unfortunately, this book does not meet this need.

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EARLY DETECTION AND TREATMENT OF LIVER CANCER

Edited by Kunio Okuda, Takayoshi Tobe, Tomoyuki Kitagama. 228 pp, illustrated. Bristol, PA, Taylor & Francis Ltd, 1991. $99.00 (hardcover)

Primary hepatocellular carcinoma, although relatively uncommon in western countries, is one of the most common tumors in the world, with an estimated annual incidence of 500,000 to 1 million cases. It occurs most frequently in parts of Asia and sub-Sahara Africa where incidence rates reach up to 500/100,000 population/year. The tumor is associated with chronic hepatitis B virus infection and possibly with hepatitis C virus infection, as well as with cirrhosis. In spite of a variety of attempted treatment methods, the five-year survival in patients presenting with symptomatic disease is less than 5%. Tumor resistance to chemotherapy and radiation therapy has made effective control of advanced disease elusive. Survival has been prolonged only when lesions are found early and resected when small.

Early Detection and Treatment of Liver Cancer is the 38th volume in the series of Gann Monographs on cancer research promoted by the Japanese Cancer Association. Edited by Kunio Okuda, Takayoshi Tobe, and Tomoyuki Kitagawa, the book's stated purpose is to review the important Japanese studies pertaining to hepatocellular carcinoma, "evaluate their impact on the study of primary liver cancer. . .and introduce them to the world."

Five main sections ("Overview," "Epidemiology," "Pathology," "Diagnosis," "Treatment") are subdivided into 20 chapters. After an initial chapter reviewing recent advances in detection and treatment, individual papers deal with specific aspects of the disease in greater detail. The chapter on epidemiology reviews age factors, geography, and the role of the hepatitis viruses, cirrhosis, aflatoxin, blood transfusion, alcohol intake, and tobacco smoking. Pertinent tables and maps are included, although the latter could have been improved by clearer reproduction. The pathology section reviews in two chapters the morphologic characteristics of early hepatocellular carcinoma and the difficulties involved in making a diagnosis of the disease early in its course. The diagnosis section contains six chapters dealing with screening high-risk groups, and applications of immuno-biochemical diagnostic techniques including α-fetoprotein, des-γ-carboxy prothrombin, novel γ-glutamyltranspeptidase, and variant alkaline phosphatase. The role of ultrasonography (including laparoscopic ultrasound and two-dimensional color Doppler methods) are reviewed, as are angiography, computed tomography (CT) (including lipoidal-CT, angio-CT, and table-sliding, rapid-sequence techniques), and the evolving role of magnetic resonance imaging. The treatment section is composed of nine chapters outlining the successes, failures, and limitations of embolotherapy, ethanol injection, lipiodolization, chemotherapy, and surgery including liver transplantation.

A bibliography follows each chapter. In keeping with the book's purpose of introducing Japanese studies to a wider readership, most of the cited references are for studies done in Japan. In most of the chapters, the references appear pertinent and up-to-date, but I was disappointed to find only 13 references in the chapter on screening high-risk groups, and of these, eight referred to studies reported in 1985 or earlier. The chapter on CT diagnosis of hepatic cancer had but 19 citations, of which 16 were for pre-1985 studies.

More rigorous editing could have removed some of the repetitious information in the separately authored chapters. In general, however, the individual efforts fit together well as a monograph and cover the topic in a thorough fashion. The chapters are usually concise, and cover a specific topic well and are easily understood. A brief concluding chapter to bring all the preceding papers together and to consider future directions for investigating this devastating tumor might have helped the overall effort.

This is a good summarizing monograph for those who wish to review the recent experience with hepatocellular carcinoma in an area of the world where the tumor is a prominent cause of cancer deaths and where experience in treatment has accumulated more rapidly than in the United States. Except for the weaknesses noted, it provides a good review of the subject.

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MODERN MEDICINE AND JEWISH ETHICS

Second Edition. By Fred Rosner, MD. 260 pp. Hoboken, NJ, KTAV Publishing House, Inc, 1991. $29.50 (hardcover)

Dr Fred Rosner's *Modern Medicine and Jewish Ethics* will undoubtedly come to be the definitive work on this subject, one that will be of practical and scholarly use to a wide and diverse audience. A pre-eminent and highly respected medical practitioner who is also well versed in Jewish law and tradition, Rosner is chairman of the Committee on Bioethical Issues of the Medical Society of the State of New York and an expert on Jewish medical ethics. A scholar and prolific author, he is uniquely qualified to discuss matters of medical practice and patient choice from an informed and thoughtful medical and Jewish perspective. The author's dual expertise is in the tradition of Maimonides, the great Jewish philosopher and physician (1135–1204), whose works he has translated and interpreted for a wide audience.

Rosner's second revised and augmented edition of *Modern Medicine and Jewish Ethics* is, like its predecessor, extremely useful for physicians and other health providers, clergy, students, patients and their families, and other interested readers. This compact volume contains 30 chapters appearing under four sections: "Physician and Patient," "The Beginning of Life," "The End of Life," and "General Issues." The author has included a great deal of up-to-date information in 460 pages. This new edition includes chapters on AIDS, the definition of death, skin grafting, pregnancy reduction, fetal therapy, and other topical issues. A number of important features add to the book's value and usefulness. Scholarly notes and an index with plentiful, useful sub-headings are important to those searching for extensive information on a topic or related subjects. For those in search of clear, concise answers to complicated questions, the chapter summaries are invaluable.

No matter what the topic under discussion, a consistent, coherent set of religious and moral principles underlies the author's work. Newly formulated queries are responded to on the basis of these traditional Jewish teachings. Judaism is guided by the concept of the supreme sanctity of human life and of the dignity of the human created in the image of God. All Jewish medical-ethical decision-making is based on this understanding. Furthermore, the preservation of human life is a Divine commandment. Jewish law requires the physician to do everything he or she can to prolong life. To save a life, all Jewish observances are automatically suspended; they are subordinated to the highest goal of preserving human life. The value of human life is infinite and beyond measure.

Rosner's religious and moral orientation is clear throughout his book. "AIDS: A Jewish View" (Chapter 6) demonstrates his ability to deal with complex, controversial issues in a concise, clear, and compassionate manner. He summaries the Jewish view of homosexuality and drug abuse, the obligation of the physician to heal, visiting patients with infectious diseases such as AIDS, allocation of resources for AIDS research and treatment, and various aspects of Jewish ritual as they relate to AIDS patients. He concludes that while Judaism does not condone homosexuality, "we are. . .duty bound to defend the basic rights to which homosexuals are entitled. . . The plight of Jewish AIDS victims doomed to almost certain death should arouse our compassion. Whether a person is a homosexual or not, we are obligated to give him proper care if he is sick, charity if he is needy, (and) food if he is hungry. . . ."

The author presents an important essay (Chapter 18) on "Rabbi Moshe Feinstein on the Treatment of the Terminally Ill." He begins by reviewing the landmark 1986 statement by the American Medical Association (AMA) on the withdrawal of artificial feeding and hydration from terminal or permanently comatose patients under certain conditions. Contrary to the view of the AMA, "Judaism views nutrition and hydration. . .not as medical treatments but as supportive care, no different from washing, turning, or grooming a dying patient." Rosner goes on to present a Jewish approach to the treatment of the terminally ill as discussed in the writings of the late Rabbi Moshe Feinstein, a renowned 20th-century scholar. For example, he presents Rabbi Feinstein's view that "for a patient with pain and suffering, who cannot be cured and cannot live much longer, it is not obligatory for physicians to administer medications briefly, to prolong his life of pain and suffering, but nature may be allowed to take its course."

In "Definition of Death" (Chapter 20), Rosner emphasizes that in Jewish law, death has traditionally been defined by the absence of spontaneous respiration. He concludes that if there is brain stem death, the person is considered dead. In terms of Jewish law, the patient is as if physiologically decapitated. This approach allows for human heart transplantation.

Regarding "Unconventional Therapies" (Chapter 29), the author discusses the patient's responsibility to pursue conventional therapies actively to achieve healing. The use of supplements to traditional medical therapy, such as prayer and amulets, is encouraged if the patient feels that these will be helpful, but these approaches should never be used as substitutes for conventional care. He also acknowledges the significance of the patient's confidence in the treatment being used. This certainly plays a role in not only the physiological, but also in the psychological well-being of the patient. He recognizes that some of the unconventional therapies, although not scientifically validated, may provide a positive placebo effect.

In his "Foreword" to the book's first edition, Lord Immanuel Jakobovits, former

Chief Rabbi of the British Commonwealth and pioneer in the field of Jewish medical ethics, describes Dr Rosner as the "leading medical writer on Jewish medical ethics" and comments on his "wide-ranging erudition." He predicts that the book "will no doubt. . .be hailed as another classic." I can think of no better way to summarize my assessment of the second edition of this work. I would only add that this book will be considered the standard text in the field of Jewish medical ethics. I will certainly consult it frequently.

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PSYCHOLOGICAL ASSESSMENT: PSYCHIATRIC DIAGNOSIS AND TREATMENT PLANNING

By Stephen W. Hurt, PhD; Marvin Reznikoff, PhD; John F. Clarkin, PhD, 492 pp, New York, Brunner/Mazel Publishers, 1991. $57.50 (hardcover)

It is not uncommon in the field of psychiatry for a trainee to present a case by describing a patient's presenting complaint, symptomatology, mental status, history of illness and treatment, assets and achievements, diagnostic formulation, and treatment recommendations. Family-oriented attendings may then ask about whether the patient's social network is maintaining the problem. A psychopharmacologist might consider the regimen of medications that were overlooked. A psychoanalyst might press for a fuller description of the patient's inner life. Each question reflects a desire to expand the presenting clinician's knowledge of the patient so that, at once, a more specific and more comprehensive treatment plan can be devised. The intern or resident looks strangely puzzled because he or she did what was expected, present a DSM-IIIR diagnosis and a treatment plan.

In this book, the authors argue that although the reliability of psychiatric diagnosis has steadily improved with the successive revisions of the DSM system, "DSM diagnosis is necessary but not sufficient for treatment planning" (p 25). In the authors' view, the DSM-IIIR and psychodiagnostic testing offer complementary viewpoints that can add accuracy to the differential diagnosis of psychiatric disorders while providing a combined frame of reference for case formulation and treatment planning. A standard psychiatric interview can provide a descriptive account of the signs and symptoms and a historical picture of the patient. Yet, additional information is often required to develop an optimal treatment strategy. Through the consulting psychologist's judicious selection and administration of structured, standardized assessment tools (eg, traditional instruments such as the WAIS, the MMPI, Rorschach, TAT, and Bender Gestalt) and careful interpretation of the test data, a psychodiagnostic assessment provides an organized framework for efficiently evaluating a patient's cognitive functioning and identifying the patient's sources of conflict, preferred defensive style, and strengths.

Drawing on their considerable experience at New York Hospital's Westchester Division, Hunt, Reznikoff, and Clarkin have carefully selected cases where treatment planning issues were informed by the data and the results of psychological assessments performed by clinical psychologists in the psychiatric hospital setting. The cases are presented in an orderly fashion, with the major diagnostic groupings of the DSM-IIIR represented. Axis I Disorders cover the disorders of childhood and adolescence, schizophrenias, major affective disorders, anxiety disorders, and adjustment disorders. Axis II Disorders include paranoid, borderline, and narcissistic personality disorders. In turn, each diagnostic classification is illustrated by two separate cases to allow for the comparison of test data and interpretation. The format for the case presentations includes the referral questions for the psychological consultation, historical material, test responses, formal scoring, the psychological report, and treatment recommendations. A summary discussion, comparing two cases, highlights differences and similarities regarding diagnostic and treatment decisions.

The strengths of this book are the detailed cases. They are clearly presented and convey the subtle interplay between the patient's history, symptoms, personality, and intellectual functioning. Although the breadth of cases is an asset, it is possible that the authors did not cast their net widely enough. For example, given the prevalence of and difficulties in making Axis II diagnoses, it is surprising that more attention was not paid to these classifications. Common diagnostic questions related to the dually diagnosed patient (psychiatric disorder and substance abuse) are also not addressed. On the other hand, the cases that are included should be of special interest to psychiatrists and psychologists who are working together as primary clinicians in psychiatric settings. They provide one model, among many, of constructive psychologist-psychiatrist collaboration. When faced with thorny diagnostic questions, an approach that draws on the respective skills of both the psychiatrist and clinical psychologist can result in a more comprehensive view of the patient.

Psychologist Assessment, Psychiatric Diagnosis, and Treatment Planning is a solid introduction to how intelligent psychological testing can enhance psychiatric practice. While there is little doubt that psychodiagnostic assessment is good for patient care, it also offers a cost-effective means of gathering essential data that can accelerate the planning of psychiatric interventions. Hunt, Reznikoff, and Clarkin have contributed a useful reference book, the kind that might be pulled down from the shelf when one is scratching one's head about a difficult case and considering the potential advantages of a psychologist's consultation.

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EMT: PREHOSPITAL CARE

By Mark C. Henry, MD, and Edward R. Stapleton, EMT-P. 865 pp, illustrated. Philadelphia, PA, WB Saunders Company, 1992. $25.95 (softcover)

Mark C. Henry, MD, and Edward R. Stapleton, EMT-P, are leaders in prehospital education. Dr Henry and Mr Stapleton are faculty members in the Department of Emergency Medicine at the State University of New York, Health Science Center at Stony Brook. Both have published numerous articles and are well known for using integrative learning techniques in several of their educational programs.

The book consists of 20 chapters with 80 skill procedures integrated into the text. The chapters adhere to the US Department of Transportation's format. All of the chapters begin with an outline of the authors' intended objectives, which can give needed guidance to a new pre-hospital care provider. The case histories, which appear throughout the book, serve to help formulate methodologies for handling the emergency patient. Each of the chapters provide an in depth focus on anatomy and physiology before pathology and management are discussed. Relevant treatment protocols are added as a means of allowing the student to "put together" their knowledge into a plan. The illustrations are detailed, organized, and appropriate in a text of this type. Finally, the review questions at the end of each chapter provide a mechanism for the student to see if the unit objectives were met.

Although the authors clearly define their intended audience (EMT-A, EMT-D, and EMT-I) in the preface, the book really lends itself to the more advanced EMT reader. This may alienate the novice provider or act as a stumbling block to the new basic level student. Perhaps the book would be used more effectively if the advanced material was isolated into separate divisions or characterized as advanced throughout the chapters.

Although most pre-hospital care texts offer very strong sections on trauma and its management, this often comes at the expense of common medical emergencies. In contrast, *EMT Prehospital Care* devotes superior coverage to assessing and treating both the medical and trauma patient. The clinical procedure sheets provide a detailed approach to the skills without offering the commonly found "cook book" format.

Overall, this is a well-written and illustrated text that puts some competition into the sometimes stale and too traditional pre-hospital care text environment.

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THE STATUS OF HEALTH OF BLACKS IN THE UNITED STATES OF AMERICA

By Valiere Alcena, MD. 217 pp, illustrated. Dubuque, Iowa, Kendall/Hunt Publishing, 1992. $26.95 (softcover)

One of the most chilling legacies of the mid 1980s and beyond has been our studied indifference as a society to the health care plight of our forgotten citizens. A kind of moral blindness has taken hold of us. As is often the case, it has taken an immigrant and outsider to go beyond the litany of neglect and prescribe a plan to improve the care of our *favelados*.

Dr Valiere Alcena, whose mother died in childbirth and whose father succumbed to an untreated perforated ulcer, has written of this American tragedy. The story of his rise from a nine-year-old orphan to becoming a hematologist/oncologist in White Plains is a truly remarkable achievement.

The book consists of 11 chapters with special emphasis on those diseases which, without proper treatment, doom African-Americans to premature disability and death. Some of the topics include hypertension, diabetes mellitus, heart disease, cancer, alcoholism, drug addiction, anemia, sexually transmitted diseases, and AIDS. This book is written as his gift to black Americans and is a primer on health care with a decided focus on prevention.

In his introduction, the author draws our attention to the increasing mortality of black Americans and calls our attention to the negative impact of racism and poverty on them. He looks at the Medicaid Program and the Health Insurance Industry and finds them to be part of the problem rather than the solution. He finds the black media is not fulfilling its function to educate the black population with regard to health care. Elected officials and Health and Human Services are also challenged in terms of their performance.

Many of the chapters are illustrated by black and white photographs. The final chapters on sexually transmitted diseases and AIDS would have been enriched by such illustrations. The chapter on fetal alcohol syndrome is too brief and does not enlighten the reader as to the specific problems that cripple these children. The same can be said about AIDS and the neonate. Teenaged pregnancy in the black population is still a major concern and this area does not appear to have been covered. Admittedly, these are the concerns of obstetricians and pediatricians and the author is an internist, but they are of such a compelling nature that their inclusion seems warranted.

This book is a clear exposition of the author's point of view. The organization, style, and technical features are suited to his purpose and the book is easy to read. He has a decided advantage over other authors in that he has lived with the topic. The book has meaning for those who will care for black patients as well as those who want to understand the black health care experience. Valiere Alcena, MD, FACP, has fulfilled his literary mission to educate us in that black health care arena of our, "Two Nations,"[1] black and white, separate and unequal.

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[1] Hacker, A: *Two Nations*, New York, Charles Scribner & Sons, 1992.

WOMEN AND AGING: CELEBRATING OURSELVES

By Ruth Raymond Thone, 152 pp. Binghamton, NY, Harrington Park Press, 1992. $9.95 (softcover).

The author has written this book to focus on the relevant issue of the aging process in women and offers suggestions on how to go about it gracefully and successfully. She uses her own personal experience as a teacher and writer and as an intuitive woman who has worked with many other women. She also draws on a comprehensive, multidimensional bibliography of other well-known women, poets, mystics, therapists, and religious writers.

She presents a number of brief, essay-like chapters on a variety of topics. She explores the feelings, the fears, and the anxieties related to aging. She encourages self recognition and a sense of self, and she disavows conformity to the stereotypes of previous generations. She offers a well-documented collection of quotes and references; ideas and examples, exhortations, and rationales. She focuses on the stages, the processes, and the loss—particularly dealing with the anguish of the death of close family members (sister and mother).

Her style is unpretentious and lucid: a person-to-person, humanistic manner of writing. This contribution is best considered in terms of its usefulness to patients, providing a self-help approach, analogous to the 12-step method, to the issues of aging. Offered is the support of others who have negotiated this period of their lives, the ways in which they approached it: the need to accept the inevitabilities and suggestions for mastery and control. Women are extolled to reflect, forgive, let go, stop wasting precious moments, and not be bogged down by bitterness. The celebration of self is experienced mutually as a group process with sharing, supportive women who sustain each other.

This treatise is a masterful presentation of a very personal assemblage of experiences and resources which the author is very generously sharing with other women who have reached the stage of their lives when they are obligated to confront the aging process.

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Charles Arak, MD, Mahopac. Died June 13, 1992; age 84. Dr Arak was a 1934 graduate of the University of Glasgow Medical Faculty. His memberships included the Queens County Medical Society and the Medical Society of the State of New York.

Theodore Baum, MD, Rome. Died November 30, 1991; age 89. Dr Baum was a 1929 graduate of the New York University College of Medicine. His memberships included the American Psychiatric Association, the Oneida County Medical Society, and the Medical Society of the State of New York.

Melvyn Berlind, MD, Brooklyn. Died June 10, 1992; age 88. Dr Berlind was a 1928 graduate of McGill University Faculty of Medicine, Montreal. He was a Diplomate of the American Board of Obstetrics and Gynecology. He was a Fellow of the American College of Obstetricians and Gynecologists and a Qualified Fellow of the International College of Surgeons. His memberships included the American Society of Tropical Medicine and Hygiene, the Kings County Medical Society, and the Medical Society of the State of New York.

Orlando Canizares, MD, Yonkers. Died February 22, 1992; age 82. Dr Canizares was a 1935 graduate of the Universite de Paris Faculte de Medecine, France. He was a Diplomate of the American Board of Dermatology, Inc. He was a Fellow of the American College of Physicians. His memberships included the New York State Dermatological Society, the American Dermatological Association, the American Academy of Dermatology, the New York County Medical Society, and the Medical Society of the State of New York.

Frank Mears Falkenbury, MD, Glen Falls. Died June 1, 1992; age 89. Dr Falkenbury was a 1929 graduate of Cornell University Medical College, France. He was a Fellow of the American Academy Family Practice. His memberships included the Warren County Medical Society and the Medical Society of the State of New York.

Armando Gatto Favazza, MD, Columbia, MO. Died April 30, 1992; age 89. Dr Favazza was a 1928 graduate of the Universita Degli Studi di Roma, Facolta di Medicina, Italy. He as a Fellow the American College of Angiology. His memberships included the Kings County Medical Society and the Medical Society of the State of New York.

G. Everett Gaillard, MD, White Plains. Died December 31, 1991; age 83. Dr Gaillard was a 1936 graduate of New York Medical College, New York. He was a Diplomate of the American Board of Allergy and Immunology. He was a Fellow of the American College of Allergists. His memberships included the Academy of Medicine, the American Academy of Allergy, the Westchester County Medical Society, and the Medical Society of the State of New York.

Henry Greenbaum, MD, Newton, MA. Died March 25, 1992; age 85. Dr Greenbaum was a 1935 graduate of the Universite de Paris Faculte de Medecine, New York. His memberships included the American Psychiatric Association, the American Academy of Psychoanalysis, the Queens County Medical Society, and the Medical Society of the State of New York.

Arthur M. Fishberg, MD, New York, NY. Died January 20, 1992; age 94. Dr Fishberg was a 1921 graduate of the Columbia University College of Physicians and Surgeons, New York. He was a Diplomate of the American Board of Internal Medicine. His memberships included the Academy of Medicine, the American Society for Clinical Investigation, the New York County Medical Society, and the Medical Society of the State of New York.

Margaret G. Grassheim, MD, New Rochelle. Died December 1, 1991; age 82. Dr Grassheim was a 1937 graduate of the Friedrich Wilhelms Universitat Mediziniche Fakultat, Berlin, Prussia. Her memberships included the New York State Society of Anesthesiologists, the American Society of Anesthesiologists, the Kings County Medical Society, and the Medical Society of the State of New York.

Salmon Carl Harvey, MD, Brooklyn. Died November 1, 1991; age 83. Dr Harvey was a 1939 graduate of Universitat Bern Medizinische Fakultat, Bern. He was a Diplomate of the American Board of Otolaryngology. He was a Fellow of the American College of Surgeons. His memberships included the American Academy of Otolaryngology, the Pan American Medical Association, the Aerospace Medical Association, the American Geriatrics Society, the Kings County Medical Society, and the Medical Society of the State of New York.

Leonard L. Heimoff, MD, Tamarac, FL. Died June 17, 1992; age 79. Dr Heimoff was a 1939 graduate of the University of Maryland School of Medicine, Baltimore. He was a Fellow of the American College of Preventive Medicine and the American College of Angiology. His memberships included the Academy of Medicine, the New York State Society of Internal Medicine, the American Public Health Association, the American Geriatrics Society, the American Society of Internal Medicine County Medical Society, and the Medical Society of the State of New York.

William Horowitz, MD, Scarsdale. Died July 1, 1992; age 77. Dr Horowitz was a 1939 graduate of the University of Wisconsin Medical School, Madison. He was a Diplomate of the American Board of Internal Medicine. He was a Fellow of the American College of Cardiology and the American College of Physicians. His memberships included the Academy of Medicine, the New York Cardiological Society, the American Society of Clinical Oncology, the Westchester County Medical Society, and the Medical Society of the State of New York.

Morrison John Hosley, MD, Long Lake. Died May 8, 1992; age 89. Dr Hosley was a 1935 graduate of the Albany Medical College of Union University, Albany. He was a Fellow of the American Academy of Family Practice. His memberships included the St. Lawrence County Medical Society and the Medical Society of the State of New York.

Samuel Lassman, MD, Victor. Died April 20, 1992; age 90. Dr Lassman was a 1927 graduate of the University of Maryland School of Medicine, Baltimore. His memberships included the American Society of Anesthesiologists, the New York County Medical Society, and the Medical Society of the State of New York.

Alice Ittner Macaulay, MD, Ardsley-On-Hudson. Died November 25, 1991; age 83. Dr Macaulay was a 1930 graduate of the New York Medical College, New York. Her memberships included the Academy of Medicine, the Westchester County Medical Society, and the Medical Society of the State of New York.

Harold L. Mamelok, MD, Middletown. Died April 25, 1992; age 73. Dr Mamelok was a 1943 graduate of New York University College of Medicine, New York. He was a Diplomate of the American Board of Pathology. He was a Fellow of the American College of Pathologists. His memberships included the New York Pathological Society, the New York State Society of Pathologists, the American Society of Clinical Pathologists, the Orange County Medical Society, and the Medical Society of the State of New York.

Thomas Vincent Miles, MD, Garden City. Died June 22, 1992; age 73. Dr Miles was a 1945 graduate of the State University of New York Health Science Center, New York. His memberships included the Queens County Medical Society and the Medical Society of the State of New York.

Felix Ottaviano, MD, Canastota. Died May 28, 1992; age 86. Dr Ottaviano was a 1934 graduate of the Universita Degli Studi di Roma, Facolta di Medicina E Chirurgia, Italy. He was a Fellow of the American College of Chest Physicians. His memberships included the American Thoracic Society, the Madison County Medical Society, and the Medical Society of the State of New York.

Jesse James Pone, Jr, MD, Old Westbury. Died June 20, 1992; age 67. Dr Pone was a 1955 graduate of the Meharry Medical College, Nashville. His memberships included the Nassau County Medical Society and the Medical Society of the State of New York.

Maria Victoria Roque, MD, Bronx. Died June 15, 1992; age 62. Dr Roque was a 1954 graduate of the Universidad Nacional de Cordoba Facultad de Ciencias Medicas, Spain. Her memberships included the Bronx County Medical Society and the Medical Society of the State of New York.

Herman L. Sheppard, MD, New York, NY. Died June 11, 1992; age 73. Dr Sheppard was a 1943 graduate of the New York Medical College, New York. He was a Diplomate of the American Board of Otolaryngology. His memberships included the American Academy of Otolaryngology, the New York County Medical Society, and the Medical Society of the State of New York.

Manoucher Taheri, MD, Cherry Hill, NJ. Died January 21, 1992; age 63. Dr Taheri was a 1958 graduate of the Madresseh Teb Government Medical School, Teheran, Persia. He was a Fellow of the American College of Anesthesiologists. His memberships included the Ulster County Medical Society and the Medical Society of the State of New York.

Herbert F. Wegweiser, MD, Rockville Centre. Died April 18, 1992; age 57. Dr Wegweiser was a 1963 graduate of the Universitat Zurich Medizinische Fakultat, Zurich. He was a fellow of the American College of Physicians. His memberships included the Kings County Medical Society and the Medical Society of the State of New York.

Thornton H. Wood, MD, New York. Died January 27, 1992; age 95. Dr Wood was a 1921 graduate of the New York University College of Medicine, New York. His memberships included the New York County Medical Society and the Medical Society of the State of New York.

Thomas Sigfried Zimmer, MD, Hollis. Died January 21, 1992; age 93. Dr Zimmer was a 1925 graduate of the Ludwig Maximilians Universitat Medizinche Fakultat, Munchen. His memberships included the New York County Medical Society and the Medical Society of the State of New York.

Julius E. Stolfi, MD

Julius E. Stolfi, MD, who served as the Associate Editor of the *Journal* from 1973 to 1982, passed away on August 21, 1992, at the Bethesda Memorial Hospital in Boynton Beach, Florida. Although Dr Stolfi had retired from the *Journal* in 1982, he continued to serve as a peer reviewer and book reviewer until the month before his death. In addition, he was always available to members of the editorial staff who sought his advice.

Dr Stolfi was born in New York City (Manhattan) in 1916 and received his BS degree in 1936 from the College of the City of New York. Two years later, he received an MS degree from New York University, and in 1942, his MD degree from St Louis University School of Medicine. After a year of internship at Kings County Hospital, he volunteered for service with the United States Army Medical Corps and served in Australia, France, and the Phillipines. After his discharge as a Major in 1946, he served as a resident in internal medicine at Kings County Hospital.

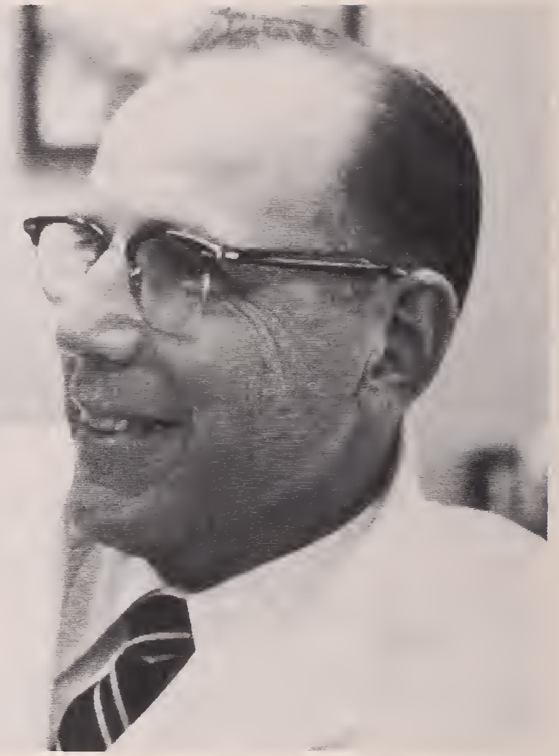
Dr Stolfi entered private practice in 1947 and joined the staffs of the Brooklyn Hospital and the Long Island College Hospital. He served as Chief of the Thyroid Clinic at the former from 1948 to 1962 and became an Attending Physician there in 1956. Between 1965 and 1967, he was Director of Medicine at the Brooklyn Hospital Division of the Brooklyn-Cumberland Medical Center. Over the years, he served as an attending and consulting physician at several hospital in Brooklyn. In addition to his hospital appointments, he served on the faculty of the Department of Medicine of the State University of New York Health Science Center at Brooklyn. In 1956, he was appointed Clinical Assistant Professor of Medicine and rose to the rank of Clinical Professor in 1967. In 1968, he was appointed Associate Dean and in this position oversaw the medical school's teaching programs at affiliated hospitals.

Certified in internal medicine in 1950, Dr Stolfi was recertified in 1974. He served the American College of Physicians in numerous important positions including those of Governor (1966–1972), Chairman of the Nominating Committee (1971–1972), Vice-President (1972–1973), Regent (1973–1978), Chairman of the Finance and Pension Committee (1975–1978), Secretary-General (1978), Chairman of the Nominating Committee of the Board of Regents (1979–1980), and many others.

From 1970 to 1972, Dr Stolfi served as the Director of the State University Hospital in Brooklyn and later assumed the position of Vice-President for Hospital Affairs at the State University of New York Health Science Center at Brooklyn. He remained in the latter position until his retirement in 1983.

Dr Stolfi served both the Medical Society of the County of Kings and the Medical Society of the State of New York (MSSNY) in a variety of capacities. He was president of the former in 1963–1964 and was a member of the MSSNY House of Delegates for several years. He served on numerous committees in both societies.

In 1971, Dr Stolfi launched a nationwide effort to teach laymen cardiopulmonary resuscitation. His initiative was endorsed in 1972 by the American College of Physicians. He also pioneered the development of mobile emergency rooms.



Julius E. Stolfi, MD, 1916–1992.

Dr Stolfi was the author of over 80 papers in scientific medical journals covering topics as diverse as hyperthyroidism and medical malpractice. His editorials on medical socioeconomic issues regularly appeared in the *Journal* during the 1970s. During his years as Associate Editor of the *Journal*, he worked closely with the late Alfred A. Angrist, MD, the editor, who was always high in his praise of him.

Dr Stolfi was a member of numerous medical and nonmedical societies and organizations. Among these were Alpha Omega Alpha, the American Medical Association, the New York Academy of Sciences, the Medical Club of Brooklyn, the New York Athletic Club, and the Brooklyn Club. He also served as a trustee at St Francis College and in 1978 was appointed by Mayor Edward I. Koch to serve on the Fire Department's Medical Practices Review Committee.

Dr Stolfi's contributions to medicine were many and diverse. He was a compassionate physician, a patient and understanding teacher, a respected hospital administrator, a leader in continuing medical education, and a medical editor. His pioneering initiatives in citizen cardiopulmonary resuscitation and the pre-hospital management of emergency patients led to programs that continue today.

Dr Stolfi is survived by his wife Marie; three daughters, Lorraine Rinella of Charlottesville, Virginia, Irene Burns of Boynton Beach, Florida, Juliann Lange of Bryn Mawr, Pennsylvania; and six grandchildren. A memorial fund has been established in his name at the Bethesda Memorial Hospital in Boynton Beach, Florida.

Pascal James Imperato, MD
Editor

New York State Journal of Medicine

INDEX

Volume 92
January–December
1992

Editor:
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Published by the
MEDICAL SOCIETY OF THE STATE OF NEW YORK
PO Box 5404
420 Lakeville Road
Lake Success, NY 11042

ISSUE	NUMBER	PAGES
January	1	1–40
February	2	41–84
March	3	85–126
April	4	127–174
May	5	175–232
June	6	233–290
July	7	291–328
August	8	329–374
September	9	375–414
October	10	415–460
November	11	461–512
December	12	513–590

CLASSIFICATION CODES

CC = Clinical Conference
CPC = Clinicopathologic Conference
CR = Case Report
ED = Editorial Commentary
EP = Essay in Pediatrics
ER = Essay Review
HIST = History of Medicine
LE = Letter to the Editor
LEN = Leads from *Epidemiology Notes*
NF = News Feature

SUBJECT INDEX

Aborigines
Experience of an American medical student with an Aboriginal community in central Australia, the, 431

Acebutolol
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Acetazolamide
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Acinetobacter calcacaeticus
Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy (LE), 162

Acquired immunodeficiency syndrome
AIDS prevention by needle exchange program, 426
AIDS-related *Pneumocystis* pneumonia complicated by diffuse pneumatocele formation (CR), 20
Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297
Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome (ED), 332
Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314
Epidemic Kaposi's sarcoma presenting with pleuropericarditis (CR), 359
Ethical considerations concerning the HIV-positive physician (committee report), 151
Evaluation and clinical management of children with HIV infection, 280
Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462
Illicit drug use should not be legalized (committee report), 59
Progressive pulmonary thromboembolism in association with HIV disease (CR), 403
Public health campaign for New York State, a (ED), 329
Public health crisis in New York City, the, 63
Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65
Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids (LE), 221
Triple jeopardy: The health hazards of unraveling insurance, uncontrolled illness, and economic adversity (ED), 513
Tuberculous peritonitis as an initial manifestation of HIV infection (CR), 269

Act for the Relief of Sick and Disabled Seamen
United States public health service and merchant seamen, the (ED), 331

Acute myeloid leukemia
Spontaneous rupture of the spleen as the initial manifestation of acute myeloid leukemia (CR), 160

Acute pain
Healing waters, 306

Acute Physiology and Chronic Health Evaluation
Intensive care for the elderly: Should the gates remain open? (ED), 175

Acute promyelocytic leukemia
Pseudoleukemia in a patient with Hodgkin's disease (LE), 449

Adolescents
Primary sarcomas of the breast in women under 20 years of age (CR), 497

Adrenal hemorrhage
Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids (CR), 552
Bilateral adrenal hemorrhage in polycythemia vera (CR), 362

Adrenocortical carcinoma
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Adrenocorticotropic hormone
Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids (CR), 552

Adult respiratory distress syndrome
Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297
Intensive care for the elderly: Should the gates remain open? (ED), 175

AIDS testing
Ethical considerations concerning the HIV-positive physician (committee report), 151

Airway disease
Pulmonary cavitary disease (CC), 193

Albany
Drug poisoning patterns: The Albany experience, 1978 and 1988, 134

Albany experience, the
Drug poisoning patterns: The Albany experience, 1978 and 1988, 134

Albany Medical Center
Exploring poisoning data collection systems (ED), 127

Albany Medical Center Hospital
Drug poisoning patterns: The Albany experience, 1978 and 1988, 134

Alcohol
Alcohol and breast cancer, 117

Alcoholism
Alcoholism-induced bone necrosis, 334
Experience of an American medical student with an Aboriginal community in central Australia, the, 431
Rhabdomyolysis associated with pneumococcal pneumonia and sepsis in an abstinent alcohol abuser (LE), 24

Alpha Beta Fraternity
Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

α_2-adrenergic agonists
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Alzheimer's disease
Golden Years or The Wasteland, the, 148
Persistent vegetative state: An analysis of clinical correlates and costs, the, 381

American Academy of Neurology
Persistent vegetative state: An analysis of clinical correlates and costs, the, 381

American Academy of Ophthalmology
Project Diabetes 2000 (LE), 115

American Academy of Pediatrics
Non-psychiatric physician's responsibilities for the suicidal adolescent, the (EP), 97

American Association of Poison Control Centers National Data Collection System
Exploring poisoning data collection systems (ED), 127

American Cancer Society
Health and economic impact of cigarette smoking in New York State, 1987–1989, 469

American College of Obstetricians and Gynecologists
Providing access to obstetric care in New York State (ED), 235

American Diabetes Association
Project Diabetes 2000 (LE), 115

American Medical Association
Medical futility, 485

American Psychiatric Association
Challenge to psychiatry by psychologists: Economic versus professional issues, the, 105

American Psychological Association
Challenge to psychiatry by psychologists: Economic versus professional issues, the, 105

Amiodarone
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Amphotericin
Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy (LE), 162

Anabolic steroid
Importance of differentiating between anabolic steroids and glucocorticoids, the (LE), 365

Anaerobic lung infection
Pulmonary cavitary disease (CC), 193

Anatomy
Leonardo da Vinci's anatomical drawings (HIST), 436

Anesthesiology
Do-not-resuscitate and the anesthesiologist (ED), 129
In lieu of a plaque (LE), 222

Aneurysm
Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156

Angiogram
Not a valentine, 386

Angiomyolipoma
Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction (CR), 217

Angiosarcoma
Cardiac tumors: A current perspective (ED), 41
Chemotherapy for hepatic angiosarcoma (LE), 322

Angiotensin-converting enzyme inhibitors
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Antiarrhythmic agents
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Anticoagulants and antiplatelet drugs
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Antidotarium of the Medical College of Bologna
Smallpox in an 18th-century Italian princess (HIST), 480

Antivivesection
Catfight in the labs, 385

Aortic aneurysm
Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156

Aortic graft
Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156

Aortoenteric fistula
Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156

Apodemus
Deer, ticks, and Lyme disease (ED), 2

Appendiceal abscess
Appendiceal abscess presenting as an infected urachal cyst (LE), 365

Arboviruses
Lassa fever, Part I—Discovering the disease, the story of (HIST), 199
Lassa fever, Part II—Learning more about the disease, the story of (HIST), 264

Arizona Infectious Disease Society
New York State Journal of Medicine, the (LE), 449

Armed forces
Bernard John Dowling Irwin, MD: Physician, naturalist, and military hero (HIST), 537

Arterial disease
Extracranial carotid arterial disease: A prognostic factor for atherothrombotic brain infarction and cerebral transient ischemic attack, 424

Arthralgias
Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Arthritis
Gouty arthritis following gunshot wound: A case report (CR), 110

Article 99
Physician and the movies, the (LE), 365

Asians
Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245

Aspirin
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Associate Editorial Board
Update on the *New York State Journal of Medicine*, an (ED), 294

Atenolol
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Australia
Experience of an American medical student with an Aboriginal community in central Australia, the, 431

Axis I disorder
Münchausen syndrome (CC), 301

α_1-antitrypsin
Eosinophilic globules resembling Mallory bodies in a renal cell carcinoma (CR), 18

β-adrenergic blockers
Adverse cutaneous reactions from cardiovascular drug therapy, 137

β-antagonist
Antihypertensive effects of exercise training, the, 250

Babesia microti
Deer, ticks, and Lyme disease (ED), 2

Beclomethasone dipropionate
Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids (LE), 221

Behavioral Risk Factor Surveillance System
Overweight in New York State (LEN), 224

Bellevue Hospital
Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181

Benzodiazepines
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Bible
Pigeons as a remedy (*segulah*) for jaundice, 189

Big Brother
Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Big Sister
Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Bioethics
Illicit drug use should not be legalized (committee report), 59
Medical futility, 485
New York State Journal of Medicine, the (LE), 449
Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489

Biopsy
Needle localization and biopsy in non-palpable lesions of the breast: An eight-year experience (LE), 71

Birth defects
Neonatal gastrointestinal complications of maternal cocaine abuse (CR), 22

Blackwell, Elizabeth, MD
History of the Women's Medical Society of New York State, a (HIST), 203

Blueberry muffin syndrome
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Board of Trustees 1991–1992
Medical Society of the State of New York, House of Delegates, 27

Body mass index
Overweight in New York State (LEN), 224

Body photography
Total body photography for melanoma surveillance, 494

Boerhaave's syndrome
Cervical esophageal perforation—a unique presentation (CR), 446
Esophageal perforation (ED), 419

Bone necrosis
Alcoholism-induced bone necrosis, 334

Books received, annotations of, 83, 229, 509, 510

Books reviewed
AIDS and Other Sexually Transmitted Diseases—Adolescent Medicine: State of the Art Reviews (Schydlower and Shafer, eds.), 164
AIDS and the Law: A Basic Guide for the Nonlawyer (Terl), 564
Aids Prevention and Treatment: Hope, Humor, and Healing (Seligson and Peterson, eds.), 226
Ambulance: The Story of Emergency Transportation of Sick and Wounded through the Centuries, the (Barkley), 81
Ambulatory Peritoneal Dialysis (Avram and Giordano, eds.), 456

American Dentist: A Pictorial History with a Presentation of Early Dental Photography in America, the (Glenner, Davis, and Burns), 505
Assessing Pediatric Practice: A Critical Study (Duff), 79
Aztec Medicine, Health, and Nutrition (de Montellano), 119
Breast: Comprehensive Management of Benign and Malignant Diseases, the (Bland and Copeland, III, eds.), 167
Carbon Monoxide Poisoning (Jain), 165
Casarett and Doull's Toxicology: The Basic Science of Poisons (Amdur, Doull, and Klaassen, eds.), 456
Casebook in Psychiatric Ethics, A (Committee on Medical Education and Group for the Advancement of Psychiatry), 327
Changing Context of Social-Health Care: Its Implications for Providers and Consumers, the (Rehr and Rosenberg, eds.), 367
Clinical Dermatology (MacKie), 225
Clinical Tests of Vision (Frisen), 165
Cocaine, Aids, and Intravenous Drug Use (Stimmel, ed.), 507
Color Atlas of Clinical Oral Pathology (Neville, Damm, White, and Waldron, eds.), 121
Contemporary Internal Medicine: Clinical Case Studies (Bowen and Mazzaferri, eds.), 285
Continuing Care: The Management of Chronic Disease (Hasler and Schofield, eds.), 81
Cope's Early Diagnosis of the Acute Abdomen (Silen), 505
Crusading Doctor: My Fight for Cooperative Medicine (Shadid), 411
Current Practice of Interventional Radiology (Kadir), 456
Defeating Pain: The War Against a Silent Epidemic (Wall and Jones), 284
Depression in the Medically Ill: An Integrated Approach (Rodin, Craven, and Littlefield), 326
Development of Medical Techniques and Treatments: From Leeches to Heart Surgery (Duke), 169
Did This Really Happen? (Berger), 410
Disinfection, Sterilization, and Preservation (Block), 285
Diving Medicine (Bove and Davis), 76
Drug-Related Problems in Geriatric Nursing Home Patients (Cooper), 283
Drunken Society: Alcohol Abuse and Alcoholism in the Soviet Union, the (Segal, ed.), 81
Early Detection and Treatment of Liver Cancer (Okuda, Tobe, and Kitagama, eds.), 564
EMT: Prehospital Care (Henry and Stapleton), 566
Etiology of Bulimia Nervosa: The Individual and Familial Context, the (Crowther, Tennenbaum, Hobfoll, and Stephens), 564
Evaluation and Treatment of the Psychogeriatric Patient (Gibson, ed.), 283
Eye Trauma (Shingleton, Hersh, and Kenyon, eds.), 169
Facilitating Self Care Practices in the Elderly (Horn, ed.), 227
Fibrinolysis, Thrombosis, and Hemostasis: Concepts, Perspectives, and Clinical Applications (Sherry), 507
Final Exit: The Practicalities of Self-Deliverance and Assisted Suicide for the Dying (Humphry), 455
Fundamentals of Psychoanalytic Technique (Etchegoyen), 326
Geriatric Radiology (Viamonte, Jr., ed.), 284
Growing up: Pastoral Nurture for the Later Years (Robb), 368
Guide to Medical Professional Liability Insurance, the (Johnson, Hatlie, and Johnson), 454
Handbook of Prescribing Medications for Geriatric Patients (Ahronheim), 455
Health Care of the Aged: Needs, Policies, and Services (Monk, ed.), 226
Hippocrates' Handmaidens: Women Married to Physicians (Nitzberg), 508
History of Contraception: From Antiquity to the Present Day, a (McLaren), 325
History of Syphilis (Quétel, Braddock, and Pike), 164
History of Yellow Fever, the (Delaporte, ed.), 367
Housing Risks and Homelessness Among the Urban Elderly (Keigher, ed.), 118
ICU Book, the (Marino, ed.), 77
In the Shade of an Acacia Tree (Lambrecht), 410
Infectious Diseases of the Female Genital Tract (Sweet and Gibbs, eds.), 75
Infectious Risks in Surgery (Howard, ed.), 326
Interpretation of Diagnostic Tests: A Synopsis of Laboratory Medicine (Wallach), 412
Jewish Values in Health and Medicine (Meier, ed.), 225
Kidney, the (Brenner and Rector, eds.), 412
Male Aesthetic Surgery (Courtiss, ed.), 369
Males with Eating Disorders (Andersen, ed.), 77
Managing Asthma (Weinberger, ed.), 78
Marketing for Health and Wellness Programs (Self, ed.), 228
Merck Manual of Geriatrics, the (Abrams, Berkow, and Fletcher, eds.), 168
Modern Medicine and Jewish Ethics (Rosner), 565
Natural Medicine for Children (Scott, ed.), 77
Neonatal Seizures (Wasterlain and Vert, eds.), 79
Neoplasms with Eccrine Differentiation (Abenoza and Ackerman, eds.), 120
Obstetrics and Gynecology Review 1991 (Sheld), 413
Pediatric Airway, the (Othersen, ed.), 368
Phacoemulsification Surgery (Devine and Banko), 166
Positive and Negative Syndromes in Schizophrenia: Assessment and Research (Kay), 286
Practical Guide to the Care of the Medical Patient (Ferri), 227
Practical Handbook of Joint Fluid Analysis (Gatter and Schumacher, eds.), 368
Principles and Practice of Pediatrics (Oski, DeAngelis, Feigin, and Warshaw, eds.), 167
Progress in Cardiology (Zipes and Rowlands), 166
Psychiatry in the Nursing Home: Assessment, Evaluation, and Intervention (Birkett), 283
Psychiatry Specialty Board Review (Easson and Rock), 226
Psychological Assessment: Psychiatric Diagnosis and Treatment Planning (Hurt, Reznikoff, and Clarkin), 566
Psychosocial Aspects of End-Stage Renal Disease (Hardy, Kiernan, Kutscher, Cahill and Benvenisty, eds.), 412
Pulmonary Manifestations of Systemic Disease: A Clinical Approach (Cooper, ed.), 78
Purification Prescription, the (Hendler), 118
Reconstruction of the Child's Hand (Carter, Peter R., ed.), 325
Renal and Electrolyte Disorders (Schrier, ed.), 507
Resources for People with Disabilities and Chronic Conditions (Resources for Rehabilitation), 327
Rheumatology for the Health Care Professional (Spiera and Oreskes, eds.), 165
Roots of the modern medical malpractice crisis, the, 279
Set No Limits: A Rebuttal to Daniel Callahan's Proposal to Limit Health Care for the Elderly (Barry and Bradley, eds.), 454
Social Work in Health Care: A Handbook for Practice (Davidson and Clarke, eds.), 120
Status of Health of Blacks in the United States of America, the (Alcena), 567
Strategic Health Planning: Methods and Techniques Applied to Marketing and Management (Spiegel and Hyman), 506
Surgery of the Ear (Glasscock, III and Shambaugh, Jr., eds.), 168
Swimming Upstream: Teaching and Learning Psychotherapy in a Biological Era (Lewis, ed.), 79
Synopsis of Neurology (Lechtenberg, ed.), 120
Techniques in Arterial Surgery (Bergan and Yao, eds.), 75
Textbook of Performing Arts Medicine (Sataloff, Brandfonbrener, and Lederman, eds.), 119
Textbook of Secretory Diarrhea (Lebenthal and Duffy, eds.), 76
Thyroid Gland, The (Greer, ed.), 75
Toxicity of Anticancer Drugs, the (Powis and Hacker, eds.), 164
Vaginal Surgery (Nichols and Randall), 169
Women and Aging: Celebrating Ourselves (Thone), 567

Borrelia burgdorferi
  Deer, ticks, and Lyme disease (ED), 2

Bosnia-Herzegovina
  Health and humanitarian assistance in conflicts and disasters (ED), 461

Bowel disease
  Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids (CR), 552

Bradykinesia
  Widespread complications of lupus erythematosus (CC), 474

Brain infarction
  Extracranial carotid arterial disease: A prognostic factor for atherothrombotic brain infarction and cerebral transient ischemic attack, 424

Breast cancer
  Alcohol and breast cancer, 117
  Leiomyoma of the nipple (LE), 408
  Mastectomy: Is it necessary for occult breast cancer? (ED), 516
  Metastatic hemangiopericytoma of the breast (CR), 158
  Needle localization and biopsy in non-palpable lesions of the breast: An eight-year experience (LE), 71
  Occult breast cancer (CR), 555
  Primary sarcomas of the breast in women under 20 years of age (CR), 497

Breast service
  Primary sarcomas of the breast in women under 20 years of age (CR), 497

Bronchial tuberculosis
  Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462

Bronchitis
  Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297

Bronchoalveolar lavage
  Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297

Bronchoscopy
  Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297
  Pulmonary cavitary disease (CC), 193

Brooklyn Jewish Hospital
  Memories of an internship (LE), 558

Budd-Chiari syndrome
  Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the (ED), 88
  Recombinant tissue-plasminogen activator for acute Budd-Chiari syndrome secondary to paroxysmal nocturnal hemoglobinuria (CR), 109

Bumetanide
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Bureau of Cancer Epidemiology
  Alcohol and breast cancer, 117

Bureau of Communicable Disease Control
  Drug poisoning patterns: The Albany experience, 1978 and 1988, 134

Calcium-channel blockers
  Adverse cutaneous reactions from cardiovascular drug therapy, 137
  Diltiazem-induced myoclonus (CR), 447

Calmette-Guerin
  Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185

Cancer
  Clavicular resection in the exposure and resection of malignant tumors (CR), 214
  Eosinophilic globules resembling Mallory bodies in a renal cell carcinoma (CR), 18
  Marital status and stage at diagnosis in cancer, 8
  Metastatic hemangiopericytoma of the breast (CR), 158
  Needle localization and biopsy in non-palpable lesions of the breast: An eight-year experience (LE), 71
  Transitional cell carcinoma of the renal pelvis in an incompletely duplicated collecting system (CR), 500

Candida albicans
  Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy (LE), 162
  Tuberculous peritonitis as an initial manifestation of HIV infection (CR), 269
  Tunneled central venous catheters complications and the iatrogenic superior vena cava syndrome (ED), 43

Capgras syndrome
  W. Somerset Maugham, Capgras syndrome, and aging, 547

CAPP v Rank
  Challenge to psychiatry by psychologists: Economic versus professional issues, the, 105

Captopril
  Adverse cutaneous reactions from cardiovascular drug therapy, 137
  Neutropenia induced by low-dose captopril (CR), 219

Cardiac disease
  Acute tricuspid valve incompetence secondary to blunt trauma (CR), 445
  Diltiazem-induced myoclonus (CR), 447
  Progressive pulmonary thromboembolism in association with HIV disease (CR), 403
  Transesophageal echo: Defining the indications (ED), 418
  Unexpected reversal of an idiopathic dilated cardiomyopathy and the urgent need for a re-evaluation of the indicators of patient survival (CR), 404

Cardiac glycosides
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Cardiac Kaposi's sarcoma
  Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome (ED), 332
  Epidemic Kaposi's sarcoma presenting with pleuropericarditis (CR), 359

Cardiac tumors
  Cardiac tumors: A current perspective (ED), 41
  Primary cardiac tumors, diagnosis, and management in 14 cases, 49

Cardiography
  Primary cardiac tumors, diagnosis, and management in 14 cases, 49

Cardiomyopathy
  Right ventricular cardiomyopathy (CR), 318
  Unexpected reversal of an idiopathic dilated cardiomyopathy and the urgent need for a re-evaluation of the indicators of patient survival (CR), 404
  Widespread complications of lupus erythematosus (CC), 474

Cardiopulmonary resuscitation
  Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181
  Medical futility, 485

Cardiovascular disease
  Cardiovascular risk factors in native American children, 378

Cardiovascular drug therapy
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Catecholamine
  Antihypertensive effects of exercise training, the, 250

Catheterization
  Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy (LE), 162

Cavitary disease
  Pulmonary cavitary disease (CC), 193

Centers for Disease Control
  Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462
  Health and economic impact of cigarette smoking in New York State, 1987–1989, 469

Centers for Disease Control's National Center for Health Statistics
  Exploring poisoning data collection systems (ED), 127

Central nervous system
  Hyperbilirubinemia in the term infant (LE), 221

Cerebral ischemic attack
  Extracranial carotid arterial disease: A prognostic factor for atherothrombotic brain infarction and cerebral transient ischemic attack, 424

Cerebrovascular disease
  Osler-Weber-Rendu disease: Diagnosis and management of spontaneous hemothorax during pregnancy (CR), 551

Cervical cancer
  Pap smear histories in a medical clinic: Accuracy of patients' self-reports, 421

Chemoprophylaxis
  Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185

Chemotherapy
  Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome (ED), 332
  Pseudoleukemia in a patient with Hodgkin's disease (LE), 449
  Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Chinese
  Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245

Chlamydia trachomata
  Experience of an American medical student with an Aboriginal community in central Australia, the, 431

Chlorthalidone
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Cholecystectomy
  Complications of laparoscopic cholecystectomy in a geriatric population group, 518
  Serious complications of laparoscopic cholecystectomy in New York State, 179

Cholecystitis
  Acute emphysematous cholecystitis: A case report and review of the literature (CR), 406

Cholesterol
  Cardiovascular risk factors in native American children, 378
  New York State Dietary Survey: Fat and cholesterol consumption, the (LEN), 73
  Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245

Chronic cold hemagglutinin disease
  Diseases associated with monoclonal immunoglobulins (ED), 463

Chronic lymphocytic leukemia
  Pneumococcal cellulitis and dysgammaglobulinemia (CR), 113

Chronic obstructive pulmonary disease
  Diltiazem-induced myoclonus (CR), 447

Chronic pain
  Healing waters, 306

Claviculectomy
- Clavicular resection in the exposure and resection of malignant tumors (CR), 214

Clinical Conferences (series)
- Münchausen syndrome, 301
- Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman, 343
- Pulmonary cavitary disease, 193
- Widespread complications of lupus erythematosus, 474

Clofibrate
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Clonidine
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Cocaine
- Illicit drug use should not be legalized (committee report), 59

Cocaine abuse
- Neonatal gastrointestinal complications of maternal cocaine abuse (CR), 22

Coma
- Myxedema coma (CR), 553

Committee on Bioethical Issues
- Medical futility, 485
- Physician-assisted suicide (committee report), 388

Committee on Publications
- Future of the *New York State Journal of Medicine*, the (LE), 408
- *New York State Journal of Medicine*, the (LE), 408

Committee on Publications, Library, and Archives
- Update on the *New York State Journal of Medicine,* an (ED), 294

Compulsive gambling
- Experience of an American medical student with an Aboriginal community in central Australia, the, 431

Congenital reticulohistiocytosis
- Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Congestive heart failure
- Neutropenia induced by low-dose captopril (CR), 219

Consultation guidelines
- Island Peer Review Organization educational posters, 57

Contaminated sharps
- Regulated medical waste: Is any of it infectious?, 349

Convention Committees
- Medical Society of the State of New York, House of Delegates, 27

Councilors 1991–1992
- Medical Society of the State of New York, House of Delegates, 27

Creutzfeldt-Jacob syndrome
- Persistent vegetative state: An analysis of clinical correlates and costs, the, 381

Crohn's disease
- Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids (CR), 552

Cystic neoplasms
- Mucinous cystic neoplasm of the pancreas (LE), 503

Cytomegalovirus
- Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65

da Vinci, Leonardo
- Leonardo da Vinci's anatomical drawings (HIST), 436

Deer ticks
- Deer, ticks, and Lyme disease (ED), 2
- Incidence and prevalence of Lyme disease in a suburban Westchester County community, 5

Dementia
- Persistent vegetative state: An analysis of clinical correlates and costs, the, 381

Dermatitis autogenica
- Münchausen syndrome (CC), 301

Dermatology
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Developmental lesions
- Pulmonary cavitary disease (CC), 193

Diabetes
- Alcoholism-induced bone necrosis, 334
- Cardiovascular risk factors in native American children, 378
- Experience of an American medical student with an Aboriginal community in central Australia, the, 431
- Project Diabetes 2000 (LE), 115

Diabetes 2000
- Project Diabetes 2000 (LE), 115

Diazoxide
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Diet
- New York State Dietary Survey: Fat and cholesterol consumption, the (LEN), 73
- Overweight in New York State (LEN), 224

Digitalis
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Dilantin
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Diltiazem
- Adverse cutaneous reactions from cardiovascular drug therapy, 137
- Diltiazem-induced myoclonus (CR), 447

Dipyridamole
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Direct vasodilator drugs
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Discharge planning guidelines
- Island Peer Review Organization educational posters, 57

Disruptive hospitalization
- Münchausen syndrome (CC), 301

Diuretics
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Do-not-resuscitate order
- Do-not-resuscitate order in a nursing home: Patient's choice or staff's decision, the, 131
- Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181
- Medical futility, 485
- Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489

Dobutamine
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Doctor, The
- Physician and the movies, the (LE), 365

Doe v Roe
- Judicial ruling on a physician violation of New York State law protecting the confidentialty of HIV-related information, 545

Dolley, Sarah R. Adamson, MD:
- History of the Women's Medical Society of New York State, a (HIST), 203

Down's syndrome
- Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Draft Manual for Infectious Waste Management
- Regulated medical waste: Is any of it infectious?, 349

Drug abuse
- Illegal drug use (LE), 276
- Neonatal gastrointestinal complications of maternal cocaine abuse (CR), 22

Drug Abuse Warning Network
- Exploring poisoning data collection systems (ED), 127

Drug legalization
- Illicit drug use should not be legalized (committee report), 59

Drug poisoning
- Drug poisoning patterns: The Albany experience, 1978 and 1988, 134
- Exploring poisoning data collection systems (ED), 127

Drug reactions
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Drug therapy
- Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the (ED), 88
- Importance of differentiating between anabolic steroids and glucocorticoids, the (LE), 365
- Update in epilepsy, Part I—Diagnosis and treatment of epilepsy, 14
- Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53
- Update in epilepsy, Part III—Surgical therapy of intractable epilepsy, 92

Drug-resistant disease
- Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462

Dysgammaglobulinemia
- Pneumococcal cellulitis and dysgammaglobulinemia (CR), 113

Echocardiography
- Acute tricuspid valve incompetence secondary to blunt trauma (CR), 445
- Transesophageal echo: Defining the indications (ED), 418

Economics
- Economic interventions to discourage the illegal sale of cigarettes to minors in New York State, 520

Triple jeopardy: The health hazards of unraveling insurance, uncontrolled illness, and economic adversity (ED), 513

Editorial Commentaries
Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome, 332
Cardiac tumors: A current perspective, 41
Challenge of controlling tuberculosis in New York City, the, 291
Common interests and shared objectives, 234
Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the, 88
Deer, ticks, and Lyme disease, 2
Diseases associated with monoclonal immunoglobulins, 463
Do-not-resuscitate and the anesthesiologist, 129
Drug development and patent law, 86
Esophageal perforation, 419
Exploring poisoning data collection systems, 127
Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the, 462
Health and humanitarian assistance in conflicts and disasters, 461
Immunization crisis in New York, the, 415
Inaugural address of the President, 233
Intensive care for the elderly: Should the gates remain open?, 175
Mastectomy: Is it necessary for occult breast cancer?, 516
Observations on the National Practitioner Data Bank, 1
Osseous sarcoidosis, radiologic imaging, and physician interaction, 177
Physician and the courts, the, 375
Pregnancy and pulmonary arterio-venous fistula, 515
Prevention of youth suicide, 85
Providing access to obstetric care in New York State, 235
Public health campaign for New York State, a, 329
Television physicians, 376
Transesophageal echo: Defining the indications, 418
Triple jeopardy: The health hazards of unraveling insurance, uncontrolled illness, and economic adversity, 513
Tuberculosis in New York: Rapid rise of a preventable disease, 293
Tunneled central venous catheters complications and the iatrogenic superior vena cava syndrome, 43
United States public health service and merchant seamen, the, 331
Update on the *New York State Journal of Medicine,* an, 294

Eighteenth-century
Smallpox in an 18th-century Italian princess (HIST), 480

Electrocardiography
Morphology of ventricular premature beats a marker for left left ventricular dysfunction? is the, 89

Electroencephalography
Update in epilepsy, Part I—Diagnosis and treatment of epilepsy, 14
Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53
Update in epilepsy, Part III—Surgical therapy of intractable epilepsy, 92

Emphysema
Acute emphysematous cholecystitis: A case report and review of the literature (CR), 406

Enalapril
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Endogenous opioid system
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Endometrioma
Large endometrioma with intrauterine pregnancy (CR), 364

Endoscopy
Gastrocolic fistula complicating a benign gastric ulcer (CR), 69
Impaction of an ingested table fork in a patient with a surgically restricted stomach (LE), 115
Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156

Enteral nutrition
Enteral nutrition supplied by jejunostomy can result in jejunal infarction (LE), 25

Environmental Protection Agency
Regulated medical waste: Is any of it infectious?, 349

Enzyme-linked immunosorbent assays
Tuberculous peritonitis as an initial manifestation of HIV infection (CR), 269

EPA Guide for Infectious Waste Management
Regulated medical waste: Is any of it infectious?, 349

Epidemics
Immunization crisis in New York, the (ED), 415
Public health campaign for New York State, a (ED), 329

Epilepsy
Update in epilepsy, Part I—Diagnosis and treatment of epilepsy, 14
Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53
Update in epilepsy, Part III—Surgical therapy of intractable epilepsy, 92

Epinephrine
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Errata, 278, 450

Erythema migrans
Incidence and prevalence of Lyme disease in a suburban Westchester County community, 5

Erythrocyte protoporphyrin
Lead screening and follow-up in an urban pediatrics clinic, 338

Esophagus
Cervical esophageal perforation—a unique presentation (CR), 446
Esophageal perforation (ED), 419

Essay Review
"Rationing America's Medical Care", 451
"William Alexander Hammond, MD—A 19th-century physician-scientist and entrepreneur," 561

Essays in Pediatrics (series)
Non-psychiatric physician's responsibilities for the suicidal adolescent, the, 97
Skin changes associated with hematologic and oncologic diseases in children, 256

Este dynasty
Smallpox in an 18th-century Italian princess (HIST), 480

Ethacrynic acid
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Euthanasia
Physician-assisted suicide (committee report), 388

Exercise
Antihypertensive effects of exercise training, the, 250

Febrile seizures
Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53

Federal Patient Self-Determination Act
Do-not-resuscitate order in a nursing home: Patient's choice or staff's decision, the, 131

Federation of Jewish Philanthropies
Memories of an internship in 1943, 307

Fever Hospital
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

Fibrosarcoma
Cardiac tumors: A current perspective (ED), 41
Clavicular resection in the exposure and resection of malignant tumors (CR), 214

Field tent hospital
Bernard John Dowling Irwin, MD: Physician, naturalist, and military hero (HIST), 537

Final Exit
Physician-assisted suicide (committee report), 388

Fine-needle aspiration
Chemotherapy for hepatic angiosarcoma (LE), 322
Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314
Mastectomy: Is it necessary for occult breast cancer? (ED), 516
Metastatic hemangiopericytoma of the breast (CR), 158
Primary sarcomas of the breast in women under 20 years of age (CR), 497
Trauma-related presentation of non-Hodgkin's lymphoma (LE), 322

Food and Drug Administration
Drug development and patent law (ED), 86

Foreign war
Health and humanitarian assistance in conflicts and disasters (ED), 461

Fraternity
Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Fungal peritonitis
Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy (LE), 162

Furosemide
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Gastrointestinal tract
Gastrocolic fistula complicating a benign gastric ulcer (CR), 69
Neonatal gastrointestinal complications of maternal cocaine abuse (CR), 22

Gastrojejunostomy
Impaction of an ingested table fork in a patient with a surgically restricted stomach (LE), 115

Geriatric homes
- Do-not-resuscitate order in a nursing home: Patient's choice or staff's decision, the, 131
- Golden Years or The Wasteland, the, 148

Geriatrics
- Complications of laparoscopic cholecystectomy in a geriatric population group, 518
- Intensive care for the elderly: Should the gates remain open? (ED), 175
- New hospice benefit available for nursing home residents (LE), 323

Glioblastoma
- Intralymphatic immunotherapy of glioblastoma (CR), 272

Glucocorticoids
- Importance of differentiating between anabolic steroids and glucocorticoids, the (LE), 365

Glyburide
- Neutropenia induced by low-dose captopril (CR), 219

Gonorrheal conjunctivitis
- Experience of an American medical student with an Aboriginal community in central Australia, the, 431

Goodpasture's syndrome
- Münchausen syndrome (CC), 301

Gouty arthritis
- Gouty arthritis following gunshot wound: A case report (CR), 110

Graft-v-host disease
- Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Grand mal
- Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53

Guinea
- Lassa fever, Part III—The disease in the community, the story of (HIST), 440

Gynecology
- Professional liability reform and access to Medicaid obstetric care in New York State, 237
- Providing access to obstetric care in New York State (ED), 235

Haemophilus influenzae
- Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297
- Immunization crisis in New York, the (ED), 415

Hahnemann University School of Medicine
- Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Handicaps
- Healing waters, 306

Headaches
- Headaches and children: A reply to "An urban primary school health program" (LE), 24

Healing waters
- Healing waters, 306

Health care coverage
- Access to health care in rural western New York State, 465
- "Rationing America's Medical Care" (ER), 451

Health Care Proxy Laws
- Do-not-resuscitate order in a nursing home: Patient's choice or staff's decision, the, 131
- Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489

Health insurance
- Triple jeopardy: The health hazards of unraveling insurance, uncontrolled illness, and economic adversity (ED), 513

Health law
- National Practitioner Data Bank, 12
- Observations on the National Practitioner Data Bank (ED), 1

Healthy People 2000
- Health and economic impact of cigarette smoking in New York State, 1987–1989, 469
- Overweight in New York State (LEN), 224

Heart disease
- Antihypertensive effects of exercise training, the, 250
- Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome (ED), 332
- Cardiac tumors: A current perspective (ED), 41
- Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the (ED), 88
- Epidemic Kaposi's sarcoma presenting with pleuropericarditis (CR), 359
- Morphology of ventricular premature beats a marker for left left ventricular dysfunction? is the, 89
- New York State Dietary Survey: Fat and cholesterol consumption, the (LEN), 73
- Pregnancy and pulmonary arterio-venous fistula (ED), 515
- Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156
- Primary cardiac tumors, diagnosis, and management in 14 cases, 49
- Pulmonary cavitary disease (CC), 193
- Right ventricular cardiomyopathy (CR), 318
- Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65
- Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245
- Tunneled central venous catheters complications and the iatrogenic superior vena cava syndrome (ED), 43

Heavy chain diseases
- Diseases associated with monoclonal immunoglobulins (ED), 463

Hemangiopericytoma
- Metastatic hemangiopericytoma of the breast (CR), 158

Hematology
- Skin changes associated with hematologic and oncologic diseases in children (EP), 256
- Trauma-related presentation of non-Hodgkin's lymphoma (LE), 322

Hemlock Society
- Physician-assisted suicide (committee report), 388

Hemophilia
- Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids (LE), 221

Hemoptysis
- Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Hemorrhage
- Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids (CR), 552

Hemorrhagic telanglectasia
- Osler-Weber-Rendu disease: Diagnosis and management of spontaneous hemothorax during pregnancy (CR), 551

Hemorrhagica histrionica
- Münchausen syndrome (CC), 301

Heparin
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Hepatic angiosarcoma
- Chemotherapy for hepatic angiosarcoma (LE), 322

Hepatitis
- AIDS prevention by needle exchange program, 426
- Illicit drug use should not be legalized (committee report), 59
- Regulated medical waste: Is any of it infectious?, 349

Herniation of omentum
- Morgagni hernia mimicking a lipomatous tumor (CR), 499

Heroin
- Illicit drug use should not be legalized (committee report), 59

History
- Memories of an internship in 1943, 307
- William Alexander Hammond, MD: A 19th-century physician-scientist and entrepreneur, 561

History of Medicine
- Bernard John Dowling Irwin, MD: Physician, naturalist, and military hero, 537
- Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen, 353
- Health care for seamen in the Port of New York: Part II—The rise of the seaman's retreat, 394
- History of Jewish attitudes toward nursing, a, 529
- History of the Women's Medical Society of New York State, a, 203
- History—The great reminder: The relationship between the Long Island College Hospital and the State University of New York Health Science Center at Brooklyn, 392
- Lassa fever, Part I—Discovering the disease, the story of, 199
- Lassa fever, Part II—Learning more about the disease, the story of, 264
- Lassa fever, Part III—The disease in the community, the story of, 440
- Leonardo da Vinci's anatomical drawings, 436
- Smallpox in an 18th-century Italian princess, 480

HIV/AIDS Clinical Resource Directory
- Evaluation and clinical management of children with HIV infection, 280

Hodgkin's disease
  Pseudoleukemia in a patient with Hodgkin's disease (LE), 449

Homosexuality
  W. Somerset Maugham, Capgras syndrome, and aging, 547

Hospice care
  Hospice care (LE), 72
  New hospice benefit available for nursing home residents (LE), 323

Hospital Association of New York State
  Providing access to obstetric care in New York State (ED), 235

House Committee on Bylaws
  Medical Society of the State of New York, House of Delegates, 27

House of Delegates
  In support of the *New York State Journal of Medicine* (LE), 558
  Inaugural address of the President (ED), 233
  Update on the *New York State Journal of Medicine,* an (ED), 294

House of Delegates resolution number 92-59
  Update on the *New York State Journal of Medicine,* an (ED), 294

House of Delegates resolution number 92-85
  Update on the *New York State Journal of Medicine,* an (ED), 294

Human Factor, The
  Television physicians (ED), 376

Human immunodeficiency virus
  AIDS prevention by needle exchange program, 426
  Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297
  Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome (ED), 332
  Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314
  Epidemic Kaposi's sarcoma presenting with pleuropericarditis (CR), 359
  Ethical considerations concerning the HIV-positive physician (committee report), 151
  Evaluation and clinical management of children with HIV infection, 280
  Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462
  Immunization crisis in New York, the (ED), 415
  Judicial ruling on a physician violation of New York State law protecting the confidentialty of HIV-related information, 545
  Low technology and the prevention of nosocomial AIDS (LE), 276
  Progressive pulmonary thromboembolism in association with HIV disease (CR), 403
  Public health campaign for New York State, a (ED), 329
  Purpura fulminans, metastatic endophthalmitis, and thrombotic thrombocytopenic purpura in an HIV-infected patient (CR), 313
  Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65
  Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids (LE), 221
  Tuberculosis in New York: Rapid rise of a preventable disease (ED), 293
  Tuberculous peritonitis as an initial manifestation of HIV infection (CR), 269

Humanitarian aid
  Health and humanitarian assistance in conflicts and disasters (ED), 461

Hydrochlorothiazide
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Hyperbilirubinemia
  Hyperbilirubinemia in the term infant (LE), 221

Hyperlipidemia
  Alcoholism-induced bone necrosis, 334
  Hyperlipidemia in monoclonal gammopathy (CR), 496

Hyperpyrexia figmentatica
  Münchausen syndrome (CC), 301

Hypertension
  Acute emphysematous cholecystitis: A case report and review of the literature (CR), 406
  Antihypertensive effects of exercise training, the, 250
  Neutropenia induced by low-dose captopril (CR), 219
  Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343
  Progressive pulmonary thromboembolism in association with HIV disease (CR), 403
  Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245

Hyperuricemia
  Alcoholism-induced bone necrosis, 334

Hypochondriasis
  Münchausen syndrome (CC), 301

Hypothyroidism
  Myxedema coma (CR), 553

Idiopathic disease
  Idiopathic segmental infarction of the greater omentum (LE), 503
  Pulmonary cavitary disease (CC), 193
  Right ventricular cardiomyopathy (CR), 318
  Unexpected reversal of an idiopathic dilated cardiomyopathy and the urgent need for a re-evaluation of the indicators of patient survival (CR), 404

Illegal drug use
  Illegal drug use (LE), 276

Immunoglobin G
  Ten-year survival in multiple myeloma: Report of two cases and review of the literature (CR), 316

Immunologic disease
  Pulmonary cavitary disease (CC), 193

Immunotherapy
  Intralymphatic immunotherapy of glioblastoma (CR), 272

In memoriam
  Milton B. Handelsman, MD: 1906–1992, 511

Index Medicus
  Plea for keeping the *New York State Journal of Medicine* alive, a (LE), 558
  Update on the *New York State Journal of Medicine,* an (ED), 294

Infarction
  Idiopathic segmental infarction of the greater omentum (LE), 503

Infectious medical waste
  Regulated medical waste: Is any of it infectious?, 349

Inflammatory bowel disease
  Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids (CR), 552

Information Services
  Update on the *New York State Journal of Medicine*, an (ED), 294

Ingested foreign bodies
  Impaction of an ingested table fork in a patient with a surgically restricted stomach (LE), 115

Institute for Scientific Information
  Update on the *New York State Journal of Medicine,* an (ED), 294

Intensive care unit
  Intensive care for the elderly: Should the gates remain open? (ED), 175
  Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Interferon-α
  Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome (ED), 332

Interlibrary loan
  Update on the *New York State Journal of Medicine,* an (ED), 294

Internship
  Memories of an internship (LE), 558
  Memories of an internship in 1943, 307

Intrauterine pregnancy
  Large endometrioma with intrauterine pregnancy (CR), 364

Intrauterine viral infections
  Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Intravenous drug use
  AIDS prevention by needle exchange program, 426

Island Peer Review Organization
  Complications of laparoscopic cholecystectomy in a geriatric population group, 518
  Island Peer Review Organization educational posters, 57
  Serious complications of laparoscopic cholecystectomy in New York State, 179

Isoniazid chemoprophylaxis
  Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185

Ixodes dammini
  Deer, ticks, and Lyme disease (ED), 2

Jaundice
  Hyperbilirubinemia in the term infant (LE), 221
  Pigeons as a remedy (*segulah*) for jaundice, 189

Jejunostomy
  Enteral nutrition supplied by jejunostomy can result in jejunal infarction (LE), 25

Jewish Hospital of Brooklyn
  Memories of an internship in 1943, 307

Jewish nurses
  History of Jewish attitudes toward nursing, a (HIST), 529

Jewish traditional medicine
  Pigeons as a remedy (*segulah*) for jaundice, 189

John Wayne Cancer Institute
- Waiting for the doctor, 262
- Waiting for the doctor (LE), 559

Joint Commission on Accreditation of Health Care Organizations
- Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181

κ-light chains
- Ten-year survival in multiple myeloma: Report of two cases and review of the literature (CR), 316

Kaplow, Raymond, MD
- In lieu of a plaque (LE), 222

Kaposi's sarcoma
- Cardiac Kaposi's sarcoma in a patient with acquired immunodeficiency syndrome (ED), 332
- Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314
- Epidemic Kaposi's sarcoma presenting with pleuropericarditis (CR), 359

Kernicterus
- Hyperbilirubinemia in the term infant (LE), 221

Kevorkiam, Jack
- Physician-assisted suicide (committee report), 388

Labetalol
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Laparoscopic cholecystectomy
- Complications of laparoscopic cholecystectomy in a geriatric population group, 518
- Serious complications of laparoscopic cholecystectomy in New York State, 179

Laparotomy
- Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156

Lassa fever
- Common interests and shared objectives (ED), 234
- Lassa fever, Part I—Discovering the disease, the story of (HIST), 199
- Lassa fever, Part II—Learning more about the disease, the story of (HIST), 264
- Lassa fever, Part III—The disease in the community, the story of (HIST), 440

Lead poisoning
- Gouty arthritis following gunshot wound: A case report (CR), 110
- Lead screening and follow-up in an urban pediatrics clinic, 338
- Low level and bystander exposure to lead among factory workers, 525
- Public health campaign for New York State, a (ED), 329

Leads from *Epidemiology Notes* (series)
- Alcohol and breast cancer, 117
- Evaluation and clinical management of children with HIV infection, 280
- New York State Dietary Survey: Fat and cholesterol consumption, the (LEN), 73
- Overweight in New York State, 224

Leiomyoma
- Leiomyoma of the nipple (LE), 408

Letters to the Editor
- Appendiceal abscess presenting as an infected urachal cyst, 365
- Bluestone, Joseph Isaac, 115
- Changing winds, the, 449
- Chemotherapy for hepatic angiosarcoma, 322
- Choice of surgical procedure for prostatic surgery, 408
- Cigarette advertising in news magazines, 502
- Enteral nutrition supplied by jejunostomy can result in jejunal infarction, 25
- Future of the *New York State Journal of Medicine*, the, 408
- Headaches and children: A reply to "An urban primary school health program", 24
- History: The great reminder, 559
- Hospice care, 72
- How we tell our doctors about pain, 162
- Human tail, the, 116
- Hyperbilirubinemia in the term infant, 221
- Idiopathic segmental infarction of the greater omentum, 503
- Illegal drug use, 276
- Impaction of an ingested table fork in a patient with a surgically restricted stomach, 115
- Importance of differentiating between anabolic steroids and glucocorticoids, the, 365
- In lieu of a plaque, 222
- In support of the *New York State Journal of Medicine*, 558
- Leiomyoma of the nipple, 408
- Low technology and the prevention of nosocomial AIDS, 276
- Memories of an internship, 558
- Mucinous cystic neoplasm of the pancreas, 503
- Needle localization and biopsy in non-palpable lesions of the breast: An eight-year experience, 71
- New hospice benefit available for nursing home residents, 323
- *New York State Journal of Medicine*, the, 408, 449
- *Nocardia brasiliensis* soft tissue infection in an immunocompromised host, 275
- Phenytoin and smoking, 71
- Phi Lambda Kappa Medical Fraternity: Religion and medicine, the, 366
- Physically handicapped children's program: A helping hand for children with serious health problems, the, 408
- Physician and the movies, the, 365
- Physician and the poor, the, 162
- Plea for keeping the *New York State Journal of Medicine* alive, a, 558
- Project Diabetes 2000, 115
- Proper obstetric care for women in New York State, 502
- Pseudoleukemia in a patient with Hodgkin's disease, 449
- Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy, 162
- Rhabdomyolysis associated with pneumococcal pneumonia and sepsis in an abstinent alcohol abuser, 24
- Role of third party payors in medical care, the, 323
- Role of third party payors in medical care, the (reply), 449
- Trauma-related presentation of non-Hodgkin's lymphoma, 322
- Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids, 221
- Ultimate care givers, the, 116
- Waiting for the doctor, 559

Leukemia
- Skin changes associated with hematologic and oncologic diseases in children (EP), 256
- Spontaneous rupture of the spleen as the initial manifestation of acute myeloid leukemia (CR), 160

Liability
- Professional liability reform and access to Medicaid obstetric care in New York State, 237

Liberia
- Lassa fever, Part III—The disease in the community, the story of (HIST), 440

Life-sustaining care
- Medical futility, 485
- Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489
- Physician-assisted suicide (committee report), 388

Limbic system kindling
- Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Lipid-lowering agents
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Lipoprotein
- Hyperlipidemia in monoclonal gammopathy (CR), 496

Literature
- W. Somerset Maugham, Capgras syndrome, and aging, 547

Long Island College Hospital
- History—The great reminder: The relationship between the Long Island College Hospital and the State University of New York Health Science Center at Brooklyn (HIST), 392

Long Island Hospital
- History: The great reminder (LE), 559

Lovastatin
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Low technology
- Low technology and the prevention of nosocomial AIDS (LE), 276

Lupus erythematosus
- Widespread complications of lupus erythematosus (CC), 474

Lyme disease
- Deer, ticks, and Lyme disease (ED), 2
- Incidence and prevalence of Lyme disease in a suburban Westchester County community, 5

Lymphadenopathy
- Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314
- Occult breast cancer (CR), 555

Lymphocytic choriomeningitis
- Lassa fever, Part I—Discovering the disease, the story of (HIST), 199

Lymphoepithelial lesions
- Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314

Lymphoma
- Trauma-related presentation of non-Hodgkin's lymphoma (LE), 322
- Unusual manifestation of testicular lymphoma, an (CR), 270

MAID regimen
- Chemotherapy for hepatic angiosarcoma (LE), 322

Malpractice
  Roots of the modern medical malpractice crisis, the, 279

Mammography
  Metastatic hemangiopericytoma of the breast (CR), 158
  Occult breast cancer (CR), 555

Marine Hospital Service
  Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353
  United States public health service and merchant seamen, the (ED), 331

Mario Cuomo, Governor
  Public health campaign for New York State, a (ED), 329

Marital status
  Marital status and stage at diagnosis in cancer, 8

Mastectomy
  Mastectomy: Is it necessary for occult breast cancer? (ED), 516
  Occult breast cancer (CR), 555

Maugham, W. Somerset
  W. Somerset Maugham, Capgras syndrome, and aging, 547

Measles
  Public health campaign for New York State, a (ED), 329

Medicaid
  Access to health care in rural western New York State, 465
  Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489
  Professional liability reform and access to Medicaid obstetric care in New York State, 237
  "Rationing America's Medical Care" (ER), 451
  Role of third party payors in medical care, the (LE), 323
  Triple jeopardy: The health hazards of unraveling insurance, uncontrolled illness, and economic adversity (ED), 513

Medicaid Prenatal Care Assistance Program
  Providing access to obstetric care in New York State (ED), 235

Medical ethics
  Ethical considerations concerning the HIV-positive physician (committee report), 151
  Judicial ruling on a physician violation of New York State law protecting the confidentialty of HIV-related information, 545

Medical Fraternity
  Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Medical futility
  Medical futility, 485

Medical insurance
  Role of third party payors in medical care, the (reply) (LE), 449

Medical law
  Do-not-resuscitate order in a nursing home: Patient's choice or staff's decision, the, 131
  Ethical considerations concerning the HIV-positive physician (committee report), 151
  Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181
  Judicial ruling on a physician violation of New York State law protecting the confidentialty of HIV-related information, 545
  Professional liability reform and access to Medicaid obstetric care in New York State, 237

Medical liability
  Physician and the courts, the (ED), 375

Medical licensing
  National Practitioner Data Bank, 12
  Observations on the National Practitioner Data Bank (ED), 1

Medical record documentation
  Island Peer Review Organization educational posters, 57

Medical school
  Physician and the poor, the (LE), 162

Medical Society of the State of New York
  Ethical considerations concerning the HIV-positive physician (committee report), 151
  History of the Women's Medical Society of New York State, a (HIST), 203
  Illicit drug use should not be legalized (committee report), 59
  In support of the *New York State Journal of Medicine* (LE), 558
  Inaugural address of the President (ED), 233
  Medical futility, 485
  Medical Society of the State of New York Universal Health Plan: A proposal for improving the United States health care system, 45
  New York State Journal of Medicine, the (LE), 449
  Physician-assisted suicide (committee report), 388
  Plea for keeping the *New York State Journal of Medicine* alive, a (LE), 558
  Providing access to obstetric care in New York State (ED), 235

Medical Society of the State of New York, House of Delegates
  Ethical considerations concerning the HIV-positive physician (committee report), 151
  Illicit drug use should not be legalized (committee report), 59
  Medical futility, 485
  Physician-assisted suicide (committee report), 388

Medical Students' Aid Society
  Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Medical waste
  Regulated medical waste: Is any of it infectious?, 349

Medical Women's National Association
  History of the Women's Medical Society of New York State, a (HIST), 203

Medicare
  Healing waters, 306
  Role of third party payors in medical care, the (LE), 323

Medline computer data base
  Update on the *New York State Journal of Medicine,* an (ED), 294

Melanoma
  Clavicular resection in the exposure and resection of malignant tumors (CR), 214
  Total body photography for melanoma surveillance, 494

Memorial
  Bernard Jacobs, MD:, 1924–1992, 372

Merchant seamen
  Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353
  Health care for seamen in the Port of New York: Part II—The rise of the seaman's retreat (HIST), 394
  United States public health service and merchant seamen, the (ED), 331

Mesotheliomas
  Cardiac tumors: A current perspective (ED), 41

Methyldopa
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Metoprolol
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Midwifery
  History of Jewish attitudes toward nursing, a (HIST), 529

Migraine
  Headaches and children: A reply to "An urban primary school health program" (LE), 24

Minority, health care
  Challenge of controlling tuberculosis in New York City, the (ED), 291
  Lead screening and follow-up in an urban pediatrics clinic, 338
  Marital status and stage at diagnosis in cancer, 8
  Proper obstetric care for women in New York State (LE), 502
  Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245

Minors
  Economic interventions to discourage the illegal sale of cigarettes to minors in New York State, 520

Minoxidil
  Adverse cutaneous reactions from cardiovascular drug therapy, 137

Miscellaneous
  Catfight in the labs, 385
  Not a valentine, 386

Missionary physicians
  Common interests and shared objectives (ED), 234

Mobile Army Surgical Hospital
  Bernard John Dowling Irwin, MD: Physician, naturalist, and military hero (HIST), 537

Monoclonal gammopathy
  Hyperlipidemia in monoclonal gammopathy (CR), 496

Monoclonal immunoglobulins
  Diseases associated with monoclonal immunoglobulins (ED), 463

Morgagni hernias
  Morgagni hernia mimicking a lipomatous tumor (CR), 499

Mortality Prediction Model
  Intensive care for the elderly: Should the gates remain open? (ED), 175

Multi-drug resistance
  Challenge of controlling tuberculosis in New York City, the (ED), 291
  Tuberculosis in New York: Rapid rise of a preventable disease (ED), 293

Multifocal osteomyelitis
  Chronic recurrent multifocal osteomyelitis in adult women (CR), 360

Multiple myeloma
Diseases associated with monoclonal immunoglobulins (ED), 463

Münchausen syndrome
Münchausen syndrome (CC), 301

Mycobacterium tuberculosis
Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462
Tuberculous peritonitis as an initial manifestation of HIV infection (CR), 269

Myeloma
Hyperlipidemia in monoclonal gammopathy (CR), 496
Ten-year survival in multiple myeloma: Report of two cases and review of the literature (CR), 316

Myocardial infarction
Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction (CR), 217
Right ventricular cardiomyopathy (CR), 318

Myoclonus
Diltiazem-induced myoclonus (CR), 447

Myxedema coma
Myxedema coma (CR), 553

Myxomas
Primary cardiac tumors, diagnosis, and management in 14 cases, 49

Nadolol
Adverse cutaneous reactions from cardiovascular drug therapy, 137

National Cholesterol Education Program
New York State Dietary Survey: Fat and cholesterol consumption, the (LEN), 73
Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245

National Health and Nutrition Survey Group
Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245

National Health Interview Survey
Pap smear histories in a medical clinic: Accuracy of patients' self-reports, 421

National Institutes of Health
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

National Library of Medicine
Update on the *New York State Journal of Medicine,* an (ED), 294

National Practitioner Data Bank
National Practitioner Data Bank, 12
Observations on the National Practitioner Data Bank (ED), 1

National Task Force on Youth Suicide
Prevention of youth suicide (ED), 85

Native Americans
Cardiovascular risk factors in native American children, 378

Natural disasters
Health and humanitarian assistance in conflicts and disasters (ED), 461

Necrotizing fasciitis
Facial necrotizing fasciitis due to *Streptococcus pyogenes* (CR), 111

Needle exchange program
AIDS prevention by needle exchange program, 426

Needle localization technique
Needle localization and biopsy in nonpalpable lesions of the breast: An eight-year experience (LE), 71

Needlestick injury
Low technology and the prevention of nosocomial AIDS (LE), 276

Neonatal
Neonatal gastrointestinal complications of maternal cocaine abuse (CR), 22
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Neonate
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Neoplasm
Mucinous cystic neoplasm of the pancreas (LE), 503

Neoplastic disorders
Pulmonary cavitary disease (CC), 193

Neuroblastoma
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Neurofibrosarcoma
Clavicular resection in the exposure and resection of malignant tumors (CR), 214

Neurologica diabolica
Münchausen syndrome (CC), 301

Neurology
Low-dose vincristine-associated bilateral vocal cord paralysis (CR), 268
Persistent vegetative state: An analysis of clinical correlates and costs, the, 381

Neutropenia
Neutropenia induced by low-dose captopril (CR), 219

New England Journal of Medicine
Physician-assisted suicide (committee report), 388

New York Academy of Medicine
Smallpox in an 18th-century Italian princess (HIST), 480

New York City
Challenge of controlling tuberculosis in New York City, the (ED), 291
Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185
Public health campaign for New York State, a (ED), 329
Total serum cholesterol levels in Asians living in New York City: Results of a self-referred cholesterol screening, 245
Tuberculosis in New York: Rapid rise of a preventable disease (ED), 293

New York City Board of Health
Public health crisis in New York City, the, 63

New York City Department of Health
Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185
Low level and bystander exposure to lead among factory workers, 525

New York Hospital
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

New York Port Entry Taxes
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

New York State
Access to health care in rural western New York State, 465
Economic interventions to discourage the illegal sale of cigarettes to minors in New York State, 520
Health and economic impact of cigarette smoking in New York State, 1987–1989, 469
Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181
Judicial ruling on a physician violation of New York State law protecting the confidentialty of HIV-related information, 545
National Practitioner Data Bank, 12
Observations on the National Practitioner Data Bank (ED), 1
Professional liability reform and access to Medicaid obstetric care in New York State, 237
Proper obstetric care for women in New York State (LE), 502
Providing access to obstetric care in New York State (ED), 235
Public health campaign for New York State, a (ED), 329
Tuberculosis in New York: Rapid rise of a preventable disease (ED), 293
Update on the *New York State Journal of Medicine,* an (ED), 294

New York State Cancer Registry
Marital status and stage at diagnosis in cancer, 8

New York State Department of Health
Immunization crisis in New York, the (ED), 415
National Practitioner Data Bank, 12
Observations on the National Practitioner Data Bank (ED), 1

New York State Dietary Survey
New York State Dietary Survey: Fat and cholesterol consumption, the (LEN), 73

New York State Health Care Proxy Law
Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489

New York State Journal of Medicine
Future of the *New York State Journal of Medicine*, the (LE), 408
In support of the *New York State Journal of Medicine* (LE), 558
New York State Journal of Medicine, the (LE), 408
New York State Journal of Medicine, the (LE), 449
Plea for keeping the *New York State Journal of Medicine* alive, a (LE), 558
Update on the *New York State Journal of Medicine,* an (ED), 294

New York State Legislature
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

New York University Hospital
Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181

New York Veterans Hospital
Impact of do-not-resuscitate legislation on the use of cardiopulmonary resuscitation in three teaching hospitals, 181

Nicotinic acid
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Nifedipine
  Adverse cutaneous reactions from cardiovascular drug therapy, 137
Nigeria
  Lassa fever, Part I—Discovering the disease, the story of (HIST), 199
  Lassa fever, Part II—Learning more about the disease, the story of (HIST), 264
  Lassa fever, Part III—The disease in the community, the story of (HIST), 440
Nitroglycerin
  Adverse cutaneous reactions from cardiovascular drug therapy, 137
Nocardiosis
  Nocardia brasiliensis soft tissue infection in an immunocompromised host (LE), 275
Nominating Committee
  Medical Society of the State of New York, House of Delegates, 27
Non-Hodgkin's lymphoma
  Trauma-related presentation of non-Hodgkin's lymphoma (LE), 322
Norepinephrine
  Adverse cutaneous reactions from cardiovascular drug therapy, 137
Nosocomial AIDS
  Low technology and the prevention of nosocomial AIDS (LE), 276
Nostrum
  Pigeons as a remedy (*segulah*) for jaundice, 189
Nursing
  History of Jewish attitudes toward nursing, a (HIST), 529
NYS Public Health Law
  Do-not-resuscitate order in a nursing home: Patient's choice or staff's decision, the, 131
Obesity
  Overweight in New York State (LEN), 224
Obituaries
  Adamo, Gaspar Mathew, 287
  Adler, Maurice, 287
  Appel, Erwin Alan, 510
  Arak, Charles, 568
  Auli, Manuel E., 458
  Auringer, Edward M., 287
  Bass, Sara, 171
  Battaglia, Russell L., 458
  Baum, Theodore, 568
  Berchenko, Frank, 287
  Bergner, Harold A., 371
  Berlind, Melvyn, 568
  Berman, Robert William, 26
  Best, Duncan Leonard, 458
  Betlejeski, Z. John, 287
  Bieringer, Heinz, 171
  Bivona, Charles Joseph, 287
  Blaber, Oliver John, 287
  Borofsky, Leatrice G., 230
  Bowen, George Loveridge, 171
  Brauer, Earle W., 458
  Bressler, Sydney, 230
  Brown, Francis Dickson, 230
  Burghardt, John Peter, 84
  Burke, John Heckman, 510
  Burwig, William Herbert, 287
  Campion, William F. X., 84
  Canizares, Orlando, 568
  Castilone, Samuel Joseph, 510
  Christman, Robert O., 510
  Clark, William Thomas, 230
  Cohen, Victor Louis, 287
  Cohn, Werner M., 230
  Colby, Edmund Donald, 26
  Conway, Alexander, 230
  Coseglia, Patrick Rudolph, 287
  Crane, Henry Aloysius, 26
  Cyrus, Earland, 171
  De La Garza, Alexander M., 371
  DeGara, Paul Frederick, 230
  Denneen, Edward Victor, 287
  DeRoetth, Andrew, 458
  DeSantis, Carlo E., 287
  Dorfman, Wilfred, 371
  Downey, Paul Vincent, 510
  Dranitzke, Jacob B., 287
  Draper, John W., 287
  Dubrow, Hilliard, 84
  Egbert, John Howard, 230
  Ehrlich, Leonard Isaac, 171
  Emery, John Royden, 26
  Engel, Werner Herman, 230
  Esbin, Leo, 26
  Falkenbury, Frank Mears, 568
  Faller, Harry Russell, 287
  Farrer, Sanford Miller, 458
  Favazza, Armando Gatto, 568
  Feldberg, Irving, 230
  Feldman, Bertold B., 287
  Feldman, Samuel A., 171
  Ferraiuolo, Vincenzo, 171
  Fineman, Abraham, 230
  Fishberg, Arthur M., 568
  Forrest, Susanne Faust, 26
  Fracasso, Joseph Ernest, 287
  Freeman, Robert Parker, 287
  Friedman, Emerick, 371
  Friedman, Murry Nathan, 287
  Fumia, Fred Joseph, 171
  Futernick, Benjamin, 288
  Gaillard, G. Everett, 568
  Galiani, Joshua L., 371
  George, H. Louis, 171
  Gitlin, Milton Felix, 458
  Gluck, Regina, 26
  Goewey, Stephen Robert, 230
  Goldbert, Donald David, 288
  Goldstein, Louis A., 171
  Goldstein, Rasela, 230
  Granata, Anthony Joseph, 171
  Grassheim, Margaret G., 568
  Greenbaum, Henry, 568
  Greenberg, Sidney Saul, 288
  Greene, David G., 171
  Greenwald, Murray, 171
  Greenwald, Murray, 171
  Gregersen, Orman, 288
  Griesau, William August, 371
  Grokoest, Albert W., 371
  Gurin, David, 371
  Gusmano, Salvatore, 371
  Habif, David V., 458
  Hain, David D., 26
  Handelsman, Milton, B., 511
  Hannan, Bernard D., 230
  Harkavy, Myron B., 171
  Harvey, Salmon Carl, 568
  Hecht, Bernard Charles, 458
  Heenan, Edmund Roet, 288
  Heimoff, Leonard L., 568
  Heinlein, John A., 458
  Herrmann, Jack W., 171
  Holtzman, Milton, 171
  Horowitz, William, 568
  Hosley, Morrison John, 568
  Howe, Suzanne Adele L., 171
  Jacobs, Bernard, 372
  Jaksy, Joseph J., 230
  Jenks, Paul C., 371
  Jenson, Leif G., 171
  Jones, Glenn Eugene, 172
  Kachalsky, Hyman Dave, 288
  Kalenderian, Hayk, 288
  Kaplan, Lawrence I., 230
  Kaplowitz, Daniel, 172
  Karetzky, Alexander, 172
  Karp, Harrison Myron, 458
  Karsch, Arnold Lewis, 26
  Kava, Harry Louis, 84
  Keenan, Robert James, 510
  Kime, James S., 288
  Kleinberg, Frederick, 510
  Koch, Jules M., 172
  Kristeller, Edith Lind, 172
  Kron, Theodor, 288
  Kuczmarski, Leo N., 26
  LaFlare, Joseph F., 458
  Langman, Louis, 172
  Lanjewar, Dattatraya G., 230
  Lassman, Samuel, 568
  Lavacca, Michael N., 230
  Lazo, Josefina C., 230
  Leavitt, Stuart Jay, 172
  LeBow, Herman A., 231
  Legato, Augustus T., 288
  Lent, Melbourne H., 371
  Leonard, Henry, 458
  Levy, Erwin, 172
  Levy, F. Robert, 231
  Lieblich, Tera, 172
  Lindenfeld, Erwin, 458
  Liu, Ping P., 84
  Loffredo, Carmine Jos, 288
  Loseke, Lucile Elise, 84
  Louria, Milton Roland, 172
  Luhr, Alfred Francis, 84
  Macaulay, Alice Ittner, 568
  Mache, Charles L., 288
  Maitland, Leo C., 510
  Mamelok, Harold L., 568
  Masotti, George Martin, 288
  Mauro, John Baptiste, 172
  McCormick, Robert C., 84
  McGowan, Lillian E., 371
  Merrill, Ambrose P., 231
  Messura, Richard Michael, 172
  Miles, Thomas Vincent, 568
  Moore, Melvin, 288
  Moskowitz, Morris, 288
  Nach, Joseph, 26
  Natt, David John, 231
  Noto, Salvatore Louis, 172
  Olivet, John Arthur, 26
  Ottaviano, Felix, 569
  Pack, Daniel Richard, 231
  Peck, Samuel Mortimor, 458
  Peluso, Joseph, 231
  Perry, Daniel Benjamin, 288
  Phillies, Eustace G., 231
  Pisani, Anthony J., 289
  Pollak, Hans, 288
  Polsky, Murray, 26
  Pone, Jesse James, Jr., 569
  Preefer, Charles Joseph, 26
  Quander, Milton Ford, 172
  Redmond, Albert C., 231
  Reid, John H., 371
  Reiger, Anthony C., 371
  Riley, Joseph Mead, 172
  Rini, Bessie L. Silver, 172
  Roizin, Leon, 371
  Roque, Maria Victoria, 569
  Rosbasch, Gustav, 172
  Rosenberg, Harold, 172
  Rosenfeld, Hyman, 172
  Rosner, Edward W., 371
  Rosso, Albert J., 26
  Rottino, Antonio, 231
  Ruppe, John Paul, Sr., 84
  Sarnoff, Jack, 510
  Sass, Edward George, 371
  Scagnelli, Dominick P., 288
  Scal, Howard Norman, 458
  Schaechter, Jakob Samson, 231
  Schechter, Samuel, 288

Schimel, John Louis, 231
Schlesinger, Oscar, 510
Schuman, Alvin, 371
Schwartz, Felix, 84
Schwarzer, Sigmund, 288
Seismann, Isabelle D. A., 26
Shapin, Sydney, 231
Sheppard, Herman L., 569
Sholtzow, Maurice N., 288
Sickles, C. Garret, 231
Siegel, William B., 458
Sierp, Michael, 371
Singer, Irving, 172
Sommer, Beatrice E. Scot, 172
Sonberg, Morris, 510
Spanbock, Joseph, 458
Springer, Morris, 173
Sreter, Oscar, 173
St. John, Elmer George, 231
Stern, Walter R., 231
Stettenbenz, Carl A., 231
Stolfi, Julius E., 570
Susbac, H. Egon, 231
Swartz, Irving, 458
Swiller, A. Irving, 289
Taheri, Manoucher, 569
Tartakow, I. Jackson, 372
Tartell, Paul, 510
Taus, H. Harold, 173
Taylor, Harry Newman, 459
Teperson, Hyman Irving, 26
Thompson, Annis Eveleen, 173
Throne, Harry Moses, 459
Tillotson, Byron Calvin, 289
Treichel, William F., 173
Tuby, Joseph J., 173
Tuchman, Marvin S., 84
Valvo, Joseph A., 289
Van Coevering, Russell J., II, 459
Venet, Louis, 231
Vilardo, William, 84
Wahrsinger, Philip Benjamin, 372
Walczak, Paul M., 289
Warner, Robert, 459
Weare, William G., 173
Wechsler, Solomon, 84
Wegweiser, Herbert F., 569
Weinstein, Harold, 372
Welner, Daniel H., 173
Wiedeman, George H., 231
Willner, Herman Herbert, 289
Winning, Stuart A., 289
Winsted, Wendy Louis, 459
Wolfe, W. Brewster, 232
Wood, Thornton H., 569
Yellen, Irving, 232
Yeon, Woon Bok Rhee, 173
Yu, Paul N., 84
Zimmer, Thomas Sigfried, 569
Zwirecki, Robert John, 232

Obstetrics
Professional liability reform and access to Medicaid obstetric care in New York State, 237
Proper obstetric care for women in New York State (LE), 502
Providing access to obstetric care in New York State (ED), 235

Obstetric cancer
Large endometrioma with intrauterine pregnancy (CR), 364

Occult breast cancer
Occult breast cancer (CR), 555

Occupational Safety and Health Administration
Low level and bystander exposure to lead among factory workers, 525

Odocoileus virginianus
Deer, ticks, and Lyme disease (ED), 2
Incidence and prevalence of Lyme disease in a suburban Westchester County community, 5

Officers 1991–1992
Medical Society of the State of New York, House of Delegates, 27

Oliguria
Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Oncology
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Onondaga County
Judicial ruling on a physician violation of New York State law protecting the confidentialty of HIV-related information, 545

Onondaga nation
Cardiovascular risk factors in native American children, 378

Opportunistic lung infection
Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297

Oral aphthous ulcers
Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids (LE), 221

Oregon Basic Health Services Act
"Rationing America's Medical Care" (ER), 451

Orphan Drug Act
Drug development and patent law (ED), 86

Orthopedic surgery
In lieu of a plaque (LE), 222

Osler-Weber-Rendu disease
Osler-Weber-Rendu disease: Diagnosis and management of spontaneous hemothorax during pregnancy (CR), 551
Pregnancy and pulmonary arterio-venous fistula (ED), 515

Osseous sarcoidosis
Lytic osseous destruction in vertebral sarcoidosis (CR), 213
Osseous sarcoidosis, radiologic imaging, and physician interaction (ED), 177

Osteomyelitis
Chronic recurrent multifocal osteomyelitis in adult women (CR), 360

Osteonecrosis
Alcoholism-induced bone necrosis, 334

Ovarian cysts
Large endometrioma with intrauterine pregnancy (CR), 364

Pancreas
Mucinous cystic neoplasm of the pancreas (LE), 503

Pancreatitis
Alcoholism-induced bone necrosis, 334

Pap smear
Pap smear histories in a medical clinic: Accuracy of patients' self-reports, 421

Parotid gland
Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314

Parotidectomy
Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314

Paroxysmal nocturnal hemoglobinuria
Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the (ED), 88
Recombinant tissue-plasminogen activator for acute Budd-Chiari syndrome secondary to paroxysmal nocturnal hemoglobinuria (CR), 109

Patent law
Drug development and patent law (ED), 86

Patient confidentiality
Judicial ruling on a physician violation of New York State law protecting the confidentialty of HIV-related information, 545

Patient Self-Determination Act
Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489

Pediatrics
Hyperbilirubinemia in the term infant (LE), 221
Lead screening and follow-up in an urban pediatrics clinic, 338
Osler-Weber-Rendu disease: Diagnosis and management of spontaneous hemothorax during pregnancy (CR), 551
Physically handicapped children's program: A helping hand for children with serious health problems, the (LE), 408
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Pediatrics diet
Cardiovascular risk factors in native American children, 378

Peer-reviewed journal
Update on the *New York State Journal of Medicine*, an (ED), 294

Pelvis
Transitional cell carcinoma of the renal pelvis in an incompletely duplicated collecting system (CR), 500

Pericardiocentesis
Epidemic Kaposi's sarcoma presenting with pleuropericarditis (CR), 359

Pericytes of Zimmerman
Metastatic hemangiopericytoma of the breast (CR), 158

Peritonitis
Resolution of fungal peritonitis after early catheter removal without amphotericin B therapy (LE), 162

Peromyscus leucopus
Deer, ticks, and Lyme disease (ED), 2

Persistent vegetative state
Patient Self-Determination Act and patient-physician collaboration in New York State, the, 489
Persistent vegetative state: An analysis of clinical correlates and costs, the, 381

Petit mal
Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53

Pharmacology
Adverse cutaneous reactions from cardiovascular drug therapy, 137
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Drug development and patent law (ED), 86
Drug poisoning patterns: The Albany experience, 1978 and 1988, 134

Phenytoin
Phenytoin and smoking (LE), 71

Phi Lambda Kappa
Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Photography
Total body photography for melanoma surveillance, 494

Physical therapy
Healing waters, 306

Physically Handicapped Children's Program
Physically handicapped children's program: A helping hand for children with serious health problems, the (LE), 408

Physician patient relationship
Waiting for the doctor, 262

Physician-assisted suicide
Physician-assisted suicide (committee report), 388

Physicians
How we tell our doctors about pain (LE), 162
Physician and the poor, the (LE), 162

Pigeons
Pigeons as a remedy (*segulah*) for jaundice, 189

Pike v Honsinger
Roots of the modern medical malpractice crisis, the, 279

Pindolol
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Plaque reduction neutralization test
Lassa fever, Part III—The disease in the community, the story of (HIST), 440

Plasma norepinephrine
Antihypertensive effects of exercise training, the, 250

Pleuropericarditis
Epidemic Kaposi's sarcoma presenting with pleuropericarditis (CR), 359

Pneumococcal cellulitis
Pneumococcal cellulitis and dysgammaglobulinemia (CR), 113

Pneumoconiosis
Pulmonary cavitary disease (CC), 193

***Pneumocystis carinii* pneumonia**
AIDS-related *Pneumocystis* pneumonia complicated by diffuse pneumatocele formation (CR), 20
Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297
Evaluation and clinical management of children with HIV infection, 280
Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65
Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids (LE), 221

Pneumonia
AIDS-related *Pneumocystis* pneumonia complicated by diffuse pneumatocele formation (CR), 20
Rhabdomyolysis associated with pneumococcal pneumonia and sepsis in an abstinent alcohol abuser (LE), 24

Widespread complications of lupus erythematosus (CC), 474

Pneumothorax
Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462

Poetry
Children of hippocrates 1992, 373

Poison control
Drug poisoning patterns: The Albany experience, 1978 and 1988, 134
Exploring poisoning data collection systems (ED), 127

Poisoning data collection systems
Drug poisoning patterns: The Albany experience, 1978 and 1988, 134
Exploring poisoning data collection systems (ED), 127

Polycythemia vera
Bilateral adrenal hemorrhage in polycythemia vera (CR), 362

Pool Program
Healing waters, 306

Port of New York
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353
Health care for seamen in the Port of New York: Part II—The rise of the seaman's retreat (HIST), 394
United States public health service and merchant seamen, the (ED), 331

Post-traumatic stress disorder
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Prazosin
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Pregnancy
Osler-Weber-Rendu disease: Diagnosis and management of spontaneous hemothorax during pregnancy (CR), 551
Pregnancy and pulmonary arterio-venous fistula (ED), 515

Procainamide
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Propafenone
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Prostate surgery
Choice of surgical procedure for prostatic surgery (LE), 408

Protoporphyrin
Lead screening and follow-up in an urban pediatrics clinic, 338
Low level and bystander exposure to lead among factory workers, 525

Pseudoleukemia
Pseudoleukemia in a patient with Hodgkin's disease (LE), 449

Pseudologica fantastica
Münchausen syndrome (CC), 301

Pseudomonas aeruginosa
Tunneled central venous catheters complications and the iatrogenic superior vena cava syndrome (ED), 43

Pseudoseizures
Update in epilepsy, Part I—Diagnosis and treatment of epilepsy, 14
Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53

Psychiatry
Catfight in the labs, 385
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400
W. Somerset Maugham, Capgras syndrome, and aging, 547

Psychology
Challenge to psychiatry by psychologists: Economic versus professional issues, the, 105

Psychopharmacology
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Public health
Public health crisis in New York City, the, 63

Public Health Campaign
Public health campaign for New York State, a (ED), 329

Pulmonary arterio-venous fistula
Pregnancy and pulmonary arterio-venous fistula (ED), 515

Pulmonary cavitary disease
Pulmonary cavitary disease (CC), 193

Pulmonary hemorrhage
Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Pulmonary hypertension
Acute tricuspid valve incompetence secondary to blunt trauma (CR), 445

Pulmonary thromboembolism
Progressive pulmonary thromboembolism in association with HIV disease (CR), 403

Pulmonary tuberculosis
Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462

Purified protein derivative
Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185
Lytic osseous destruction in vertebral sarcoidosis (CR), 213
Tuberculous peritonitis as an initial manifestation of HIV infection (CR), 269

Purpura fulminans
Purpura fulminans, metastatic endophthalmitis, and thrombotic thrombocytopenic purpura in an HIV-infected patient (CR), 313

Purulent bronchitis
Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297

Quarantine Station
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

Quinidine
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Radiologic imaging
Osseous sarcoidosis, radiologic imaging, and physician interaction (ED), 177

Raynaud's phenomenon
Widespread complications of lupus erythematosus (CC), 474

Readership survey
Update on the *New York State Journal of Medicine,* an (ED), 294

Recombinant tissue-plasminogen activator
Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the (ED), 88
Recombinant tissue-plasminogen activator for acute Budd-Chiari syndrome secondary to paroxysmal nocturnal hemoglobinuria (CR), 109
Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction (CR), 217

Referees
Our referees, 328

Reference Committees
Medical Society of the State of New York, House of Delegates, 27

Religion
Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Renaissance, the
Leonardo da Vinci's anatomical drawings (HIST), 436

Renal disease
Eosinophilic globules resembling Mallory bodies in a renal cell carcinoma (CR), 18
Neutropenia induced by low-dose captopril (CR), 219
Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction (CR), 217
Transitional cell carcinoma of the renal pelvis in an incompletely duplicated collecting system (CR), 500

Research Based Relative Value Scales
Inaugural address of the President (ED), 233

Reserpine
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Residential health care facility
Do-not-resuscitate order in a nursing home: Patient's choice or staff's decision, the, 131

Reticulohistiocytosis
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Retinopathy
Project Diabetes 2000 (LE), 115

Retroperitoneal hemorrhage
Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction (CR), 217

Rhabdomyolysis
Rhabdomyolysis associated with pneumococcal pneumonia and sepsis in an abstinent alcohol abuser (LE), 24

Rhabdomyosarcoma
Cardiac tumors: A current perspective (ED), 41
Primary cardiac tumors, diagnosis, and management in 14 cases, 49
Skin changes associated with hematologic and oncologic diseases in children (EP), 256

Royal Flying Doctors Service
Experience of an American medical student with an Aboriginal community in central Australia, the, 431

Rural health care
Access to health care in rural western New York State, 465

SAD-PERSONS
Non-psychiatric physician's responsibilities for the suicidal adolescent, the (EP), 97

Sarcoidosis
Lytic osseous destruction in vertebral sarcoidosis (CR), 213
Osseous sarcoidosis, radiologic imaging, and physician interaction (ED), 177

Sarcomas
Primary sarcomas of the breast in women under 20 years of age (CR), 497

Saturnism
Gouty arthritis following gunshot wound: A case report (CR), 110

Schizophrenia
W. Somerset Maugham, Capgras syndrome, and aging, 547

School health program
Headaches and children: A reply to "An urban primary school health program" (LE), 24

Screening
Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185

Seaman's Fund
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

Seaman's Retreat Hospital
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353
Health care for seamen in the Port of New York: Part II—The rise of the seaman's retreat (HIST), 394

Seamen's Fund and Retreat Hospital
United States public health service and merchant seamen, the (ED), 331

Segulah
Pigeons as a remedy (*segulah*) for jaundice, 189

Self-reported medical histories
Pap smear histories in a medical clinic: Accuracy of patients' self-reports, 421

Serratia marcescens
Intralymphatic immunotherapy of glioblastoma (CR), 272

Sickle cell disease
Alcoholism-induced bone necrosis, 334

Silicosis
Pulmonary cavitary disease (CC), 193

Simplified Acute Physiology Score
Intensive care for the elderly: Should the gates remain open? (ED), 175

Sjögren's syndrome
Cystic benign lymphoepithelial lesion of the parotid gland in an HIV-infected patient (CR), 314

Skeletal disease
Chronic recurrent multifocal osteomyelitis in adult women (CR), 360
Osseous sarcoidosis, radiologic imaging, and physician interaction (ED), 177

Sleep physiology
Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Smallpox
Smallpox in an 18th-century Italian princess (HIST), 480

Smallpox Hospital
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

Smith v Goodyear
Roots of the modern medical malpractice crisis, the, 279

Smoking-attributable health care costs
Health and economic impact of cigarette smoking in New York State, 1987–1989, 469

Society for the Right to Die
Physician-assisted suicide (committee report), 388

Somalia
Health and humanitarian assistance in conflicts and disasters (ED), 461

Somatoform disorders
Münchausen syndrome (CC), 301

Spironolactone
Adverse cutaneous reactions from cardiovascular drug therapy, 137

Splenic rupture
Spontaneous rupture of the spleen as the initial manifestation of acute myeloid leukemia (CR), 160

Spouses
Ultimate care givers, the (LE), 116

Staphylococcus
Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297
Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65
Tunneled central venous catheters complications and the iatrogenic superior vena cava syndrome (ED), 43

State University of New York Health Science Center at Brooklyn
History: The great reminder (LE), 559
History—The great reminder: The relationship between the Long Island College Hospital and the State University of New York Health Science Center at Brooklyn (HIST), 392

Staten Island quarantine
Health care for seamen in the Port of New York: Part I—Recognizing the special needs of seamen (HIST), 353

Status epilepticus
Update in epilepsy, Part I—Diagnosis and treatment of epilepsy, 14
Update in epilepsy, Part II—Special considerations in the treatment of epilepsy, 53

Steroid
Alcoholism-induced bone necrosis, 334

Steroids
Adrenal hemorrhage in a patient with inflammatory bowel disease treated with steroids (CR), 552
Importance of differentiating between anabolic steroids and glucocorticoids, the (LE), 365
Treatment of human immunodeficiency virus associated with oral aphthous ulcers with inhaled steroids (LE), 221

Streptococcal lung infection
Pulmonary cavitary disease (CC), 193

Streptococcus pneumoniae
- Pneumococcal cellulitis and dysgammaglobulinemia (CR), 113
- Purpura fulminans, metastatic endophthalmitis, and thrombotic thrombocytopenic purpura in an HIV-infected patient (CR), 313
- Rhabdomyolysis associated with pneumococcal pneumonia and sepsis in an abstinent alcohol abuser (LE), 24

Streptococcus pyogenes
- Facial necrotizing fasciitis due to *Streptococcus pyogenes* (CR), 111

Streptococcus viridans
- Bronchitis mimicking opportunistic lung infection in patients with human immunodeficiency virus infection/AIDS, 297

Strongyloides stercoralis
- Strongyloides hyperinfection syndrome, the (CR), 67

Strongyloidiasis
- Strongyloides hyperinfection syndrome, the (CR), 67

Sudan Interior Mission
- Lassa fever, Part II—Learning more about the disease, the story of (HIST), 264
- Lassa fever, Part III—The disease in the community, the story of (HIST), 440

Suicide
- Non-psychiatric physician's responsibilities for the suicidal adolescent, the (EP), 97
- Physician-assisted suicide (committee report), 388
- Prevention of youth suicide (ED), 85

Superior vena cava syndrome
- Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65
- Tunneled central venous catheters complications and the iatrogenic superior vena cava syndrome (ED), 43

Supreme Court
- Drug development and patent law (ED), 86

Surgeon general
- Bernard John Dowling Irwin, MD: Physician, naturalist, and military hero (HIST), 537

Sympathetic nervous system
- Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Sympathomimetics
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Syphilis
- Public health campaign for New York State, a (ED), 329

Syringes
- AIDS prevention by needle exchange program, 426

Systemic lupus erythematosus
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Tachycardia
- Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Talmud
- Pigeons as a remedy (*segulah*) for jaundice, 189

Teenage smokers
- Economic interventions to discourage the illegal sale of cigarettes to minors in New York State, 520

Testis
- Unusual manifestation of testicular lymphoma, an (CR), 270

Therapy
- Medical futility, 485
- Occult breast cancer (CR), 555

Third party payors
- Role of third party payors in medical care, the (LE), 323
- Role of third party payors in medical care, the (reply) (LE), 449

Thoracic
- Primary and secondary aortoenteric fistula and thoracic aortic aneurysm (CR), 156

Thrombocytopenic purpura
- Purpura fulminans, metastatic endophthalmitis, and thrombotic thrombocytopenic purpura in an HIV-infected patient (CR), 313

Thromboembolic disease
- Pulmonary cavitary disease (CC), 193

Thrombolytic therapy
- Renal angiomyolipoma presenting as a retroperitoneal mass following thrombolytic therapy for acute myocardial infarction (CR), 217

Thrombosis
- Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the (ED), 88
- Widespread complications of lupus erythematosus (CC), 474

Tobacco
- Cigarette advertising in news magazines (LE), 502
- Economic interventions to discourage the illegal sale of cigarettes to minors in New York State, 520
- Health and economic impact of cigarette smoking in New York State, 1987–1989, 469
- Phenytoin and smoking (LE), 71

Tocainide
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Tracheostomy
- Low-dose vincristine-associated bilateral vocal cord paralysis (CR), 268

Transesophageal echocardiography
- Acute tricuspid valve incompetence secondary to blunt trauma (CR), 445
- Cardiac tumors: A current perspective (ED), 41
- Transesophageal echo: Defining the indications (ED), 418

Transference as remedy
- Pigeons as a remedy (*segulah*) for jaundice, 189

Transitional cell carcinoma
- Transitional cell carcinoma of the renal pelvis in an incompletely duplicated collecting system (CR), 500

Trauma centers
- Healing waters, 306

Triamterene
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Tricuspid valve
- Acute tricuspid valve incompetence secondary to blunt trauma (CR), 445

Tricyclic antidepressants
- Do tricyclic antidepressants have a protective effect in post-traumatic stress disorder? (CR), 400

Tropical Disease Center of Lenox Hill Hospital
- Common interests and shared objectives (ED), 234

Tuberculosis
- Challenge of controlling tuberculosis in New York City, the (ED), 291
- Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185
- Hazard of bronchial lesions in the surgical treatment of drug-resistant pulmonary tuberculosis, the (ED), 462
- Public health campaign for New York State, a (ED), 329
- Tuberculosis in New York: Rapid rise of a preventable disease (ED), 293

Tuberculosis contact investigation
- Contact investigation and isoniazid chemoprophylaxis in New York City, 1979–1987, 185

Tuberculous peritonitis
- Tuberculous peritonitis as an initial manifestation of HIV infection (CR), 269

Tubular necrosis
- Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Tumors
- Large endometrioma with intrauterine pregnancy (CR), 364

Uniform requirements for manuscripts
- Uniform requirements for manuscripts submitted to biomedical journals, 122

United States health care system
- Medical Society of the State of New York Universal Health Plan: A proposal for improving the United States health care system, 45

Universal Health Plan
- Medical Society of the State of New York Universal Health Plan: A proposal for improving the United States health care system, 45

University of California, Los Angeles
- Healing waters, 306

University of Pennsylvania Medical School
- Phi Lambda Kappa Medical Fraternity: Religion and medicine, the (LE), 366

Urachal cyst
- Appendiceal abscess presenting as an infected urachal cyst (LE), 365

Urothelial carcinoma
- Transitional cell carcinoma of the renal pelvis in an incompletely duplicated collecting system (CR), 500

US Centers for Disease Control
- Lassa fever, Part III—The disease in the community, the story of (HIST), 440

US Department of Health and Human Services
- Prevention of youth suicide (ED), 85

Valvular regurgitation
- Acute tricuspid valve incompetence secondary to blunt trauma (CR), 445

Vasculitis
- Oliguria, hemoptysis, and arthralgias in an otherwise healthy woman (CC), 343

Venereal Disease Research Laboratory
- United States public health service and merchant seamen, the (ED), 331

Venous catheters
- Superior vena cava syndrome: A complication of Hickman catheter insertion in patients with the acquired immunodeficiency syndrome (CR), 65
- Tunneled central venous catheters complications and the iatrogenic superior vena cava syndrome (ED), 43

Venous thrombosis
- Continuing problem of thrombosis in paroxysmal nocturnal hemoglobinuria, the (ED), 88
- Recombinant tissue-plasminogen activator for acute Budd-Chiari syndrome secondary to paroxysmal nocturnal hemoglobinuria (CR), 109

Ventricular dilated cardiomyopathy
- Right ventricular cardiomyopathy (CR), 318

Ventricular premature beats
- Morphology of ventricular premature beats a marker for left left ventricular dysfunction? is the, 89

Verapamil
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Vincristine
- Low-dose vincristine-associated bilateral vocal cord paralysis (CR), 268

Visiting Nurse Service
- Hospice care (LE), 72

Vocal cord paralysis
- Low-dose vincristine-associated bilateral vocal cord paralysis (CR), 268

von Münchausen, Karl Friederich Hieronymus, Baron
- Münchausen syndrome (CC), 301

Waldenström macroglobulinemia
- Diseases associated with monoclonal immunoglobulins (ED), 463

Warfarin
- Adverse cutaneous reactions from cardiovascular drug therapy, 137

Wasteland, the
- Golden Years or The Wasteland, the, 148

Westchester County
- Incidence and prevalence of Lyme disease in a suburban Westchester County community, 5
- Primary cardiac tumors, diagnosis, and management in 14 cases, 49

Whipple, Electra B., MD
- History of the Women's Medical Society of New York State, a (HIST), 203

Women, Infants, and Children program
- Proper obstetric care for women in New York State (LE), 502

Women's issues
- A history of the Women's Medical Society of New York State (HIST), 203
- Changing winds, the (LE), 449
- Ultimate care givers, the (LE), 116

Women's Medical Society of New York State
- History of the Women's Medical Society of New York State, a (HIST), 203

Worker's health
- Low level and bystander exposure to lead among factory workers, 525

World War II
- Memories of an internship in 1943, 307

Yale Arbovirus Research Unit
- Lassa fever, Part I—Discovering the disease, the story of (HIST), 199
- Lassa fever, Part II—Learning more about the disease, the story of (HIST), 264
- Lassa fever, Part III—The disease in the community, the story of (HIST), 440

Youth suicide
- Non-psychiatric physician's responsibilities for the suicidal adolescent, the (EP), 97
- Prevention of youth suicide (ED), 85

Zinc protoporphyrin
- Lead screening and follow-up in an urban pediatrics clinic, 338

AUTHOR INDEX

Abdalla, Mazan A., 503
Abunassar, Solange G., 301
Ahn, Chul, 424
Ahronheim, Judith C., 181
Alcorn, Charles M., 179, 518
Alfredsunder, P., 268
Allam, Azmy A., 297
Allen, Steven L., 474
Alpern, Harlan D., 343
Alpert, Barbara, 5
Amis, E. Stephen, Jr., 217
Anderson, Christian G., 275
Angeles, Katherine, 89
Ansari, Zaeem, 496
Aronoff, Michael S., 105
Aronow, Wilbert S., 424
Asaikar, Shailesh, 447
Asase, Danilo, 500
Aschkenasy, Jean R., 97
Aswad, Charles N., 45
Avran, Morell M., 412

Balz, Thomas, 81
Barek, Dennis, 369
Barman, A. Addison, 156
Barnowsky, Lawrence, 343
Barrera, Raphael, 111
Barth, Robert, 24
Bartolomeo, Robert S., 552
Becker, Melvin H., 284
Bennett, Allen J., 59, 151, 388, 485
Berger, Barbara J., 275
Bernstein, Anne Elayne, 326
Betzu, Robert, 89
Bevelaqua, Frederick A., 551
Bharathan, Thayyullathil, 503
Bianco, Joseph T., 507
Birkhead, Guthrie, 415
Biro, Frank, 164
Birrer, Richard B., 24
Bixon, Ronda, 217
Blackshear, Joseph L., 418
Blackwood, James, 25
Bloom, Alan A., 276
Bloomfield, Randall D., 75, 116, 365, 375, 449, 567
Bluestone, Naomi R., 385, 410
Bluestone, Peter, 474
Boba, Antonio, 276
Bordley, Donald R., 446
Botash, Ann S., 378
Boyce, John G., 421
Brandstetter, Robert D., 175
Brar, Harleen, 110
Brecher, David B., 77
Breitbart, Arnold S., 158
Brennessel, Debra, 360
Brickner, Philip, 118, 454
Broe, Daniel M., 506
Brown, Algie C., 120
Brown, William, 365
Bunkin, Irving A., 148
Burlage, Robb K., 506
Buscaglia, Anthony, 59, 151, 388, 485
Butensky, Michael S., 69

Cahill, Kevin M., 234
Camilien, Louis, 421
Campbell, Carla, 525
Cantos, Eric, 213
Cantu, Roberto, 555
Canver, Charles C., 445, 558
Caruso, Robert, 168
Cash, Mary, 313
Cassaro, Sebastiano, 115
Cassell, Eric J., 59, 151, 388, 485
Cerda, Jorge, 134
Chapnick, Edward K., 221
Charatan, Fred B., 408
Chartash, Elliott, 474
Chaudhuri, Gautam, 169
Chechani, Vijay, 297
Chien, Allen, 318, 404
Chmiel, James F., 276
Clark, David C., 97
Cone, David C., 338
Constantiner, Arturo, 301
Cooper, David, 474
Coppersmith, Harold, 301
Crofford, Marsha, 322
Cummings, K. Michael, 469, 520

Daliana, Maurizio, 115
Danyluk, Andrew, 166
Davis, II, William Arthur, 326
Davis, John S., 558
De Antonio, Mark, 286
De Fries, Zira, 105
DeHovitz, Jack A., 564
De La Torre, Robert, 245
Del Giacco, Eric J., 343
Desai, Himanshu P., 552
Dibianco, Joseph T., 283
Dick, Robbin S., 446
Dietrich, Kathleen A., 318, 404
Dilello, Edmund, Sr., 79
Disla, Eddys, 110
Dooley, Frances, 404
Doran, Maureen, 228
Drusin, Lewis M., 164
Dugan, Sean F.X., 349
Dunbar, Christopher C., 250
Dunn, John M., 553
Durgin, Francis J., 449

Edbauer, Michael J., 365
Edelstein, Martin, 373
Edson, John N., 166, 285, 392, 559
Eggers, Arnold E., 272
Emm, Nancy, 378
Ente, Gerald, 116
Esin, John, 5

Falkenstein, David B., 69
Farhangi, Mehdi, 463
Farmer, Peter, 474
Farnsworth, Peter B., 59, 151, 388, 485
Feldman, Joseph G., 8
Felice, Philip V., 111
Fernandez, Henry, 1
Fins, Joseph J., 489
Firlik, Andrew D., 426
Fishbein, Alf, 525
Fitzpatrick, Annelle, 368
Fleischman, Heidi, 162
Fleisher, Jay, 165
Flood, Katherine M., 327
Flynn, John T., 301
Fortunato, Lucio, 555
Foster, Zelda, 120
Frame, John D., 199, 264, 410, 440
Frank, Ronald G., 500
Frankel, Etta B., 322
Friedman, Eli A., 162
Friedman, Sandor A., 403
Frishman, William H., 137
Fruchter, Rachel G., 421
Furie, Richard, 474

Galloway, Jacqueline, 262, 284, 306
Ganick, Dorothy J., 256
Gargiulo, Janet E., 323
Gasner, M. Rose, 455
Gelfand, Maxwell L., 362
Gerard, Perry S., 499, 500
Gershberg, Herbert, 162
Gerst, Paul H., 503
Giella, John, 217
Giorgi, Diane F., 301
Giuliano, Armando E., 559
Glaser, B., 507
Glatt, Aaron E., 269
Golbey, Stuart, 499
Gold, Mark S., 456
Golub, Robert A., 555
Goodman, Bevery Hallquist, 567
Gordon, Milton, 294
Gordon, Ronald E., 18
Gordon, Wally, 462
Gould, Lawrence A., 89
Gould, Perry C., 552
Gradon, Jeremy D., 221
Graner, John L., 412
Greenberg, Michael D., 566
Greenwald, Earl, 129
Groeger, Jeffrey S., 77
Grossman, Marc, 137
Grünwald, Hans W., 316
Guiney, William, 343
Gulati, Subhash C., 507
Gupta, Buhpendra K., 214
Gutstein, Hal, 424

Halpern, Abraham L., 59, 151, 388, 485
Hamburg, Margaret A., 291
Hammad, Ali, 113
Handwerger, Sandra, 65
Hansard, Paul, 109
Hanson, Eric C., 41
Harper, Rita G., 22
Harrington, Paul C., 314
Harris, David T., 564
Harris, Matthew N., 158
Helbig, Donald, 325
Hena, Muhammad A., 408
Henein, Sherif, 20
Henry, John B., 59, 151, 388, 485
Herbert, Lawrence, 71
Hertz, Alan, 226, 327
Heurich, Albert, 78
Hill, A. Ross, 20
Hilton, Eileen, 67
Hilton, Thomas C., 418
Hinman, Edward J., 331
Hinterbuchner, L. P., 162
Hirschfield, Larry, 270
Hitti, Ibrahim, 322
Hochman, Melvin C., 268
Hoefer, Jean M., 203
Hoffman, Robert S., 127
Hoisington, Samuel A., 359
Hollenberg, Norman K., 412
Holmes, Valerie F., 400
Hooper, Michele M., 162
Horowitz, Steven F., 245
Hughes, Joseph, 553

Ifudu, Onyekachi, 24, 162
Imparato, Anthony M., 75
Imperato, Pascal James, 63, 179, 372, 461, 511, 518, 561, 570
International Committee of Medical Journal Editors, 122

Jacob, Christina N., 20
Jacobs, Bernard, 334
Jain, Pramod C., 360
Jamil, Syed A., 67
Jarcho, Saul, 480
Jelveh, Zeba, 213
Jeret, Joseph S., 447
Johnson, Lois, 221
Jones, Bessie C., 118
Jones, Diane, 378
Joseph, Paul R., 221
Joseph, Sovi, 503
Josephson, Adele, 285

Kabakow, Bernard, 388
Kalman, Andrea C., 316
Kalmuk, Eugene, 113
Kamholz, Stephan L., 297
Kaneko, Mamoru, 18
Kanick, Virginia, 59
Kape, Salomea, 222
Kaplan, Barry H., 268
Kaplan, Mark, 474
Kaplan, Steven A., 217
Karakousis, Constantine P., 214
Kark, Pieter R., 59, 151, 388, 485
Kaufman, David Myland, 381
Kavaler, Elizabeth, 431
Kavaler, Florence, 353, 394
Kavey, RaeEllen W., 378
Kemeny, M. Margaret, 516
Kenigsberg, Kenneth, 22, 116
Kerr, Paul, 156
Kinne, David W., 497
Kizer, Kenneth W., 76
Kohman, Leslie J., 203
Kohn, Ira, 217
Koplin, Richard S., 169
Korenbrot, Carol C., 237
Kovin, Leo J., 307
Krafchuk, John D., 323
Krasner, Melvin I., 513
Kshettry, Vibhu R., 503
Kurtz, Morton M., 233
Kwan, Tak, 109

Lamb-Abel, Barbara J., 237
Landau, Stuart W., 318, 404
Landolt, Allison B., 59, 151, 388, 485
Lane, Lewis B., 325
Lange, Richard H., 408
Laraja, Raymond D., 71
Lastavica, Catherine C., 2
Lavie, Sol, 275

Leachman, Robert Dewitt, 166
Leber, Mark, 78
Lee, Bok Y., 406
Lefleur, Richard, 551
Levine, Ann Marie, 365
Liebert, Peter S., 502
Liebowitz, Martin R., 72
Lindsay, Kris, 500
Lipton, Richard B., 381
Locharernkul, Chaichon, 14, 53, 92
Loeb, Laurence, 59, 151, 388, 485
Logan, Bruce, 301
Lomanto, Charles, 129
Lomax, James D., 226, 227, 283, 283, 455
Lowenstein, Robert, 59, 151, 388, 485
Lucas, Alexander R., 77
Lucas, Cheryl A., 465
Luftschein, Stanley, 137
Luna Vega, Alberto R., 497
Luo, Jiin-Chyuan J., 525
Lutwick, Larry I., 221

Maheswaran, Sathya, 181
Mahoney, Martin C., 469
Maliakkal, Roy, 403
Mannheimer, Sharon, 449
Marino, Catherine, 227, 360, 368
Markel, Mariana, 24
Mason, Maryanne, 474
Maus, Timothy P., 227, 456
Mauskop, Alexander, 24
McDonald, Elizabeth, 165, 360
McGoldrick, M. Donald, 134
McGroarty, James, 115
McKellar, Peter P., 449
Megna, Daniel, 360
Meier, Levi, 79, 565
Menghini, Mario V., 45
Menter, Alan, 505
Mentzer, Robert M., Jr., 445
Mercurio, Vito, 474
Meyer, B. Robert, 474
Migliozzi, Joseph A., 359
Miller, John I., 272
Miller, O. Fred, 225
Millner, Bernard N., 408
Milman, Doris H., 558
Miskovitz, Paul, 76
Mitnick, Julie R., 158
Moggio, Richard A., 49
Monsaert, Ronald P., 75
Moreno, Jonathan D., 376
Morilla, Carlito V., 406
Moussa, Zobidatte, 219
Moy, Donald R., 545
Munoz, Frank, 1
Mylotte, Joseph, 113

Narasimhan, Parthas, 322
Nayeri, Kamran, 8
Nenner, Raphael P., 57, 179, 518
Niezgoda, Jeffrey A., 419
Noh, Jae-Mun, 322
Norante, John D., 314
Nordlicht, Stephen, 564
Novick, Lloyd F., 293, 329
Numann, Patricia J., 59, 151, 388, 485
Nusbaum, Neil J., 86, 276

Odom, John W., 25
Ojemann, George A., 14, 53, 92
Ojemann, Linda M., 14, 53, 92
Ona, Fernando V., 59, 151, 388, 485
Ordorica, Steven A., 551
O'Shaughnessy, Kathryn, 412
O'Toole, Kathleen M., 217

Palmer, Michael, 301
Parikh, Virendra A., 503
Parithivel, Vellore S., 503
Pascarelli, Emil, 119
Pastena, Janis A., 25
Patel, Jay K., 322
Patel, Minal, 322
Pechacek, Terry, 520
Penzer, Paul, 116
Perino, Giorgio, 18
Pettit, William, 165
Pfeffer, Cynthia R., 85
Phibbs, Ciaran, 237
Pilcher, Webster H., 14, 53, 92
Pilon, Verne, 408
Pinnelas, David, 245
Pitaro, Gregory, 8
Pooley, Richard W., 49
Posner, Gerald, 503
Presant, Cary A., 43
Primrose, David, 14, 53, 92
Pryce, Edith H., 71
Pucillo, Anthony L., 49
Pugh, Judith, 245

Ragnauth, Paul, 111
Rait, Douglas S., 566
Ratner, Harold, 71
Reddy, Kumar S., 360
Redo, S. Frank, 368
Reed, George E., 49
Reiner, David M., 137
Remick, Scot C., 359
Resnick, Gene D., 164
Resnik, Charles S., 177
Richmond, Gary, 65
Richtsmeier, Anthony J., 97
Ring, Malvin E., 115, 505
Risemberg, Herman M., 59, 151, 388, 485
Ritota, Perry, 25
Robb, James, 12, 454
Rogatz, Peter, 59, 151, 388, 485
Rones, Kathie, 421
Rosen, Zachary B., 81, 119
Rosenberg, Carl, 181
Rosenbluth, Michael A., 367
Rosenthal, Thomas C., 465
Rosner, Fred, 59, 151, 189, 276, 316, 388, 485
Roth, Tracy, 421
Rothenberg, Robert E., 71
Rowe, Jacob M., 449
Rumore, Peter, 213

Sacher, Howard L., 318, 404
Sacher, Michael L., 318, 404
Sadovsky, Richard, 226
Salman, Shamin, 219
Salomi, Ira L., 111
Santella, Robert N., 343
Sapan, Melissa, 474
Sarabu, Mohan R., 49
Sawczuk, Ihor S., 365
Sawicki, Janusz, 111
Schechter, Andrew G., 49
Schiano, Thomas, 219
Schiff, Howard, 18
Schneck, Jerome M., 547
Schoenfeld, Myron, 424
Schoenfeld, Norman, 65
Schrieber, Katrina, 426
Schwartz, Gary J., 552
Schwarz, Richard H., 235, 502
Sciandra, Eva, 520
Sciandra, Russell, 469
Sclafani, Salvatore, 272
Sechzer, Philip H., 59, 151, 388, 485
Sekovski, Blaze, 445
Selman, Jay, 79, 120
Senderoff, Elliot, 499
Serebriakov, Leonid, 81
Serur, David, 160
Shah, Bharat S., 71
Shaikh, Shahin, 213
Shelton, Clough, 168
Sherman, Jack, 116
Sherris, David A., 314
Shinbach, Kurt, 301
Shire, Nora Krisch, 436
Shivaram, Urmila, 313
Sholar, Pam Westermoreland, 88
Sicuranza, Genevieve B., 413
Silen, William, 167
Silk, Yusuf N., 408
Silverstone, Felix A., 131
Sivak, Steven L., 5
Sloan, Don, 502
Slue, William E., Jr., 494
Smith, Peter R., 297
Sokoloff, Leon, 529
Somasundaram, Mahendra, 447
Sordillo, Peter P., 151, 388, 485
Sorrento, Joseph J., 555
Soubani, Ayman O., 213, 269
Spatoliatore, Giuseppe, 219
Spiegel, Allen D., 367, 376, 411, 451, 537
Spielberger, Lawrence, 558
Spinazzola, Regina, 22
Sridhar, Sundara, 403
Standfast, Alfred L., 116
Stapleton, F. Bruder, 365
Stenzel, Kurt, 456
Stiles, Jeffrey, 469
Stolfi, Julius, 169
Stone, Lois Greene, 386, 508
Stotka, Jennifer L., 332
Strand, Calvin, 245
Strasser, Alexander L., 449
Swartz, Louis, 552
Swinburne, Andrew J., 515

Taheri, Paul A., 445
Talavera, Wilfredo, 65
Taranta, Angelo, 110
Telischi, Fred F., 168
Terjanian, Terenig, 160
Thaler, Otto F., 326
Thung, Swan N., 18
Tizes, Reuben, 408
Tortella, Bartholomew J., 25
Tretola, Michael, 566
Turchan, Steven J., 400
Turkewitz, David, 167

Unger, Pamela, 18
Urizar, Rodrigo, 134
Usmani, Shaista S., 22

Van Scoy-Mosher, Michael B., 225
Vazquez, Madeline, 158
Vecchio, Andrew J., 301
Vennema, Alje, 185
Vetto, John T., 497

Wagner, Carolyn S., 131
Wasserman, Charles S., 400
Webber, Carolyn A., 297, 421
Weiss, Jeffrey N., 270
Wesson, Donald E., 507
Wilck, Eric, 499
Wise, Gilbert, 500
Wolf-Klein, Gisele P., 131
Wolpaw, Jonathan R., 59, 151, 388, 485
Wong, Ping Fai, 301
Wormser, Gary P., 5
Wright, Karen, 318
Wright, Kenneth W., 559

Young, Bruce, 551
Young, Richard A., 279

Zambito, Raymond F., 121
Zaslau, Stanley, 366
Zavin, Shirley A., 353, 394
Ziegelbaum, Michael, 270
Zinn, L. David, 97
Zittel, Molly, 113
Zografos, George C., 214

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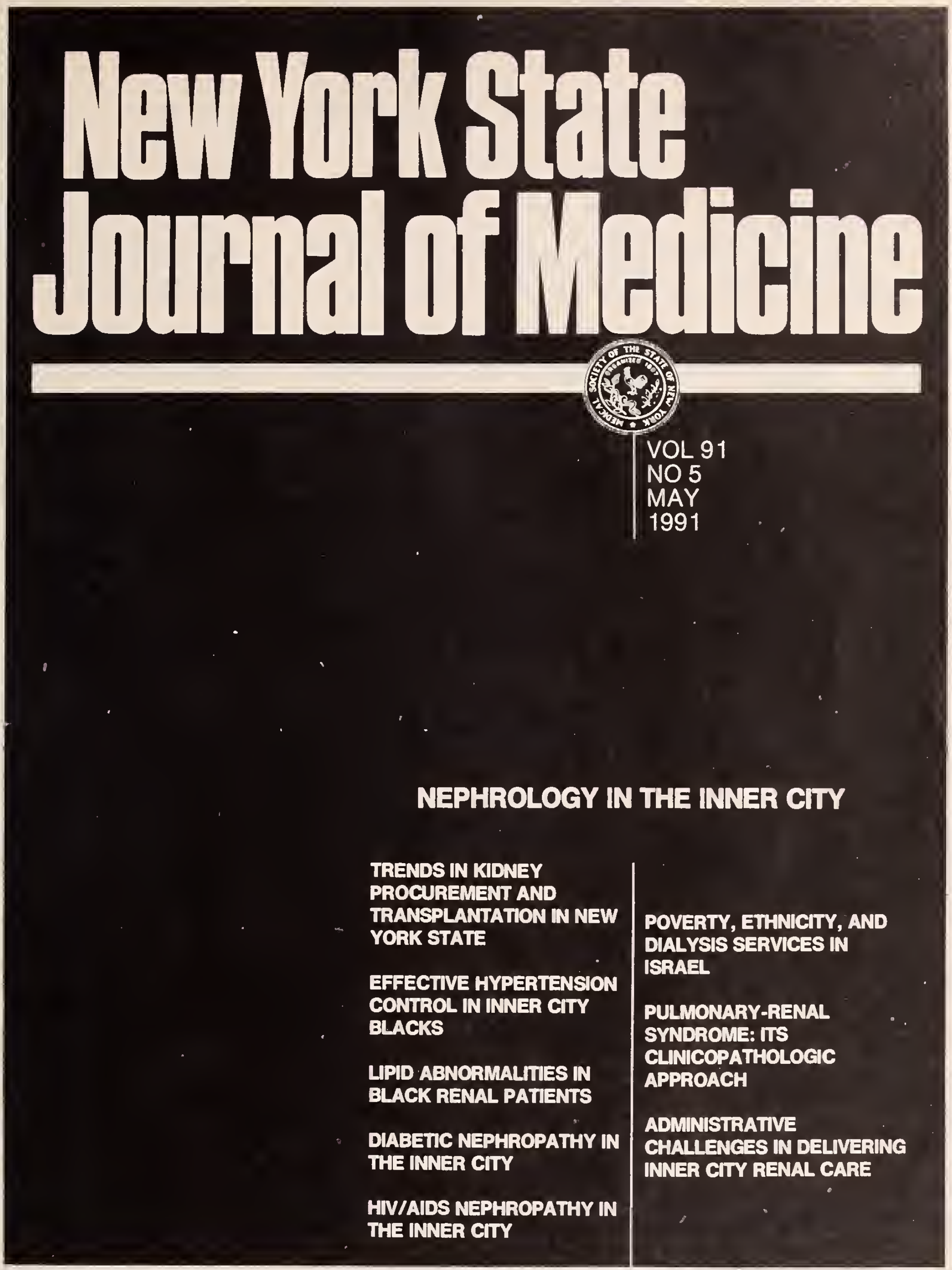

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VOL 91
NO 5
MAY
1991

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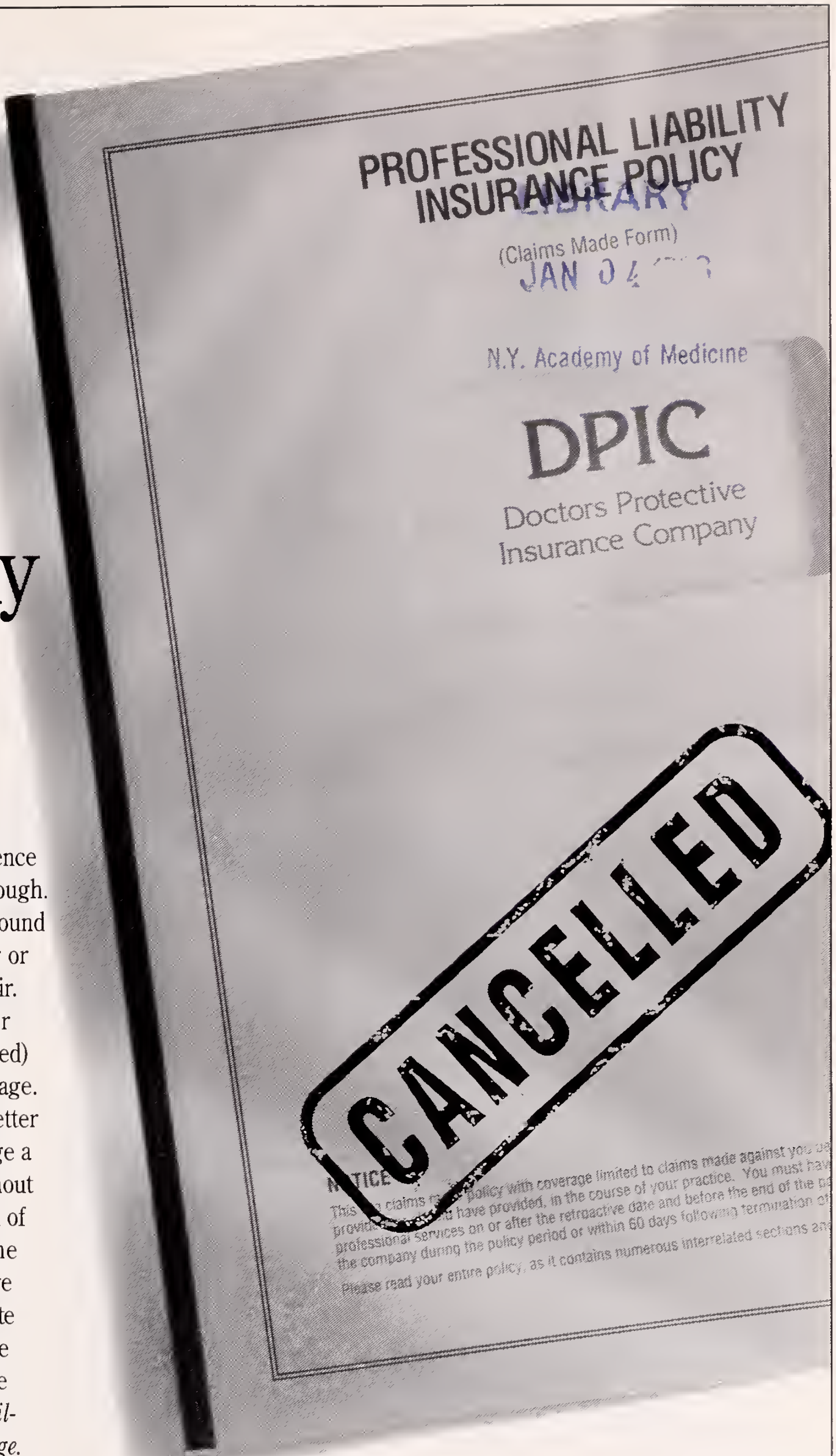

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